immediately on standing after a period of rest or when walking about. These symptoms disappeared between the fifth and tenth days after the last dose of propranolol. There were no associated complaints of angina, sweating, tremor, or agitation.

Comment

Long-term treatment with propranolol lowers the blood pressure and increases the plasma noradrenaline concentration, probably due to baroreflex stimulation resulting from the fall in cardiac output. Sudden withdrawal of propranolol produced a steady reversal of these features over four days. There was no evidence of excessive sympathetic activity during the withdrawal phase. Our results do not rule out a moderate degree of receptor hypersensitivity on withdrawing long-term beta-blockade, which may have been partly responsible for the heart rate increase 48 hours after the last dose, but these changes were slight.

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Penicillamine causing acute colitis

We report a case of acute colitis in a patient with rheumatoid arthritis who was being treated with penicillamine.

Case report

A 61-year-old woman with a 30-year history of classical, seropositive rheumatoid arthritis began taking penicillamine, 125 mg twice daily, in April 1978 because of active, progressive disease. Other treatment consisted of indomethacin suppository, 100 mg at night, and methyl dopa, 250 mg twice daily, for mild hypertension. In May the penicillamine was increased to 125 mg thrice daily and by June she had improved considerably, with no signs of active disease or adverse side effects from the penicillamine. On 7 August 1978, four and a half months after starting penicillamine, she developed colicky abdominal pain relieved by defecation, tenesmus, and profuse diarrhoea containing fresh blood and mucus with increasing frequency by day and night. She stopped the penicillamine three days before being admitted to hospital on 21 August.

On examination she was afebrile and had a pulse of 80/min, sinus rhythm, and blood pressure 150/90 mm Hg. There were signs of chronic, severe rheumatoid disease that was clinically inactive. Abdominal examination disclosed a distended caecum with tenderness over the descending colon. Bowel sounds were increased. Rectal examination was extremely painful, but no other abnormality was detected.

Investigations showed haemoglobin 11.7 g/dl; white cell count $7.3 \times 10^9/l$ ($7300/mm^3$), normal differential; platelets $210 \times 10^9/l$ ($210\,000/mm^3$); erythrocyte sedimentation rate 30 mm in first hour; differential agglutination test for rheumatoid factor positive at titre of 1/64. Liver function tests showed albumin 30 g/l but were otherwise normal. Analysis of urea and electrolytes showed potassium 3 mmol(mEq/l) but was otherwise normal. Tests for occult blood in faeces were positive. No pathogens were isolated on faecal culture. A plain abdominal x-ray film taken on admission was normal. Sigmoidoscopy to 10 cm showed a friable, inflamed mucosa with contact bleeding and flecks of pus. Mucosal biopsy showed inflammatory changes. Double-contrast barium enema gave normal results.

Indomethacin suppositories were stopped on admission but the methyl dopa was continued, and dextropropoxyphene and paracetamol (Distalgic) was used to control her pain. Her diarrhoea settled, and a repeat sigmoidoscopy one week after admission was entirely normal. Her rheumatoid arthritis became active again, and on 2 September she restarted penicillamine, 125 mg twice daily. Eleven days later she developed bloody diarrhoea and stopped the penicillamine after suffering symptoms for three days. Her bowel

function recovered completely over the next two days, but in the ensuing three weeks her rheumatoid disease became active and she began intramuscular gold, 50 mg weekly. Her bowel symptoms have not returned, and her rheumatoid disease is coming under control after three months of chrysotherapy.

Comment

Acute colitis occurring in patients taking penicillamine has not been reported. The drug has many common gastrointestinal side effects, including nausea and vomiting, hypogeusia, indigestion, and possibly upper gastrointestinal bleeding¹ and diarrhoea.² The temporal association between taking the drug and developing the colitis, and the fact that symptoms recurred after rechallenge with penicillamine suggest strongly a direct causal relation. Although indomethacin suppositories cause rectal bleeding^{3,4} and ulcerative colitis may be associated with oral indomethacin,⁵ we think that indomethacin was not responsible for the symptoms in this patient, as she had been using indomethacin suppositories for many years without ill effect and did not resume using them after the initial episode, yet the colitis still recurred. She has subsequently restarted indomethacin suppositories and had no return of her acute bowel symptoms, but she still complains of occasional loose motions. Sigmoidoscopy and rectal biopsy performed in April 1979 were entirely normal. We conclude, therefore, that this was a case of penicillamine-induced acute colitis that remitted rapidly when the drug was stopped.

