mg dicobalt edetate over three minutes and, as his condition did not improve, a further 300 mg again over three minutes.

He then developed periorbital oedema, which was thought to have been caused by immersion in the hot vat rather than to be a reaction to the drug. He was given 200 mg hydrocortisone intravenously, and laryngoscopy after 20 mg etomidate showed early laryngeal oedema. Intubation was performed using an orotracheal cuffed Portex 9·0 tube (under suxamethonium relaxation and with cricoid pressure). Laryngoscopy 60 seconds later showed gross laryngeal oedema obscuring the cords and epiglottis. Immediately after successful atraumatic intubation gentle tracheal suction produced copious pink frothy fluid.

He received sodium bicarbonate 100 mmol(mEq)/l intravenously to correct his acidosis (pH=6·9, base excess=−20 mmol(mEq)/l), further arterial samples were taken (from a left radial arterial catheter), and a catheter was inserted in the right internal jugular vein to assess central venous pressure. There was still no improvement, and three ampoules of amyl nitrite were broken into a reservoir bag and administered while sodium thiosulphate and sodium nitrite were given intravenously.

Clinical evidence of pulmonary oedema was now overwhelming, and intermittent positive pressure ventilation was started using a Manley Servovent and pancuronium bromide and papaveretum to aid ventilation. Effective ventilation required positive end expiratory pressure of 5 cm H_2O. A catheter was inserted, 450 ml of contaminated urine drained, and the catheter left in situ to measure urine output. After gastric lavage 100 ml thiosulphate solution was left in his stomach. Blood gas tensions, cyanide and glucose concentrations, and packed cell volume were measured, and he was transferred to the intensive care unit.

He had severe facial oedema, and it was impossible to assess his pupillary reflexes or open his mouth. His central venous and blood pressure were satisfactory, but he showed peripheral vasoconstriction and gross plasma extravasation (packed cell volume 0·75, haemoglobin concentration 200 g/l, white cell count 40×10^9/l and had a low urine output (0·5 ml/kg). Four litres of human plasma protein fraction were transfused over two hours with no change in his central venous pressure (+8 cm H_2O). His urine output returned to normal, and his peripheral perfusion improved. His blood glucose concentration rose to 16 mmol/l (288 mg/100 ml) over six hours, and he required an infusion of insulin and potassium to re-establish a normal concentration.

After 16 hours his neuromuscular blockade was reversed and he was found have regained awareness during insertion of the catheter and to have full recall from that time. He was not, however, distressed and tolerated intubation for another 36 hours until extubation was considered to be safe (due to his slowly resolving oedema).

He recovered completely from immersion in cyanide and the effects of his specific antidote, dicobalt edetate.

Comment

Subsequent review showed that gross oedema after treatment with dicobalt edetate is not unusual,[1 2] and it has been recommended that dicobalt edetate be used only in severe cases when patients do not respond to repeated doses of thiosulphate and nitrites. When dicobalt edetate is being used its potentially dangerous effect should be borne in mind and facilities for intubation and resuscitation should be immediately available.

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Is cardiac ultrasound mandatory in patients with transient ischaemic attacks?

A cerebral embolus is a much dreaded complication of cardiac disease as it often occurs unexpectedly in patients whose symptoms are few and whose prognosis is otherwise good.[1] Emboli from within the heart are well recognised as a frequent cause of stroke and there is growing evidence that occult structural cardiac abnormalities are a main cause of cerebral and retinal transient ischaemic attacks.[2-4] This has led to the routine use of echocardiography in all such patients. Echocardiography is a non-invasive technique but requires considerable investment in equipment and needs skilled operation and interpretation of results, neither of which may be readily available.

We have examined the diagnostic yield of echocardiography and tried to establish whether this investigation provides new information of importance to the management of these patients.

Patients, methods, and results

Two hundred consecutive patients who had been investigated for cerebral or retinal transient ischaemic attacks (focal neurological episodes lasting less than 24 hours) at the National Heart Hospital non-invasive laboratory between 1982 and 1985 form the basis of this report. Cases were referred from within the hospital (n=78) and from other neurological centres because of symptoms. A clinical history and examination, 12 lead electrocardiogram, and chest radiograph were obtained in each patient. None had known evidence of cerebrovascular disease.

M mode and cross sectional echocardiograms were recorded on Hewlett-Packard phased array apparatus (model 77020A) with a 3·5 or 5·0 mHz transducer. Complete echocardiographic studies using parasternal, subxiphoid, suprasternal, and apical views were routinely obtained and recorded on 12·7 mm video tape for later analysis.

