Swallowing-Induced Tachycardia; Three Modalities of Autonomic Nervous Effects

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Three cases are reported who have short runs of atrial premature contractions (APCs) induced by swallowing and cough. The occurrence of the APCs were affected by autonomic nervous system in all cases. The precipitating factor is considered to be the increase of vagal tone in case 1, sympathetic tone in case 2 and both of them in case 3. The autonomic mechanism of swallowing-induced tachycardia could be divided into three categories and the treatment of these arrhythmias might be different in each group. Pharmacological autonomic blockade with atropine and propranolol is useful to reveal the underlying autonomic mechanisms.

Key words: Pharmacological autonomic blockade, Atropine, Propranolol, Swallowing arrhythmia

Swallowing-induced bradycardia is not a rare clinical entity and its mechanism has been considered to be due to the vagotonic reflex (1, 2). On the other hand, swallowing-induced tachycardia is very rare and its mechanism remains to be clarified. There are some controversies about whether the autonomic nervous tone is involved in it or not. This paper reports three cases of atrial tachycardia induced by swallowing or cough, which are considered to be affected by the autonomic nervous system in three different manners and also reports the usefulness of the pharmacological autonomic blockade to reveal involvement of the alteration of autonomic nervous tone.

CASE REPORT

Case 1: A 57-year-old man was referred to our clinic for the evaluation of arrhythmia before an operation for retinal detachment. His history showed no cardiovascular disease. Physical examination, ECG, chest X-ray, fluoroscopic examination of the esophagus with barium and blood laboratory study revealed no abnormal findings. Holter 24 hours

ECG revealed occasional paroxysmal atrial tachycardia (PAT) only when he had a cough or swallowed liquid and food (Fig). His PAT terminated spontaneously within ten beats. P-P interval was approximately 0.28–0.30 second. During a treadmill exercise test, frequent atrial premature contractions (APCs) but no runs of APCs (i.e. PAT) were observed. No diagnostic ST-T changes were detected.

His APCs and PAT were repeatedly induced by swallowing or cough. APCs and PAT was provoked immediately after the stimulation, and APCs lasted several seconds after the stimulation was stopped. We compared the effects of several drugs on these arrhythmias. When 2 mg of atropine was administered intravenously, sinus rhythm was restored as soon as the stimulation was stopped, though his APCs induced by cough or swallowing could not be suppressed completely. However, PAT with more than ten beats were induced when 1.2 mg of deslanoside, which have a cholinergic effect, or 10 mg of propranolol was given intravenously, respectively. Pharmacological interventions were

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case 1

case 2

case 3

Fig. 1. Swallowing-induced arrhythmia.

Arrow is the beginning of swallowing. "p" is ectopic atrial beat. Bars under ECG traces are the calibrations of 1 second.

Case 1: Swallowing initiates PAT.

Case 2: Swallowing induces PAT and APC (a) but swallowing under the administration of propranolol abolishes them (b).

Case 3: Swallowing provokes APCs (a). 2 mg of atropine (b) or 10 mg of propranolol (c) does not suppress them completely. In the panel (c), pacemaker shift following APC is seen.

also applied to APCs induced by treadmill exercise test. We counted the number of APCs and heart beats during the last thirty seconds of a treadmill test at stage 2 of Bruce protocol. Atropine was the only effective drug for APCs among the trials of various drugs (Table 1). Electrophysiologic study with informed consent was normal. The earliest atrial recording was obtained at high right atrium during PAT induced by swallowing. Single, couple or burst atrial pacing did not induce APCs.

Case 2: A 35-year-old man came to our clinic;

Table 1. Drug effects on exercise induced APC (Case 1).

Drug	APC/HB
Control	24/56 (43%)
Atropine	17/72 (24%)
Verapamil	28/43 (60%)
Digitalis	25/49 (52%)

APC/HB: Atrial premature contractions per total heart beats during last 30 seconds of treadmill exercise test.

his chief complaint was chest discomfort when he coughed. Physical examination, chest X-ray, ECG, fluoroscopic examination of esophagus with barium and blood chemistry showed normal findings. Holter ECG revealed chest discomfort with PAT and APCs provoked by swallowing and exercise such as climbing stairs (Fig. 1). We failed to find either APCs or significant ST-T changes of ECG during the treadmill exercise test.

Eye ball massage, carotid sinus massage and Valsalva maneuver did not induce APC. Swallowing, cough or mechanical stimulation of the pharynx induce PAT and APCs reproducibly. Swallowing under intravenous atropine (2 mg) or intradermal adrenaline (1 mg) also induced frequent PAT and APCs. On the other hand, the administration of oral propranolol (60 mg/day) abolished the PAT induced by swallowing. Local anesthesia given to the pharynx with lidocaine also abolished his arrhythmias. The data from his electrophysiologic study was normal. High right atrial pacing including single, couple and burst pacing did not induce APC. Intravenous infusion of isoproterenol (0.3 μ g/min) increased the number of APCs and PAT induced by swallowing.

