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SHORT REPORTS

Successful emergency transoesophageal cardiac pacing with subsequent endoscopy

Transoesophageal cardiac pacing was described in 1969.[1] Since the introduction of temporary transvenous pacing, however, it has not been used routinely in clinical practice. The recent introduction of a specially designed flexible electrode and pulse amplifier has allowed the technique to be used rapidly in asystolic patients before inserting an intracardiac pacemaker. The equipment and technique have been described.[2]

The condition of the oesophagus using earlier equipment was assessed in necropsy studies on patients and dogs.[1 3] We can find no record of the endoscopic appearances of the oesophagus after using the flexible electrode. We therefore report the successful use of transoesophageal pacing and describe the endoscopic findings both at two days and at two months.

Case report

A 71-year-old woman was admitted after feeling dizzy at home. Her general practitioner had recorded a pulse of 20 beats/min. She was known to have had dizzy episodes but had not lost consciousness. An electrocardiogram had shown left bundle-branch block. On admission electrocardiography showed complete heart block. While the transvenous pacing was being prepared the patient developed asystole and became unconscious. A Vygon bipolar oesophageal electrode (Oesocath No 1128-02) was inserted transnasally to its full length. The battery-powered demand pacing box and Vygon Oesocath pulse amplifier were connected, giving a 30 V direct current pacing impulse. Pacing blips were seen on the cardiac monitor. The electrode was then withdrawn until ventricular capture occurred; this procedure was completed in less than 30 seconds. The patient regained consciousness within minutes but complained of chest pain associated with the pacing complexes. Over the next 30 minutes transvenous pacing was established and transoesophageal pacing discontinued. She remained fully conscious and had no further arrhythmia. Oesophagogastroduodenoscopy was performed 45 hours later. No evidence of any erythema, erosions, or ulceration was found. The patient was subsequently given a permanent pacemaker. Two months later she complained of mild dyspepsia; appearances on repeat oesophagogastroduodenoscopy were again completely normal.

Comment

This is only the second report of successful resuscitation of patients in asystole by emergency pacing with the new specifically designed electrodes via the oesophagus. This case demonstrates the speed of the technique and the rapidity with which the patient may recover. Our patient experienced severe chest pain, which may have been due to oesophageal contraction stimulated by the pacing impulse. Most patients who have a cardiac arrest do so after a myocardial infarction and are thus unsuitable for invasive procedures. This was not the case with our patient. Oesophagogastroduodenoscopy showed no evidence of any damage to the oesophagus.

Our experience of this new technique is limited but it seems to be a rapid and safe method of beginning resuscitation in asystolic patients. It allows subsequent intracardiac pacing to be performed with adequate time for good technique. Thus, like Hale *et al*,[2] we consider that transoesophageal pacing has a definite place in an emergency.

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Toxocaral and toxoplasmal antibodies in cat breeders and in Icelanders exposed to cats but not to dogs

Surveys using the immunosorbent assay (ELISA) technique have shown that 2·6% of healthy adults have toxocaral antibody in their plasma, indicating past or present infection. Woodruff *et al*[1] however, found toxocaral antibody in 16 of 102 exhibitors at a large dog show. The question arises whether cat breeders and owners are similarly infected. We sought an answer to this with the co-operation of exhibitors at the 1979 Bedford and district cat club show. Epidemiological data were also obtained from examination of sera from 307 adult Icelanders. In Iceland dogs have been prohibited for 40 years to control hydatid disease but there has been no restriction on ownership of cats, of which there is a large population.

Subjects, methods, and results

Questionnaires were answered voluntarily by 67 cat breeders or owners exhibiting at the 1979 Bedford show and blood was obtained by venepuncture at the same time. Serum was separated immediately and stored at 4°C until examined by the ELISA technique of de Savigny *et al*.[2] Sera obtained from healthy blood donors in Iceland were examined in the same way as those from 922 healthy adults obtained by courtesy of the Brentwood and Milton Keynes district blood transfusion services. ELISA values of 0·5 or more indicate past or current infection. The toxoplasmal antibody estimations were carried out using the dye test.

