Swallowing Syncope: Complex Mechanisms of the Reflex

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Abstract

A 69-year-old woman was admitted to our hospital for the examination of syncope. When she ate solid food, she had dizziness or loss of consciousness. The ambulatory ECG suggested sino-atrial block during swallowing with a maximum sinus pause of 6 seconds. An electrophysiologic study revealed pre-existing sinus node dysfunction, which was exaggerated by the balloon inflation in the esophagus. Atropine counteracted the slowing of the basal sinus rate induced by esophageal pressure, but it did not block the effect on the maximum sinus node recovery time. This observation suggested that the syncope was mediated partly by a non-vagal mechanism.

(Key words: electrophysiologic study, esophageal manometry, sinus node)

Introduction

Faintness or syncope during deglutition is a rare clinical entity (1), but has been described in the literature as swallowing syncope (2, 3). Although some patients have an anatomical abnormality of the esophagus (tumor (4) or diverticulum (5)), the mechanism of this syndrome has been usually attributed to an abnormal reflex via the vagus between the esophageal afferent fiber and the cardiac efferent terminals because atropine sulfate could block the phenomenon (2, 3). In this report, we describe a case of swallowing syncope whose abnormal reflex could not be blocked by atropine. This case suggested the complex mechanism of the syndrome.

Case Report

A 64-year-old woman was admitted to our hospital for the examination of repeated syncope during deglutition. Her symptom had been recognized for 5 years, but was worsening in terms of frequency. She felt dizziness or faintness every time she ate solid food such as rice or sponge cake. Syncope during a meal was experienced once or twice daily. Physical examination revealed only mild hypertension (160/84 mmHg), but other findings were normal. Heart rate was regular at 66/min. Routine laboratory tests were all within normal limits. ECG was normal. Chest X-ray film showed left pleural thickening, which was due to tuberculous pleuritis at the age of 20. Echocardiography was normal. The fluoroscopic examination of the esophagus and the upper alimentary tract was normal.

Ambulatory monitor recordings of the ECG revealed sinus arrest only during a meal with accompanying faintness (Fig. 1). Most of the sinus pause was considered to be sino-atrial block due to the multiplicity of the preceding P-P intervals (Fig. 2). The maximum sinus arrest was 6 seconds (Fig. 1B). Sinus pause was never detected during day or night when she did not eat anything.

Some provocative tests were tried. Hypotension and/or significant bradycardia were not induced by bilateral carotid massage, or Valsalva maneuver. Liquid (hot or cool) intake, pharyngeal pressure, or cough failed to induce bradycardia. When she ate rice or sponge cake repeatedly, sinus pause was observed.

An invasive electrophysiologic study was performed to examine the sinus node function. Esophageal pressure was controlled by means of a Sengstaken-Blakemore tube, which was inserted normally from the mouth. The esophageal balloon was connected to a sphygmomanometer. The same pacing protocol was repeated in the presence and the absence of the esophageal pressure and/or autonomic drug(s). A written, informed consent of the patient was obtained before these procedures. The results are shown in Table 1. In the basal condition, sinus rate was reduced as the esophageal pressure was increased from 0 mmHg to 100 mmHg. Blood pressure remained stable, and no sinus pause was induced during the maneuver. This phenomenon was blocked completely after the venous injection of atropine sulfate (0.05 mg/kg). Sinus node recovery time was measured as the interval between the last stimulus to the first sinus beat after cessation of the continuous pacing at the high right atrium for 30 seconds. The pacing rate was incremented by 20/min from 100/min to 200/min. An interval of at least 30 seconds was allowed between the trials. At 0 mmHg in the
A) Monitor recording during dinner

B) Maximum pause at lunch

Figure 1. ECG monitor recordings during meals. ECG was continuously monitored during the hospital stay. Long P-P (R-R) intervals were recorded repeatedly when the patient ate solid food A). The maximum pause detected in the record was 6 seconds B). Length of a lane in A) is 60 seconds, that in B) is 30 seconds.

Swallowing syncope has been known as a reflex-mediated syncope (2, 8). Although a different reflex arc is involved, it has been considered that this syndrome belongs to the same clinical entity as vaso-vagal syncope (2, 8, 9). The afferent fibers originate from the esophageal surface and the efferent fibers are distributed to the heart via the vagus. This hypothesis has been tested to be true because atropine sulfate could block the reflex. The present case suggested, however, that the reflex arc could be more complex than previously considered: the prolongation of the maximum sinus node recovery time and the sino-atrial conduction time under the esophageal pressure were not blocked by atropine. On the other hand, the increased esophageal pressure decreased the basal sinus rate, and this phenomenon was blocked by atropine.

