Swallow Syncope

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ABSTRACT: Swallow syncope is an often misdiagnosed rare disorder due to enhanced vagal tone during eating in patients with underlying esophageal and/or cardiac abnormalities. We present three cases of this disorder, one related to digitalis toxicity and the other two with diffuse esophageal spasm. The investigation, differential diagnosis, prognosis and management of swallow syncope are discussed.

RÉSUMÉ: La syncope de déglutition. La syncope de déglutition est un problème rare, souvent mal diagnostiqué, dû à une augmentation du tonus vagual pendant l'alimentation chez des patients présentant des anomalies oesophagiennes et/ou cardiaques sous-jacentes. Nous présentons trois cas de syncope de déglutition, dont un est relié à une intoxication digitalique et les deux autres à un spasme oesophagien diffus. L'investigation, le diagnostic différentiel, le pronostic et le traitement sont discutés.

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Recurrent spells of loss of consciousness during eating or swallowing is a rare symptom. Differential diagnosis includes swallow syncope, a vagus-mediated form of reflex cardioinhibition with swallowing[1,2]; eating epilepsy, a rare variety of reflex epilepsy[3,4] and extremely rarely glossopharyngeal neuralgia triggered by swallowing with secondary vagally-mediated cardiac syncope.[5-7] The following 3 cases of swallow syncope are presented to draw attention to this often misdiagnosed disorder and its management.

Case Reports

Patient 1 — Swallow syncope responding to propanthelene bromide (Pro-Banthine R) This woman was first seen in 1978 at the age of 62 with a 2-year history of loss of consciousness with meals occurring once or twice per month. There was a brief aura of a hot sensation followed by a typical syncopal attack and on one occasion her head fell into her plate. She would regain consciousness within 5-10 seconds. There was no preceding dysphagia, choking or throat pain. The attacks tended to occur toward the beginning of a meal and were rare with breakfast. The attacks occurred with drinking as well as eating and bore no relationship to whether she was sitting or standing, size of the meal, speed of eating or surroundings. She had no other cardiac or gastro-intestinal symptoms.

Physical examination was unremarkable. She was normotensive and had a normal cardiac examination. Valsalva manoeuvre produced a moderate bradycardia.

Resting EKG, barium swallow, chest x-ray and mediastinal tomograms, esophagoscopy, CT of the head, EEG and EEG telemetry with video monitoring during eating were all normal. Esophageal manometry done with EKG monitoring revealed abnormally high pressures (160 mmHg) in the upper esophagus and 140 mmHg in the lower esophagus during swallowing. Inflation of an esophageal balloon to a pressure of 20 mmHg 35 cm from the lower lip induced spontaneous high-pressure (100 mmHg) esophageal contractions. The patient felt nauseous and lightheaded and went into first and then briefly second degree heart block (Figure 1). EEG was not monitored. Blood pressure did not fall. IV Atropine was given.

On propantheline bromide taken 45 minutes before meals she had only 4 attacks, possibly due to poor compliance, over the next 4 years.

Patient 2 - Swallow syncope due to digoxin toxicity This 62-year-old man with longstanding rheumatoid arthritis on prednisone 2.5 mg per day, penicillamine, aspirin and a nonsteroidal anti-inflammatory was also receiving digoxin 0.25 mg per day and a diuretic for congestive heart failure. On Christmas Eve 1981 he suddenly fainted while eating, fell out of his chair and broke his glasses. No abnormal movements were seen and he regained consciousness within seconds. He had 2 other similar episodes while eating, 6 days and 4 months later. He also had several lesser episodes of lightheadedness while eating, especially when eating quickly and taking large mouthfuls.

Physical examination was normal apart from findings related to rheumatoid arthritis.

EKG revealed a rate of 60 with first degree heart block. Holter monitor testings showed brief periods of second degree heart block, type II. A barium swallow study was normal except for mild reflux and marked reduction in peristalsis in the lower third of the esophagus. EEG was normal. Digoxin level was 2.8 ng/ml (therapeutic: 1-2 ng/ml).

During EEG telemetry/video/EKG monitoring while eating (given peanut butter to swallow) he had 3 episodes of lightheadedness associated with heart rate of 30 and second degree heart block (Figure 2). The EEG was unaltered at this time.