Toxocariasis—Out of the 67 cat breeders and exhibitors, only one gave a positive response to the ELISA test (1·5%), compared with 24 (2·6%) of the 922 blood donor controls. The mean age of the cat breeders and exhibitors was 38·4 years (±SD 11·5) and that of the healthy controls 34·5 years

(±SD 12·1). Most (39) of the cat breeders and exhibitors lived in cities or towns. At the time of the survey the exhibitors owned 455 cats (mean 6·6); 12 owned more than 10 cats each, and 44 had lived with cats for more than 10 years. Their exposure to cats and to a feline environment was therefore substantial. Moreover, 14 either never wormed their animals or only rarely did so. The evidence therefore indicates that substantial exposure to cats is not associated with a higher prevalence of toxocariasis than is found in the general public. The results of this study were reinforced by the study of the Icelanders (mean age 31·1 years (±SD 8·4)), none of whom gave a positive response to the toxocara ELISA test.

Toxoplasmosis—In Britain 10-40% of persons aged 11 to 40 years have titres of 1/16 or more on toxoplasma dye testing (Dr D G Fleck, Toxoplasma Reference Laboratory, London SW17 0QT). In comparison 24 of the 67 cat breeders (35·8%) and 38 of 208 (18·3%) of the Icelanders from whom there was sufficient serum to test for toxoplasmosis after testing for toxocariasis had titres of 1/16 or more, a highly significant difference ($\chi^2 = 8·9$, $n = 1$, $p < 0·01$).

Comment

Toxocariasis—Our study indicates that cat owners do not acquire toxocaral or toxoplasmal infection more frequently than the general British population, and cats in a community such as that of Iceland do not give rise to detectable toxocaral infection. This contrasts with dog exhibitors, of whom six times as many[1] have toxocaral antibodies compared with the general population (15·7% *v* 2·6%). The difference between those exposed to the canine as compared with the feline environment probably results from the cat's practice of burying its faeces and its more domestic habits. Thus cats are seldom found in public parks and, unlike dogs, do not go with their owners for walks into public parks and there deposit their faeces. The severity of the Icelandic winter does not prevent survival of toxocaral ova in the soil, as they survive the more severe winter in Montreal: Ghadirian *et al*[3] carried out a study there similar to that of Borg and Woodruff,[4] who found that 24% of soil samples in public parks in Britain contained the ova. It is also important to note that the toxocara ELISA detects the presence of antibody to *Toxocara cati* and *Toxocara canis*.

Toxoplasmosis—The 18·3% of Icelandic subjects who yielded titres of 1/16 on toxoplasma dye testing is similar to that found in other Icelandic surveys. The proportions of subjects with such titres in the groups studied here fell within the range expected among the general population in Britain, but that among the cat breeders was near the upper limit of the normal range. Why should contact with cats lead to toxoplasmal but not toxocaral infection? Cats excrete resistant and highly infective oocysts within three to 10 days of ingesting *Toxoplasma gondii* cysts from tissues of animals. Infective forms of *Toxoplasma gondii* are readily disseminated from floors and concrete areas[5] and do not depend on soil for transmission to the same extent as do ova of *Toxocara cati* or *Toxocara canis*. Cats infected with toxoplasma thus cause more infective pollution of the environment than cats infected with *Toxocara cati*.

Cats clearly constitute a much lesser risk to the general population than do dogs in respect of transmission of toxocaral infection but not in respect of toxoplasmosis.

We thank Dr O Jensson, Reykjavik Blood Bank, for collecting the Icelandic sera; Drs H H Gunson and J Harrison, of the Blood Transfusion Service; and Dr A H Balfour, of Leeds Public Health Laboratory Service, for performing the toxoplasmal dye tests.

