Syncope as an Indication of Digitalis Toxicity

By Sol Glotzer, M.D.

Increased sensitivity of the carotid sinus to stimulation is known to accompany digitalis toxicity. Syncope induced by deglutition as a manifestation of this hypersensitivity is exceedingly unusual and warrants description.

Toxic reactions to digitalis are familiar to all who treat cardiac disease, but occasionally an unusual reaction is seen that does not at first seem related, and only in the light of later events becomes obvious as a greater than ordinary degree of toxicity. Such a situation is reported here.

Case Report

A. H., a 77-year-old woman, was admitted to the Williamsburgh General Hospital on October 5, 1955, with a history of left radical mastectomy for malignant disease 15 years previously with no recurrence to date. Hypertension was of many years' duration. She had shown slight evidences of congestive heart failure some months prior to admission, had been digitalized, and was comfortable on a maintenance dose of 0.2 mg. of digitoxin for a while. Beginning in July 1955 she had noted mild dysphagia, with a feeling of a "lump" behind the sternum after swallowing. For 11 days prior to admission syncope occurred immediately after swallowing water, but not after food. Each episode was manifested by pallor, cyanosis, loss of consciousness, and mild convulsive movements, lasting for a short time.

On admission, blood pressure was 200/110, the heart was enlarged, regular, with occasional extrasystoles, and a loud musical murmur was heard at the apex, extending upward over the aortic area. No signs of congestive failure were present. X-ray of the esophagus was normal, and calcification was seen in the abdominal aorta.

The electrocardiogram showed left axis deviation, with the "hinge-door" depression of S-T segments indicative of digitalis effect. Q waves in leads II, III, and aVF suggested old damage of the posterior wall of the myocardium (fig. 1).

Pressure on the right carotid sinus induced a typical syncopal attack with cardiac arrest and nodal escape. The patient was given a swallow of cold water on 2 separate occasions, and each time she lost consciousness, with a fall in blood pressure to zero, sinus arrest, and nodal or ventricular escape. Each episode, whether induced by swallowing or by carotid sinus pressure, was identical.

Digitalis was discontinued, and potassium chloride, 2 Gm. daily, was given. After 2 days she was able to swallow liquids with no distress and carotid pressure no longer caused syncope.

Discussion

The carotid sinus is supplied by the nerve of Hering, which joins the glossopharyngeal nerve and communicates with the vagus. Pressure on the right sinus thus decreases the activity of the sinoatrial node and slows the heart.

Sensitivity of the carotid sinus is increased in a variety of conditions, particularly in myocardial infarction,1 congestive heart failure,2 and arteriosclerotic hypertensive vascular disease.3 Digitalis in excess also causes sinus bradycardia or arrest, in addition to the more familiar effects, i.e., ectopic beats, atrial tachycardia, and atrioventricular block. Sensitivity of the sinus can be increased by the administration of as little as 3 gr. of digitalis folia.2 In our patient such an increase apparently was enough to permit only the slightest vagal stimulation to cause cardiac arrest, the stimulus probably arising from rapid distention of the lower esophagus. We presume that the reason a swallow of water precipitated an attack whereas solid food did not is related to the speed of descent of the ingested material and therefore to the speed of distention. Another suggestion is that the sinus is excited by the pull exerted on the sinus wall during deglutition, either by the cervical musculature, or by the actual passage of food through the esophagus.4 That digitalis was implicated in this mechanism may be inferred by the complete disappearance of the symptom complex after discontinuance of the drug.

The association between digitalis and syncope may be purely coincidence, but it was striking enough to be suggestive. Proof of the
association, by again administering digitalis to the point of toxicity, seemed too hazardous to attempt.

**Summary**

This report presents a patient with hypertensive arteriosclerotic heart disease, in whom attacks of syncope were induced by swallowing water but not solid food. The cause was traced to increased carotid sinus sensitivity resulting from digitalis toxicity, and no further episodes appeared after digitalis was discontinued. The possibility exists of a purely coincidental relationship between syncope and digitalis toxicity, but, even in the absence of proof by repetition, the association is very suggestive.

**Summario in Interlingua**

Es reportate le caso de un paciente con morbo cardiac arteriosclerotic hypertensive in qui
attacco di sincope essesse inducito per le inglutition de aqua sed non de alimentos solide. Le causa essesse trovate in un augmentate sensibilitate del sinus carotic resultante ab toxicitate per digitalis. Nulle episodios additional de syncope occureva quando le uso de digitalis essesse cessate. Il remane possibile que il se tracta in iste caso de coincidentia, sed mesmo in le absentia de provas per repetition, le association de syncope con toxicitate per digitalis es molto plausibile.

REFERENCES


According to the Bronsted theory, an acid is a hydrogen ion donor and a base a hydrogen ion acceptor. In this simple framework, mechanisms of normal and deranged acid-base physiology have been sketched. Renal function in acid-base balance has been considered and the role of carbonic anhydrase in proton excretion has been emphasized. Carbon dioxide transport has also been examined; the role of hemoglobin in this process and the concomitant electrolyte shifts are easily understood in terms of the Bronsted theory. It is in the realm of clinically deranged acid-base balance, however, that the Bronsted theory appears most valuable. A number of pathologic conditions have been discussed and, in all, this theory seems to lend clarity and simplicity. In acidosis, blood hydrogen ion concentration increases. Renal compensation for this increase in hydrogen ion is by liberation of bicarbonate into the blood with concomitant excretion of protons—as ammonium ions and as dihydrogen phosphate ions—in an acid urine. This proton excretion mechanism occurs, whether renal sodium ion excretion is increased, as in acid-gaining acidosis, or decreased, as in respiratory, or in base-losing acidosis. This mechanism applies whether serum sodium is decreased, as in acid-gaining acidosis and base-losing acidosis or increased, as may occur in respiratory acidosis. In alkalosis, the normal kidney conserves acid by failing to excrete ammonium ion and by excreting monohydrogen phosphate rather than forming the more highly protonated dihydrogen phosphate. The kidney can also excrete base as bicarbonate and possibly trimetacarboxylates. Renal compensation for alkalosis is thus simply conservation of acid and excretion of base.

The unity inherent in the Bronsted theory seems to afford a simple and precise presentation of the mechanisms of acid-base balance. Once the primary hydrogen ion regulation is understood, the electrolyte alterations become apparent from the requirements of electroneutrality.

Maxwell
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