In 106 cases abnormalities were found on physical examination or in the chest radiograph or electrocardiogram or both (group 1) which led to a diagnosis that was confirmed by echocardiography in valvular cases (table). In no case was mitral valve prolapse or valvular disease diagnosed by echocardiography in the absence of the appropriate physical signs elicited by the referring physician. Coronary artery disease was found in 23 patients (history and electrocardiogram), and though two of these had left ventricular thrombus and seven aneurysms, 16 had a normal echocardiogram. The other causes of transient ischaemic attacks included cardiomyopathy in six patients, myxoma in one, endocarditis in six, and valve replacement in 17. One patient in this group had a suspected aortic valve mass as the cause of transient ischaemic attacks; at exploratory thoracotomy, however, no mass was found.

Distribution of abnormal and normal cross sectional echocardiograms from 106 patients with clinical signs suggestive of cardiac source of emboli (group 1) and 94 patients with no clinical signs suggestive of cardiac source of emboli (group 2)

	Abnormal	Normal	Total
Group 1	79*	27	106
Group 2	1	93	94

*Includes one false positive result (see text).

Hypertension was present in 20 patients, nine of whom showed no evidence of left ventricular hypertrophy. The remaining 94 patients showed nothing abnormal on physical examination or in the electrocardiogram or chest radiograph (group 2). In 93 cases the echocardiogram confirmed the absence of structural cardiac disease, but in one the echocardiogram showed a pedunculated mitral valve papilloma, which was confirmed pathologically (table).

Comment

Intracardiac masses and valvular disease are a common and potentially the most easily prevented cause of transient ischaemic attacks and stroke.[2-4] In patients with established valvular disease or valve replacement (with or without infective endocarditis) we found that there was no additional evidence to be gained from echocardiography and that treatment must rest on clinical grounds. When hypertension was the only abnormality the echocardiogram was unhelpful. In patients with coronary artery disease the presence of a left ventricular aneurysm or intracavity thrombus may possibly be the cause of emboli, but in our series this was not an unexpected finding as each patient had previously suffered a myocardial infarction and had cardiomegaly. This was an uncommon problem, however, as these patients represented 23 of 742 with coronary artery disease (without transient ischaemic attacks) studied during the period, of whom 188 had evidence of left ventricular aneurysm or thrombus; this group of patients requires further study. In patients without clinical evidence of cardiac disease the use of echocardiography presents a clinical dilemma. Presumably all patients under 50 with unexplained transient ischaemic attacks should undergo cardiac ultrasound. The diagnostic yield of such investigations is very low (0·5%), however, and, given an estimated unit cost of £200, each unexpected diagnosis costs £40 000, which needs to be weighed against the costs of a completed stroke in a young patient.

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Psoas muscle hypertrophy: mechanical cause for "jogger's trots?"

Although gastrointestinal disturbances occur in up to 30% of recreational or competitive runners,[1] they are poorly recognised by the medical profession, and the mechanism of their production remains speculative.[2 3] We report a case where "jogger's trots" may have been produced by mechanical compression of the colon.

Case report

A 36 year old man presented with a three year history of frequent bowel motions since starting competitive running. During training (10 miles (16 km), five times weekly) and competitive marathons he had the urge to defecate every 30 minutes; he passed formed stools without blood, mucus, or abdominal pain. He had less frequent bowel motions during fell running than road running, and cycling did not cause an increase in frequency. He had no other symptoms. When resting he produced two formed stools daily.

Results of physical examination, including sigmoidoscopy, were normal. Stools were formed and without occult blood. The following investigations gave normal or negative results: full blood count; plasma viscosity; liver function tests; urea and electrolyte concentrations; serum calcium, thyroxine, vitamin B_{12}, and folate concentrations; stool culture; three day faecal fat concentration; jejunal biopsy, including disaccharidase enzymes; and barium follow through. A profile of gut hormones was not performed. Abdominal ultrasonography showed grossly enlarged psoas muscles occupying half of the anteroposterior diameter of the abdomen. Barium enema showed reproducible extrinsic compression of the colon by both psoas muscles during flexion of the hip (figure).