Digoxin was discontinued and no further syncopal attacks occurred over the next 2 years. He was re-admitted in December 1982 with inferior myocardial infarction and third-degree heart block requiring a permanent pacemaker insertion.

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Patient 3. Swallow syncope mistaken for epilepsy This 40-year-old woman had a 9-month history of dysphagia with food or liquids tending to stick in her pharynx. About twice weekly, while eating she suddenly felt lightheaded, became pale and lost consciousness for about 1 minute. *Stiffening and at times some jerks of her limbs were observed with* these spells. She was often drowsy for about ½ hour afterwards.

Physical examination was unrevealing. EKG, CT of the head and chest x-ray were all normal. Barium swallow was normal aside from a small sliding hiatus hernia. EEG/telemetry/EKG/video monitoring during eating was normal on three occasions. Two-dimensional echocardiography was normal. Esophageal manometry revealed high-pressure waves (average 150 mmHg) recorded around the upper esophagus and cricopharyngeus muscle.

Angina pectoris was subsequently diagnosed as well as type IV hyperlipidemia. She was treated with desipramine hydrochloride for depression. There were only a few syncopal attacks over the next year.

Discussion

Only twenty-nine patients with swallow syncope have been documented in the literature. A recent report of 5 cases[1] seen over 4 years in one hospital suggests that the diagnosis may be more common than previously thought and can be easily missed by those unfamiliar with this entity. Our experience with 3 cases supports the conclusion that the diagnosis may be initially overlooked, even by experienced clinicians.

Almost all patients have shown esophageal or cardiac pathology which coexist in many cases. The esophageal disorders have either been structural[8,9] (eg. carcinoma, achalasia, stricture, diverticula) or functional[10] (eg. diffuse spasm or sphincter spasm). In several cases high esophageal pressures and peristaltic abnormalities were recorded by manometry as in our cases 1 and 3.[10] In a few cases inflation of an esophageal balloon produced cardiac arrhythmias (usually transient block or asystole) with faintness as in our case 1.[10] The most common cardiac abnormalities which have been associated with swallow syncope are ischemic heart disease, inferior infarction and conduction disturbances producing heart block.

The arrhythmias provoked by swallowing which may lead to syncope include various degrees of A-V block, nodal or sinus bradycardia, ventricular asystole and atrial fibrillation.[11] In at least 2 other reported cases[1,12] digitalis was a contributing factor as in our second case. Presumably the mechanism relates to the vagotonic effects of digitalis with a tendency to produce A-V conduction block especially on the background of pre-existing cardiac conduction abnormalities.

The pathophysiology of swallow syncope relates to an abnormal esophagocardiac reflex whereby there is vagal excitation and cardioinhibition during swallowing or esophageal distension. The reflex is likely mediated through vagal esophageal afferents synapsing in the medullary nucleus of the fasciculus solitarius connecting with vagal efferents from the nucleus ambiguus to the heart.[2] Weiss & Ferris[13] were able to abolish swallow syncope in a patient with an esophageal diverticulum by infiltration of the vagus trunk high in the neck. In a unique patient with swallow syncope studied by Levin & Posner[2] an autopsy study revealed demyelination of the vagus nerve and carcinomatous infiltration of the glossopharyngeal nerve adjacent to the brainstem. In rare patients without demonstrated esophageal or cardiac abnormalities, subclinical deficits of intracardiac conduction have been postulated. A vasodepressor mechanism may also be postulated in some cases where bradycardia is not a feature.

The differential diagnosis of swallow syncope includes eating epilepsy[3,4] and syncope secondary to glossopharyngeal neuralgia triggered by swallowing.[5,6,7] As with other forms of syncope epileptic phenomena such as a brief tonic seizure or a few clonic jerks may occur with swallow syncope, especially if the patient remains in the upright position after losing consciousness. Our case 3 and one of Armstrong et al's[1] cases were initially referred with a diagnosis of epilepsy. Eating epilepsy, a rare form of reflex epilepsy, occurs with chewing and swallowing of food but not with liquids. By contrast swallow syncope also occurs with drinking and in some cases may be precipitated most readily by swallowing iced or hot drinks.[1,14] Patients with eating epilepsy also usually have seizures at times unassociated with eating. The seizures in eating epilepsy are usually complex partial seizures or generalized tonic-clonic seizures, often secondarily generalized. The recovery from swallow syncope is rapid with unconsciousness usually lasting less than 10 seconds. Interictal temporal EEG abnormalities are also frequent in patients with eating epilepsy and esophageal or cardiac pathology is not associated. In cases where the diagnosis is in doubt intensive EEG/video monitoring to record an attack while eating may resolve the problem.