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Relief from digital vasospasm by treatment with captopril and its complete inhibition by serine proteinase inhibitors in Raynaud's phenomenon

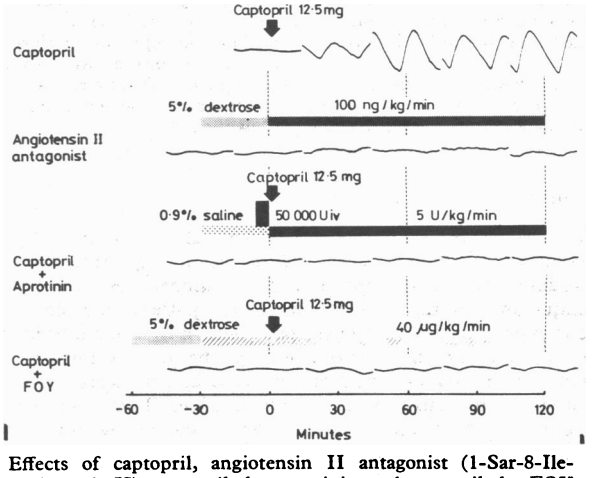
A patient with Raynaud's phenomenon was successfully treated both immediately and long term with the angiotensin-converting enzyme inhibitor captopril.[1] The acute vasodilator action of captopril was completely blocked by the serine proteinase inhibitors, aprotinin, and ethyl-p-(6-guanidino hexanoyloxy) benzoate methanesulfonate (FOY),[2] whereas infusion of angiotensin II antagonist (1-Sar-8-Ile-angiotensin II) had no effect on digital blood flow but merely an agonistic action on the systemic blood pressure. This suggests that captopril may be a promising candidate for the treatment of Raynaud's phenomenon and, more interestingly, that the vasodilator action of captopril in Raynaud's phenomenon may be caused mainly by accumulated circulating kinins, not by the inhibited production of angiotensin II.

Case report

A 51-year-old Japanese woman visited the outpatient clinic in February 1980 for recurrent episodes of pallor of the fingers and toes which had gradually increased in frequency and duration since onset in October 1977. Her Raynaud's phenomena, though the duration was less than three years, were considered to be primary according to Allen's criteria for Raynaud's disease, and physical and laboratory examinations were suggestive of no other diseases.

The patient was kept recumbent throughout the experiments in a room with a controlled temperature of 20-21°C with approximately 50% humidity. Photoplethysmography of the second finger of the right hand and second toe of the right foot, blood pressure, and heart rate were monitored at five minute intervals. After one hour, when she had adapted to the room temperature, she was treated according to four different protocols. Firstly, 12·5 mg of captopril was given by mouth. Secondly, angiotensin II antagonist was infused at a rate of 100 ng/kg/min. As blood pressure rose by 32 mm Hg systolic and 16 mm Hg diastolic 10 minutes after the infusion was started, the infusion rate was not increased and was fixed at that dose for two hours. In the next two experiments serine proteinase inhibitors, aprotinin and FOY, were used after confirming that no change in the digital circulation occurred by infusion of the solvent alone. Thirdly, aprotinin (50 000 U) was first given intravenously and then by infusion at a rate of 5 U/kg/min for two hours. Captopril 12·5 mg was given by mouth after the bolus injection of aprotinin (50 000 U). Fourthly, after FOY was infused at a rate of 40 μg/kg/min for 30 minutes captopril (12·5 mg) was given by mouth. FOY was continuously infused for two hours more thereafter.

The figure shows that administration of captopril by mouth resulted in appreciable improvement in the digital circulation, subjectively, objectively, and plethysmographically. Digital spasm neither disappeared nor worsened with infusion of angiotensin II antagonist, and only an agonistic action was shown in the systemic blood pressure with such a low dose. Digital vasodilation by captopril was completely inhibited by the serine proteinase inhibitors aprotinin and FOY. After these tests she was treated with oral captopril in a dose of 37·5 mg daily for several months with remarkable improvement.



Effects of captopril, angiotensin II antagonist (1-Sar-8-Ile-angiotensin II), captopril plus aprotinin, and captopril plus FOY on digital photoplethysmography in a patient with Raynaud's phenomenon.