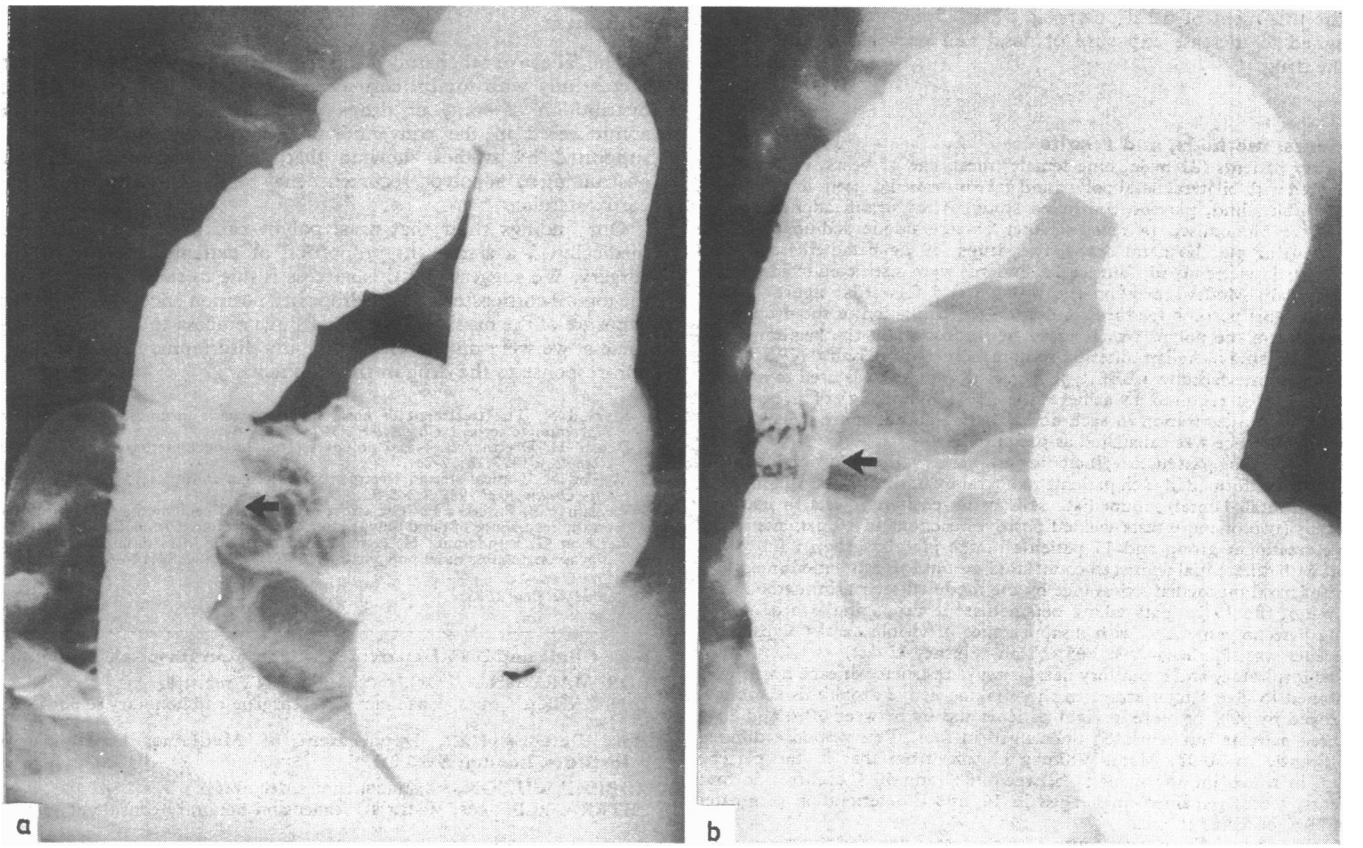
The median whole gut transit time of 50 faecal markers contained in a standard test meal[4] was 47·3 hours at rest and 44·5 hours after a 32 kilometre run. All markers were passed by 75·5 and 82·0 hours, respectively, and total weights of stools were 824·6 and 882·5 g, respectively.

His symptoms were controlled by not eating food on days of races and taking loperamide one hour before running.

Comment

The cause of gastrointestinal symptoms during running is unknown, but excessive parasympathetic activity is unlikely to be responsible as it is persistent in the trained athlete while diarrhoea is intermittent. Severe symptoms usually occur after a rapid increase in the amount of training or particularly severe exertion[2] when it is postulated that hypovolaemia and splanchnic vasoconstriction lead to ischaemia of the gut. This in turn may produce bloody diarrhoea and symptoms that mimic acute appendicitis or Crohn's disease.[3] Hormonal or vascular changes might produce diarrhoea by speeding intestinal transit or increasing intestinal secretion, but neither mechanism occurred in our subject as transit time and weights of stools were similar at rest and after exercise. Similarly, effects of stress during competition would not account for symptoms during training.

The gross psoas muscle hypertrophy in our patient led to obvious colonic compression during flexion of his hip, and this continuous mechanical message may have produced diarrhoea by emptying the colonic contents without increasing total transit or intestinal secretion. Interestingly, his symptoms were reduced by decreasing his intake of food, were worse during road running when mechanical jarring



Radiographs showing compression of ascending colon (arrow) during flexion of right hip—(*a*) extension, (*b*) flexion.