Glossopharyngeal neuralgia is frequently triggered by swallowing liquids or solids or by touching the posterior pharynx. An attack may be followed by a brief period of cardiac asystole and syncope due to vagal excitation in the brainstem. In some cases the attacks have proven fatal and pacemaker insertion is frequently recommended. Pain in the throat prior to syncope is a distinguishing feature from swallow syncope.

Investigation of patients with swallow syncope should include upper gastrointestinal radiography; cine-esophageal studies; esophagoscopy; esophageal manometry; mediastinal radiographic studies; EKG monitoring during eating, drinking cold and hot liquids or esophageal balloon inflation.

The management of swallow syncope has been controversial. Surgical procedures to deafferent the esophageal wall in the area where distension appears to provoke syncope have been generally unsuccessful. Because of the potentially fatal nature of the condition, some authors[1] recommend permanent pacemaker insertion. Others have found, as have we, that in the absence of obvious cardiac pathology requiring a pacemaker, anticholinergic agents such as propantheline bromide taken 1 hour before meals provide effective treatment.[10] In any case treatable predisposing factors such as esophageal structural pathology, mediastinal lesions or digitalis toxicity should be sought and eliminated where possible.

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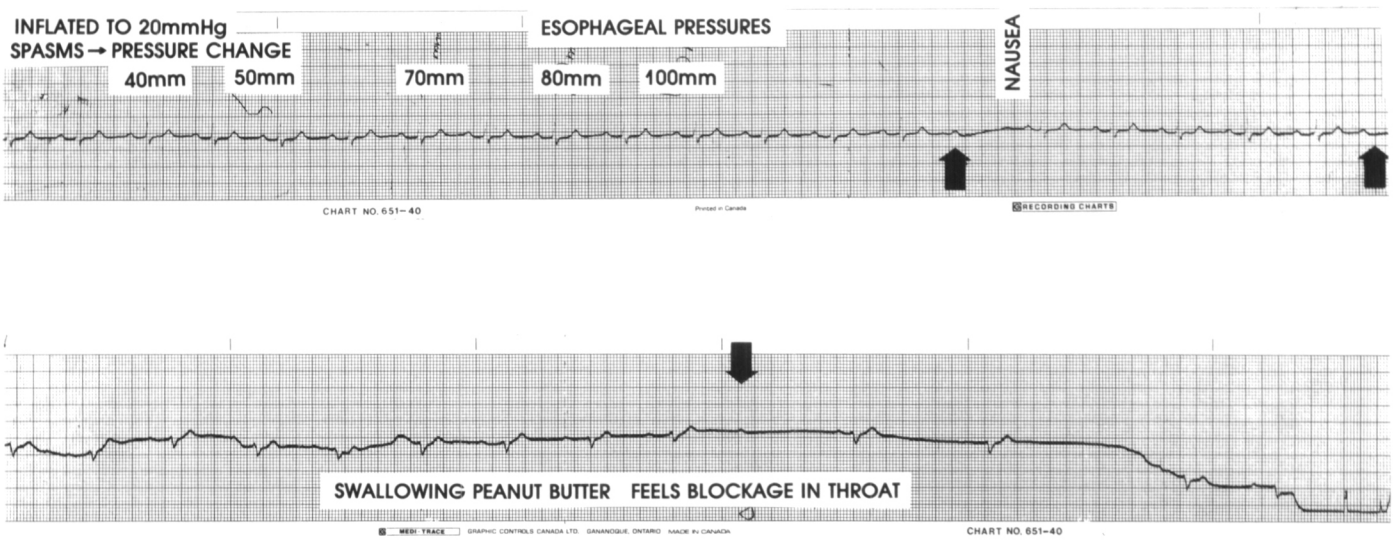

Figure 2 — EKG recorded in patient 2 while swallowing peanut butter. Second-degree atrioventricular heart block is shown (arrow) preceded by bradycardia. No syncope occurred.

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