When the vagal nerve is blocked, the sino-atrial conduction time and the sinus cycle length should decrease by analogy of atrio-ventricular conduction. In this case, however, the sino-atrial conduction time was prolonged after the atropine injection. This phenomenon (rate-dependent prolongation) itself means an abnormality of the conduction. Moreover, rate-dependent prolongation could cause entrance block to the sinus node when sinus node recovery time was measured. This mechanism could alleviate the sinus pause after the rapid pacing in the presence of atropine (see Table 1, control and atropine at 0 mmHg). This mechanism is not the case after the balloon inflation in the esophagus, because the maximum sinus node recovery time was prolonged even in the presence of atropine.

In this case, syncope or faintness due to sino-atrial block often occurred when she ate solid food, especially rice or sponge
Swallowing Syncope

Figure 2. Strips of monitor recordings. Long P-P intervals were multiples of the preceding P-P (sinus) interval, although some variations were seen. This calculation could be applied to most of the sinus pauses, which suggested sino-atrial block as a mechanism.

Table 1. Esophageal Manometry and the Results of Electrophysiological Study

<table>
<thead>
<tr>
<th>Esophageal pressure (mmHg)</th>
<th>Control HR (/min)</th>
<th>mSRT (msec)</th>
<th>SACT (msec)</th>
<th>BP (mmHg)</th>
<th>Atropine HR (/min)</th>
<th>mSRT (msec)</th>
<th>SACT (msec)</th>
<th>BP (mmHg)</th>
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<td></td>
<td>67</td>
<td>2,940 (180)</td>
<td>100</td>
<td>162/70</td>
<td>88</td>
<td>1,130 (200)</td>
<td>145</td>
<td>182/90</td>
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<td></td>
<td>50</td>
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<td>170/80</td>
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The dosage of atropine was 0.05 mg/kg. R: heart rate, mSRT: maximum sinus node recovery time. Pacing rate (/min) was indicated in the following parenthesis. SACT: sino-atrial conduction time by Narula method, BP: blood pressure.

cake. These types of food possibly expand when water is absorbed in the esophagus. The increased pressure in the lower esophagus could evoke the abnormal reflex resulting in sino-atrial block and long sinus arrest. This phenomenon was partly reproduced in the esophageal balloon inflation because the sinus rate was reduced and a long sinus pause was induced by the electrical stimulation. However, balloon inflation acted as a stimulus to the esophagus in a different way from that of solid food, because no sinus arrest was reproduced by this method itself. We used a Sengstaken-Blakemore tube for this purpose because of its availability in the hospital and the stability during the maneuver. Deglutition of a smaller spherical balloon might have been better to induce the arrhythmia.

Although the increased esophageal pressure by our method failed to induce sino-atrial block, the results of the electrophysiological study clearly indicated that the maneuver exaggerated the sinus node dysfunction. The maximum sinus node recovery time and sino-atrial conduction time were prolonged in the
presence of the increased esophageal pressure. These results are concordant with the clinical observation of sinus arrest and sino-atrial conduction block in this patient. This mechanism was possibly different from that of sinus slowing, because the former phenomenon was not blocked by atropine whereas the latter was blocked. This observation suggested that two or more mechanisms could be involved in the deglutition syncope, particularly in the present case.

It has been reported that some cases of swallowing syncope have an esophageal abnormality, such as neoplasm (4), diverticulum (5), and achalasia (10). The present patient did not have any disease in the esophagus. However, the patient had sick sinus syndrome because the sinus node recovery time was prolonged after total autonomic blockade without esophageal pressure (7). The patient had worsening of symptoms before admission possibly because sinus dysfunction progressed and more readily responded to the influence of the reflex. This could be an explanation of why the patient had sinus arrest instead of atrio-ventricular block, which is far more often reported in swallowing syncope.

**Conclusion**

A case of swallowing syncope was reported. Esophageal pressure imposed by balloon inflation induced 1) sinus slowing, and 2) the prolongation of the sinus node recovery time. Because atropine blocked only the former phenomenon, at least two mechanisms were involved in this reflex-mediated syncope.

**References**