Case 3: A 28-year-old man visited our clinic because of palpitation on swallowing and its increase in frequency. Physical examination, chest X-ray, ECG including a treadmill exercise test and blood chemistry was normal. Holter ECG showed PAT when he swallowed liquid or food (Fig. 1).

Valsalva maneuver, intradermal adrenaline (1 mg), intradermal pilocarpine (10 mg) had no effect on his arrhythmias. Intravenous administration of either 2 mg of atropine or 10 mg of propranolol did not abolish the arrhythmia. However, Pharmacological autonomic blockade (PAB) using

both of atropine (2 mg) and propranolol (10 mg) completely abolished his arrhythmias. In the electrophysiologic study, the only abnormal findings is the prolonged sinus node recovery time, but PAB made corrected sinus node recovery time normal from 730 ms to 390 ms. Atrial pacing on the same way as Cases 1 and 2 did not induce APC. APCs induced by swallowing originated from high right atrium.

All of the three cases showed normal findings in ^{201}Tl myocardial scintigraphy and ultrasonic examination including Doppler echocardiography.

DISCUSSION

Since Sakai & Mori reported "Schlucktachycardie" (3), several case reports have been published. There have been controversies about the mechanisms of this arrhythmia. At the present time, it is attributed to a reflex of autonomic nervous system and/or a direct mechanical compression on the left atrium.

Regarding the autonomic nervous system, the vagal reflex should be responsible for this arrhythmia because the stimulation of the alimentary tract is a predisposing factor. When vagal tone increases, the repolarization of the atrial muscle is dispersed because the refractory period varies in a different part of the atrium (4). It might favor the occurrence of the microentry in the atrium. In the previous reports, however, vagal tone was not always considered to be a causative factor; there reported the cases in whom neither vagolytic nor sympatholytic agents could eliminate APCs induced by swallowing (5-7). In those cases, the arrhythmias have been attributed to the other factor than autonomic nervous tone.

PAT and APCs of the three cases we reported here are considered to be triggered somehow under the influence of autonomic nervous tone, though each of the cases reveal themselves in different manners. In Case 1, vagotonia is a predisposing factor because atropine suppressed his PAT and APCs. Digitalis, a cholinergic agent, increased the number of APCs. Case 2 shows that increased heart rate favors the occurrence of PAT. Because they were induced by exercise such as climbing stairs or running, sympathetic tone seems to play some role in the PAT induction. In this case, the Valsalva maneuver and carotid sinus massage did not induce

PAT and swallowing under the administration of propranolol abolished them. These facts also suggest sympathetic tone is a causative factor.

In the third case, atropine or propranolol could not abolish his arrhythmias. Similar cases have been reported in which a single administration of atropine or propranolol did not have any influence on swallowing-induced tachycardia (5-7). The authors speculated that autonomic nerves did not participate in those cases. Nevertheless, PAT and APCs of Case 3 were abolished completely with both atropine and propranolol. We think this effect could not be explained without the autonomic nervous effects on the PAT induction. Therefore, pharmacological autonomic blockade should be applied to patients with swallowing-induced tachycardia in order to reveal the underlying autonomic mechanism when their tachycardia is unaffected by either vagolytic or sympatholytic agents. Deviation from the stable equilibrium between vagal and sympathetic tone may induce arrhythmias.

Direct mechanical effect on the left atrium is another causative factor of swallowing-induced tachycardia. Although we did not use the intra-esophageal manometric stimulation, arrhythmias of our cases are considered to be less attributable to this mechanism because local anesthesia to the pharynx abolished the induction of APCs. Local anesthesia might prevent the reflex at the afferent nerve endings in the pharynx.

Vaughan-Williams class 1a agents might be the drug of first choice for therapy of atrial arrhythmias. However, disopyramide reduced the number of APCs in Case 3 alone. In previously reported cases (5-7) whose arrhythmias could not be controlled by atropine or propranolol, quinidine was effective (3). Disopyramide has an effect similar to quinidine on APC so that it was applicable successfully to Case 3. In Case 1, the APCs increased in number probably because of the cholinergic effect of this agent.

Because PAT and APCs were evoked by increased sympathetic tone in Case 2, propranolol was the most reasonable drug for therapy. There have been case reports in which the patients were treated successfully with propranolol and quinidine (6, 8).

The treatment of PAT and APCs in Case 1 was difficult. Although his arrhythmias were provoked under the influence of increased vagal tone, the

vagolytic agent did not abolish them completely. Disopyramide had no effect. Digitalis and verapamil worsened the arrhythmia. In the previous cases (9, 10) in which the vagotonic influence was responsible, vagolytic agents were not always effective. Although they treated the patients with combination therapy, complete disappearance of the arrhythmias could not be achieved. As for Case 1, his symptoms were not serious so we prescribed occasional administration of atropine for him. Therapy for this group of cases remains to be investigated.

CONCLUSION

We report three cases of swallowing-induced tachycardia. Their arrhythmias were induced under the influences of autonomic nerves. Pharmacological autonomic blockade was useful to reveal the underlying autonomic mechanism. We supposed the possibility that the autonomic mechanisms of this arrhythmia were divided into three categories. The treatment of this arrhythmia might be different in each group.

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