Case Reports

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Syncope on swallowing

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Reflex stimulation of the vagus produced by swallowing and leading to ventricular asystole was observed in the patient reported herein. The results of investigations designed to delineate the reflex pathway are reported. The efferent impulses have been shown to travel in both vagi. Surgical denervation of the affected segment of the oesophagus abolished the reflex in our patient.

A variety of abnormalities of cardiac rhythm can lead to cerebral ischaemia and syncope. Perhaps the least well known of these are the reflex changes in the origin and spread of excitation in the heart appearing in relation to swallowing. In view of the rarity of the condition and somewhat interesting clinical and physiological implications, the following patient's case is reported.

Case report

A 29-year-old housewife had for about four years experienced several 'dizzy' attacks while swallowing. The attacks were essentially stereotyped but varied in duration. She first noticed blurring of vision followed rapidly by a sensation of fainting, and she would collapse to the floor if standing. Loss of consciousness for a brief duration occurred on a total of eight occasions. She remembered the fall, and witnesses did not notice any epileptic phenomenon. On two occasions she had suffered minor injuries and once dropped her baby from her arms. Minor attacks, consisting mainly of blurring of vision and fainting, were much more frequent and latterly had been occurring at each meal or in between if she happened to swallow even a piece of chocolate. There was, however, no relation with the size, consistency, or temperature of the bolus. The symptoms were worse when standing. Over the preceding year she had also been aware of food sticking at about the level of the manubrium sterni.

In view of this unusual history, the absence of any clinically detectable abnormality, and normal results from routine investigations, she was referred to one of us (P.H.G.) for a psychiatric opinion.

Electrocardiographic monitoring at this stage revealed periods of ventricular asystole during swallowing which could be blocked by the prior administration of atropine. She was then referred to Professor J. F. Goodwin at the Royal Postgraduate Medical School for further investigation and treatment.

FIG. 1 *Electrocardiogram (lead II) during swallowing showing periods of atrioventricular block of varying duration. Note the absence of slowing of the sinus rate and the lack of any escape beats. Retching also produced similar changes.*

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FIG. 2 (A) Electrocardiogram (lead II) during swallowing and during phase IV of the Valsalva's manoeuvre. During swallowing periods of atrioventricular block without any change in the sinus rate are observed, while during phase IV of Valsalva's manoeuvre the sinus rate slows. (B) These changes are no longer present after intravenous administration of 1.2 mg atropine sulphate.

FIG. 3 *Electrocardiogram (lead II) during the inflation of a balloon in the oesophagus. The event marker shows the onset and duration of the inflation. Atrioventricular block was produced when the balloon was inflated in the oesophagus (upper two rows) but not when the balloon had accidentally slipped into the stomach (third row). Local instillation of 2 per cent xylocaine into the affected segment of the oesophagus did not block the changes produced by the distension of the balloon.*

Clinical examination again revealed a normal, apparently fit, intelligent young woman. Routine haematological and biochemical tests were normal; the haemoglobin was 12.6 g/100 ml. Likewise, radiographs of the chest, tomograms of the mediastinum, and the resting electrocardiogram were all normal, as was the electroencephalogram.

A pulmonary arteriogram was performed in an attempt to delineate any vascular abnormalities which might not be visible otherwise; both the pulmonary arterial and aortic branches, the left atrium, and left ventricle were normal; right heart pressures obtained at the time were also normal.

Electrocardiographic monitoring during swallowing and during the release phase (phase IV) of Valsalva's manoeuvre showed interesting changes (Fig. 1 and 2A) which are summarized in the Table. These reflex changes were successfully abolished by the prior administration of 1.2 mg atropine (Fig. 2B).

Other manoeuvres such as carotid sinus

pressure, eye-ball pressure, and somatic pain did not produce any electrocardiographic abnormalities, and neither did gargling nor aerophagy.

Repeated barium swallow examinations in the upright, supine, and Trendelenburg positions did not reveal any abnormalities, and the oesophageal motility was considered to be normal. However, a timed barium swallow showed that the patient experienced the symptoms when the head of the bolus was close to the cardiac end of the oesophagus; the electrocardiogram at this time showed two dropped ventricular complexes.

