ally.¹ Enterotoxin, detected by its cytopathic effect on Hep2 cells in tissue culture and neutralisable by *Cl sordellii* antitoxin (Wellcome Foundation Research Laboratories), was found in the stool in high titre (table). Treatment was instituted with oral metronidazole 400 mg thrice daily and continued for 10 days. Within 36 hours the stool no longer contained visible blood and within three days was well formed. Sigmoidoscopy one week after beginning metronidazole showed a normal mucosa. After treatment the patient remained asymptomatic with no demonstrable *Cl difficile* organisms or toxin in the stool.

Stool cultures for Clostridium difficile and neutralisable Cl difficile toxin titres throughout metronidazole treatment

Duration of metronidazole treatment	Stool culture for <i>Cl difficile</i>	Neutralisable <i>Cl difficile</i> toxin titre in stool*
Pretreatment	Positive	1/1000
2 days	Positive	1/500
4 days	Negative	Nil
10 days	Negative	Nil
2 weeks after treatment	Negative	Nil

*One gram wet-weight stool extracted in 5 ml physiological saline.

Comment

Acute colitis, with or without pseudomembrane formation, associated with *Cl difficile* enterotoxin is an increasingly recognised complication of antibiotic treatment.² Lincomycin and clindamycin have been especially incriminated, but the role of other antibiotics including tetracycline, Co-trimoxazole, the cephalosporins, and the penicillins (particularly ampicillin) has also been well documented.³ This case, in which oral neomycin is incriminated, re-emphasises the potential hazard of broad-spectrum antibiotics, especially when given specifically to alter the colonic bacterial flora and for hepatic failure. The dramatic response to oral metronidazole, with disappearance of organism and toxin together with symptomatic improvement, strengthens the case for using oral metronidazole in treating *Cl difficile*-associated colitis.⁴

I thank Dr R E Barry for permission to report this case and Dr P D Walker (Wellcome Foundation Research Laboratories) for providing *Cl sordellii* antitoxin.

¹ Hafiz, S, and Oakley, C L, *Journal of Medical Microbiology*, 1976, **9**, 129.

² *British Medical Journal*, 1979, **2**, 349.

³ George, W L, et al, *American Journal of Clinical Nutrition*, 1979, **32**, 251.

⁴ Pashby, N L, Bolton, R P, and Sherriff, R J, *British Medical Journal*, 1979, **1**, 1605.

(Accepted 27 September 1979)

University Department of Medicine, Bristol Royal Infirmary, Bristol BS2 8HW

R P BOLTON, MA, MRCP, medical registrar

Syncope after running

Exertional hypotension is often seen in patients with various cardiac disorders¹ including severe coronary artery stenosis,² and in normal subjects during maximal exhaustive exercise. We describe a patient with mild ischaemic heart disease who developed syncopal episodes after running and a definite fall in blood pressure accompanying bradycardia after exercise.

Case report

A 48-year-old trained soldier was admitted on 7 May 1979 because of syncopal episodes after running. In the seven years before admission he had had several episodes of syncope while he was standing still after running 1-2 km, especially on cold mornings; he did not suffer syncopal attacks when he jogged instead of suddenly stopping running. Two years earlier he had been found to have an abnormal electrocardiogram, suggesting ischaemic heart disease, but he had no chest pain, palpitations, or shortness of breath even after running. He took no medication and continued his work and physical training. On examination he was well nourished and looked healthy. He was 163 cm tall and weighed 61 kg. His blood pressure was 100/58 mm Hg in the right arm and 100/56 mm Hg in the left. There was no orthostatic hypo-

(a) Fall of blood pressure with bradycardia after augmented 2-step test. (b) Disappearance of fall in blood pressure after pindolol.

tension. Physical examination showed no abnormality except a grade 2 systolic murmur at the apex. Peripheral vessels were easily palpable and no bruit was heard. There was no neurological abnormality. Laboratory values were all normal, but he was positive for hepatitis B surface antigen.

At cardiac catheterisation the left ventriculogram and left ventricle end-diastolic pressure were normal. Coronary arteriography showed 50% stenosis of the left circumflex artery close to its origin and 25% stenosis in the right coronary artery. Direct brachial artery pressure measurement using a telemonitor showed a significant fall in blood pressure from 152/64 mm Hg during exercise (Master augmented 2-step test) to 100/40 mm Hg immediately after ceasing exercise. This lasted for 10-15 cardiac cycles and, after a transient increase, blood pressure then fell to 80/12 mm Hg with bradycardia for about one minute, when he felt light-headed but no syncope occurred (see figure, a). The fall of blood pressure after exercise disappeared by giving pindolol, 20 mg orally (figure, b).

His response to Valsalva manoeuvre, cold pressor test, and mental arithmetic test was normal. Plasma norepinephrine, epinephrine, and dopamine concentrations were, respectively, 260 ng/l, 25 ng/l, and 115 ng/l in a supine position and 706 ng/l, 98 ng/l, and 278 ng/l in an upright position for 10 minutes. Blood gas, plasma lactate, and plasma pyruvate values before and after exercise were normal.

Comment

Thomson and Kelemen³ stated that a fall in systolic pressure during exercise is a sign that the left ventricular blood supply is severely compromised. Levites *et al*² found hypotension during exercise testing in 2.7% of the patients they tested, but the extent and distribution of coronary lesions were no different from those of patients with a normal blood pressure response to exercise. In their patients hypotension occurred during exercise but not after it. In our patient the extent of coronary artery narrowing was not significant and seemed unlikely to have caused the syncope and fall in blood pressure after exercise. His normal response to the Valsalva manoeuvre indicated an intact baroreceptor reflex arch. Another possible explanation of the syncope is a vasodepressor syncope associated with exercise. A beta-blocker prevented the fall in blood pressure and bradycardia in this patient, which suggested that the syncope may have been caused by a β -receptor-mediated neurogenic or metabolic mechanism induced by exercise.

¹ Bruce, R A, et al, *Circulation*, 1959, **19**, 543.

² Levites, R, Baker, T, and Anderson, G J, *American Heart Journal*, 1978, **95**, 747.

³ Thomson, P D, and Kelemen, M H, *Circulation*, 1975, **52**, 28.

(Accepted 2 October 1979)

Department of Internal Medicine, Institute of Constitutional Medicine, University of Kumamoto, Kumamoto, Japan

ETSURO TSUTSUMI, MD, physician

HIROSHI HARA, MD, physician