(mEq)/l to 4.5 mmol/l. The ventricular fibrillation was unresponsive to a further 10 cardioversions. A total of 400 mg disopyramide was given but after 75 minutes of resuscitation and 15 cardioversions he remained in ventricular fibrillation. Amiodarone 200 mg was then given intravenously over 30 seconds. Two to three minutes later a final 400 J cardioversion restored sinus rhythm. A total of 600 mg amiodarone was infused over the next 24 hours and then discontinued. His course continued to be stormy but he remained in sinus rhythm and was discharged home eight weeks after his cardiac arrest.

Comment

Amiodarone has been shown to be effective when given by mouth for a wide variety of arrhythmias. There is a latent period of three to seven days before the effects are noted.²⁻³ Experience with intravenous amiodarone suggests, however, that it is rapidly effective for supraventricular tachycardias with a maximum effect 10 minutes after injection.⁴ In ventricular arrhythmias excellent results have been observed when sufficient time has elapsed for oral amiodarone to take effect. Success was reported in 34 out of 44 cases of recurrent ventricular tachycardia and in six of eight patients with recurrent ventricular fibrillation.²

Amiodarone has a protective effect against ventricular fibrillation induced by coronary artery ligation in dogs² and rats.⁵ Protection in rats was shown when the drug had been administered within two minutes by the intravenous route. Clinical experience suggests that intravenous amiodarone is also rapidly effective against ventricular ectopic beats and ventricular tachycardia success being reported in 19 out of 35 cases.⁴

Our report shows that the use of intravenous amiodarone may extend to the control of highly dangerous arrhythmias in acute cases.

We thank Mr D J Parker and Dr D Bennett for permission to report the case histories of patients under their care.

¹ Opie LH. Drugs and the heart. IV Antiarrhythmic agents. *Lancet* 1980;i: 861-8.

² Rosenbaum MB, Chiale PA, Halpern MS, *et al.* Clinical efficacy of amiodarone as an antiarrhythmic agent. *Am J Cardiol* 1976;**38**:934-44.

³ Coutte R, Fontaine G, Frank R, *et al.* Etude électrocardiologique des effets de l'amiodarone sur la conduction intracardiaque chez l'homme. Comparaison entre les voies intraveineuse et orale. *Ann Cardiol Angeiol (Paris)* 1976;**25**:543-8.

⁴ Benaim R, Uzan C. Less effets antiarythmiques de l'amiodarone injectable (a propos de 153 cas). *Revue de Médecine* 1978;**19**:1959-63.

⁵ Lubbe WF, McFadyen ML, Muller CA, *et al.* Protective action of amiodarone against ventricular fibrillation in the isolated perfused rat heart. *Am J Cardiol* 1979;**43**:533-40.

(Accepted 19 December 1980)

Cardiac Department, St George's Hospital, London SW17 0QT

J R CHAPMAN, MB, BCHIR, senior house officer

M J BOYD, MB, MRCP, lecturer in medicine

Paroxysmal atrial tachycardia provoked by swallowing

Although bradycardia after swallowing is fairly common and is due to exaggerated vagal reflex activity, deglutition tachycardia, which was first reported in 1926,¹ is uncommon and its mechanism remains unclear. Several explanations have been postulated to explain previous examples of deglutition tachycardia.²⁻⁵ We report on a patient with tachycardia induced by swallowing who underwent full evaluation.

Case report

A 52-year-old man presented with a three-month history of palpitations associated with light-headedness, pallor, and discomfort in the lower chest that occurred only on swallowing. The symptoms were worse with solids than liquids, and occurred with most meals but not at other times. He denied any other cardiac symptoms, and the only other relevant symptom was occasional dyspepsia. A barium-meal examination performed three years previously had been normal.

Clinical examination was normal, apart from mild hypertension. All routine haematological and biochemical investigations, chest-x-ray film, resting electrocardiogram, and echocardiogram were normal. Barium swallow

disclosed a small sliding hiatus hernia and absent oesophageal peristalsis. On swallowing, runs of irregular atrial tachycardia at rates of 160-220 beats/min occurred (figure). The QRS complexes of the tachycardia occasionally showed intraventricular aberration and functional atrioventricular block. The tachycardia was consistently produced by inflating a balloon within the oesophagus, using 4-20 ml of air and placing the balloon anywhere between 25-45 cm from the incisors. With large volumes the tachycardia was initiated immediately the balloon was inflated and continued until deflation.

Episode of paroxysmal atrial tachycardia provoked by swallowing (arrow indicates initial act of swallowing).

The effects of various physiological and pharmacological manoeuvres were studied with the balloon placed 30 cm from the incisors. Vertical tilt to 60° reduced the response to balloon inflation but shortened the average duration of the tachycardia cycle from 320 ms to 275 ms. Both quinidine and propranolol appreciably reduced the response to balloon inflation and increased the average duration of the tachycardia cycle to 375 and 400 ms respectively. Temporary right stellate ganglion block, atropine, and carotid sinus massage had no effect. Disopyramide totally abolished the tachycardia response to balloon inflation.

An intracardiac study showed normal conduction intervals, sinus node function, and refractory periods. In response to oesophageal balloon inflation tachycardia arose in the high lateral right atrium but could not be initiated by programmed extrastimulation.

Comment

Physiological and pharmacological evaluation of this uncommon form of tachycardia was facilitated by using a simple and reproducible method of provoking tachycardia. Because the tachycardia could be provoked at will an effective form of antiarrhythmic treatment was easily and rapidly designed and assessed. As in this case, it may be apparent from the clinical history which methods of initiating tachycardia should be evaluated before having recourse to invasive stimulation techniques.

The mechanism of deglutition tachycardia remains in dispute. The repetitive and reproducible nature of the tachycardia and its fixed relation to the act of swallowing suggest either a direct mechanical effect or a neural reflex. In this case a direct mechanical effect was suggested by the abnormal oesophagus and the fact that the tachycardia was initiated immediately by balloon inflation and sustained throughout the inflation but was relatively short lived in response to swallowing. The tachycardia arose in the right atrium and was initiated by stimulation of almost the entire oesophagus, so a direct effect on the left atrium is therefore unlikely. The lack of effect of atropine, right stellate ganglion block, and carotid sinus massage suggests that neural reflex mechanisms are not important. The dramatic effect of tilting and the appreciable effect of beta-blockade, however, favour a neural mechanism affecting the sympathetic limb of the autonomic nervous system.

¹ Sakai D, Mori F. Über einen Fall von sog 'Schlucktachykardie'. *Z Gesamte Exp Med* 1926;**50**:106-9.

² Engle TR, Laporte SM, Meister SG, Frankle WS. Tachycardia upon swallowing. Evidence for a left atrial automatic focus. *J Electrocardiol* 1976;**9**:69-73.

³ Lindsay AE. Tachycardia caused by swallowing: mechanisms and treatment. *Am Heart J* 1973;**85**:679-84.

⁴ Bajaj SC, Ragaza EP, Silva H, Goyal RJ. Deglutition tachycardia. *Gastroenterology* 1972;**62**:632-35.

⁵ Kramer P, Harris L, Kaplan R, Hollander W. Recurrent supraventricular paroxysmal tachycardia precipitated by swallowing. *Proceedings of the New England Cardiovascular Society* 1962-3;**21**:21.

(Accepted 9 January 1981)

St Bartholomew's Hospital, London EC1A 7BE

R S BEXTON, MA, MRCP, research registrar

A W NATHAN, MB, MRCP, registrar

K J HELLESTRAND, MB, FRACP, research registrar

A J CAMM, MB, MRCP, consultant physician