In order to localize the afferent site more exactly a balloon titration of the oesophagus was performed. Inflation of the oesophageal balloon with 5 to 15 ml air resulted in reproducible electrocardiographic changes similar to those seen during swallowing (Fig. 3). The reflex could be elicited from the segment of the oesophagus lying between D5 and D9 vertebral bodies. Attempted mucosal block produced by 2 per cent lignocaine

TABLE Comparison of electrocardiographic changes seen during swallowing and phase IV of Valsalva's manoeuvre

	Swallowing	Valsalva's manoeuvre phase IV
Sinus rate	No change	Slowed
Origin of atrial impulse	Sinoatrial	Ectopic atrial
PR interval	Unchanged	Shortened
Conduction of atrial impulse to ventricles	Blocked, longest observed period of complete ventricular asystole 7.4 sec; no ventricular escape beats	Normal

instilled into the affected segment failed to abolish the reflex (Fig. 3).

In view of the differences between the response to vagal stimulation produced during phase IV of Valsalva's manoeuvre and that produced by swallowing, it was considered possible that the efferent impulses in the swallowing reflex might be carried selectively by one vagus nerve only. For this reason unilateral vagal block was carried out consecutively on the two sides by the local instillation of 2 per cent lignocaine in the region of the IX and X cranial nerves at the base of the skull (Mushin, 1945). The block was effective, as judged by the development of conjunctival suffusion, bovine cough, tachycardia, and a rise in the systemic blood pressure. The vagal block thus produced on either side, however, failed to abolish the reflex, though intravenously administered atropine did (Fig. 4).

These investigations indicated that the reflex could be elicited from an extensive segment of the oesophagus and that the efferent impulses were carried by both vagi, since atropine completely abolished the reflex while vagal block on either side did not do so.

The patient was initially treated with oral propantheline (15 to 30 mg t.d.s.) for two months, but this produced severe side effects and did not completely abolish the symptoms. Atropine sulphate tablets (0.6 mg) were then substituted and by trial and error the patient found that she needed to take a tablet 30 minutes before a scheduled meal. Even this dosage of atropine produced some side effects and only partially relieved the symptoms. In view of the constant threat to life from prolonged ventricular asystole which could not be completely eliminated on such a regimen, we, in consultation with Mr W. P. Cleland, felt that an attempt should be made to interrupt this reflex pathway at some point. We were further encouraged by the finding that the reflex could be elicited under conditions of general anaesthesia, but without atropine premedication, by the inflation of a balloon in the oesophagus. She was, therefore, operated upon by Mr W. P. Cleland through a right thoracotomy incision. No gross structural abnormalities were found. Inflation of an oesophageal balloon confirmed the

FIG. 4 Block of either vagus nerve did not prevent the electrocardiographic changes produced by distending the oesophageal balloon. However, after 1.2 mg atropine given intravenously the reflex could not be elicited.

presence of the reflex even when the heart was manually held away from the oesophagus. The affected segment of the oesophagus was then mobilized and all the branches of the vagus and sympathetic nerves entering the region were divided and sealed by cauterization. It was clear, on the operating table, that this procedure completely abolished the reflex.

The postoperative course was uneventful, and when investigated four weeks after the operation, the reflex could no longer be elicited (Fig. 5). It is now two years since the operation and she remains completely asymptomatic except for a mild diarrhoea.

Discussion

There are only a few published reports in which disorders of impulse formation in the heart and its propagation have been observed in relation to swallowing. Mackenzie in 1906

reported probably the first such documented case: a patient in severe heart failure who used to develop atrioventricular block on swallowing. Since then two patients have been reported by Weiss and Ferris (1934), and one each by Iglauer and Schwartz (1936), Correll and Lindert (1949), James (1958), Deuchar and Trounce (1960), and Kopald *et al.* (1964). In spite of an otherwise normal heart, swallowing in these patients produced a variety of conduction abnormalities including sinus bradycardia, sinus arrest, varying degrees of atrioventricular block, and escape rhythms.

Like our patient, most of the reported cases came to light because of repeated Adams-Stokes attacks. The youngest reported patient, a 28-year-old woman, had symptoms since the age of 2 years (Weiss and Ferris, 1934). The remaining patients were in their 5th to 7th decades, having been symptomatic for periods ranging from 2 to 10 years.

Of the 7 reported cases, 3 had oesophageal diverticula (Weiss and Ferris, 1934; Correll and Lindert, 1949; James, 1958), 1 had achalasia of the cardia and megaesophagus (Iglauer and Schwartz, 1936), and 2 were subject to paroxysms of diffuse oesophageal spasm (Deuchar and Trounce, 1960; Kopald *et al.*, 1964). No mention has been made of any anatomical abnormalities in 1 of the 2 reported cases of Weiss and Ferris (1934). Barium meal examinations failed to reveal any structural or functional abnormality in the oesophagus of our patient.

There seems little doubt that a neurogenic reflex was responsible for the electrocardiographic changes produced by swallowing in these patients. The afferent site of this reflex has been shown to be in the oesophagus in our study and in the reports of Weiss and Ferris (1934), Correll and Lindert (1949), James (1958), and Kopald *et al.* (1964). Provocative testing with an oesophageal balloon in our patient as also in the reports of Correll and Lindert (1949) and Deuchar and Trounce (1960) has shown that the reflex can be elicited from a fairly extensive segment of the oesophagus. It seems doubtful, therefore, that the presence of oesophageal diverticula in some of the reported cases was in any way responsible for the reflex changes.

The precise nature of the abnormality in this afferent site is unknown. In the patient with diffuse oesophageal spasm reported by Kopald *et al.* (1964), oesophagomyotomy and bilateral vagotomy afforded partial relief of symptoms; however, no structural abnormalities were found in the excised segment of the oesophagus. While local anaesthesia of the mucosa did not abolish the reflex in our case,

FIG. 5 Postoperative record. Neither swallowing nor the inflation of an oesophageal balloon produced any reflex electrocardiographic changes.

it is uncertain that complete anaesthesia was, in fact, obtained.

The stimulus for exciting this reflex is also debatable. Though inflation of a balloon inside the oesophagus produces the reflex, it does not follow that actual stretching of the walls is necessary. Swallowing even small quantities of liquid or saliva has precipitated the symptoms in some of the reported cases. In our patient visible distension of the oesophagus, as seen at operation, was not essential to elicit the reflex. No evidence is available on the possible reflex effects of changes in the oesophageal wall tension.

It has been assumed, without formal proof, that the afferent pathway for this reflex is in the vagus nerves. The possible role of the sympathetic nerves in this function remains to be explored. Though splanchnic block in one case (Kopald *et al.*, 1964) did not abolish the reflex, we feel that the sympathetic block needs to be at a much higher level before any conclusions can be reached on this point; a spinal block was not considered justifiable in our patient. The term 'vagovagal reflex' often applied to this phenomenon (Weiss and Ferris, 1934; Correll and Lindert, 1949; Kopald *et al.*, 1964) is therefore, in our

opinion, best avoided, at least for the present.

The effective block produced by atropine suggests that the efferent pathway for the reflex is almost certainly in the vagus nerves. The intriguing differences between the electrocardiographic changes observed during swallowing and those occurring during phase IV of Valsalva's manoeuvre, especially the absence of any slowing of the atrial rate in the swallowing reflex, prompted us to examine the possibility of selective transmission of the efferent impulses in either of the two vagal trunks. However, unilateral vagal block failed to abolish the reflex, indicating that both vagi transmit the impulses. It was not technically feasible to obtain the response to the Valsalva's manoeuvre during selective vagal block. The differences in the responses to two stimuli (i.e. swallowing and Valsalva's manoeuvre), both of which presumably have a common efferent pathway (viz: both vagi), remain unexplained.

We were also impressed by the absence of any 'escape' beats during periods of ventricular asystole induced by swallowing. The role of the vagus nerves in suppressing pacemaker activity in the ventricles in man is conjectural. The anatomical demonstration of vagal fibres in the ventricles has been claimed recently (Hirsch, Kaiser, and Cooper, 1965), and stimulation of the vagal trunks in suitable canine preparations was shown to depress myocardial function (DeGeest *et al.*, 1965). More recently, Vassalle *et al.* (1967) have shown that after maximal vagal stimulation the period of complete suppression of impulse formation distal to the atrioventricular node is a function of the preceding heart rate; at higher heart rates maximal vagal stimulation produces longer pauses. We did not find any correlation between the period of ventricular suppression and the preceding heart rate, presumably because of the unavoidable variation in the strength of the sensory stimulus.

The most suitable treatment for these patients may be debated. Atropine, though effective in abolishing the reflex in all the reported cases, may not be acceptable to some patients because of the side effects produced by an effective dose of the drug. Digitalis glycosides should be avoided since they may

potentiate the reflex (Correll and Lindert, 1949). Sympathomimetic agents like adrenaline, isoprenaline, and ephedrine also provide symptomatic relief by preventing the reflex electrocardiographic changes (Correll and Lindert, 1949; Kopald *et al.*, 1964) or by enhancing the idioventricular rate (Weiss and Ferris, 1934).

Our patient was offered operation because of the conspicuous side effects produced by atropine, the limitations such therapy placed on her social life, and the relative insecurity inherent in the circumstances. The operation appears to have been successful.

We are grateful to Professor J. F. Goodwin for his permission to study the patient under his care and for his helpful advice. We are also grateful to Mr W. P. Cleland who operated upon the patient, and to Professor R. E. Steiner for radiological help.

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