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Relief of causalgia in limbs by regional intravenous guanethidine

Causalgia is a rare and intractable complication of injury to major peripheral nerves characterised by severe burning pain. Sympathetic block is the only consistent way of relieving this type of pain, and I have found that using guanethidine in an intravenous regional technique after applying a tourniquet has considerable advantages compared with repeated local-anaesthetic ganglion blocks. I report using guanethidine in 10 consecutive patients with causalgia affecting the upper limb.

Patients, methods, and results

Causalgia was associated with damage to the ulnar nerve in five patients (four after surgery for ulnar-nerve transposition and one after a bullet wound); the median nerve in four patients (two after slashing wounds, one after a bullet wound, and one after surgery for carpal-tunnel decompression); and the brachial plexus in one patient after radiotherapy. The patients were aged between 21 and 70 and had failed to respond to various kinds of treatment before their referral to this centre.

Routine guanethidine blocks were carried out,^{1,2} each patient receiving two blocks at an interval of three weeks. Pain relief was scored on a simple scale of 0-3 and the figure summarises the results. Though the first block produced considerable relief, the effects of the second were better and lasted longer. The patients fell conveniently into two equal groups according to the duration of their causalgia, the average durations being five months and 15 months from onset. Pain relief was superior in those treated earlier.

Comment

None of the patients treated early required further blocks, whereas those treated after an average of 15 months did. All patients, however, affirmed that life had become tolerable by the end of the period of

Pain relief after regional intravenous blocks with 20 mg of guanethidine made up to 30 ml with physiological saline in five patients treated after average of five months' causalgia and five treated after average of 15 months. Arterial tourniquet on for 10 minutes.

study. The success of the guanethidine technique has important implications for understanding causalgia and related types of sympathetic dystrophy.^{3,4}

I suggest that surgeons should take the opportunity of blocking the sympathetic nerve supply to an extremity with guanethidine before the tourniquet is released after elective or emergency operations on major peripheral nerves whenever there is good reason to fear the onset of causalgia in the affected limb. The increased blood supply will also help to promote healing generally in traumatised tissues.

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Type A behaviour and coronary heart disease

Type A behaviour, first associated with coronary heart disease (CHD) in America in 1959,¹ is characterised by competitiveness, ambition, hurriedness, and impatience and is traditionally assessed by interview or a lengthy "activity-survey" questionnaire.² A short, 14-item questionnaire was described by Bortner in 1969³ and subsequently validated against other measures of type A behaviour.⁴ I report the relation between type A behaviour (assessed by the Bortner questionnaire) and CHD in a sample of British men.

Subjects, methods, and results

A population-based case-control study was mounted among the participants of the UK Heart Disease Prevention Project, a large controlled trial in industry of the multifactorial prevention of CHD in middle-aged men.⁵ Cases were participants who had had a myocardial infarction (according to WHO criteria) and were each matched with a control subject in the same factory who was closest in age and of the same marital state, with no evidence of CHD on symptom history or electrocardiography. Subjects receiving treatment for hypertension were excluded.

The Bortner questionnaire was completed independently by each subject: it has 14 items, each comprising two statements separated by a horizontal line of fixed length. One of the statements represents the type A pattern and the other the opposite, or type B. The subject is asked to draw a vertical line across the horizontal one at the point where he considers himself to lie between the two extremes. A score is obtained for each item by measuring

from the B end, and the total Bortner score is derived by adding up the scores of all 14 items. The maximum score for each item is 24; the higher the score the more type A is the behaviour pattern.

I studied 50 pairs of subjects, the mean ages of both cases and controls being 51 years 4 months. The mean Bortner score of the case subjects was 174.3 (SD=42), which was significantly higher than that of the controls (155.3; SD=37). For most of the items the difference between cases and controls was not normally distributed, and so a non-parametric test was used to determine significance. The case subjects scored higher than controls in 12 of the 14 items, although the difference was significant in only four (table).

Mean scores for each item of Bortner questionnaire

Type A characteristic	Mean score		Difference	
	Cases N=50	Controls N=50	Mean	Significance*
Never late	20.9	21.4	-0.5	0.29
Very competitive	12.2	12.9	-0.7	0.21
Anticipates what others are going to say and interrupts	9.2	6.4	2.8	0.02
Always rushed	11.1	9.4	1.7	0.10
Impatient	11.9	8.9	3.0	0.03
Goes "all out"	14.8	13.9	0.9	0.34
Tries to do too many things at once	12.6	8.5	4.1	0.003
Emphatic in speech	11.0	10.6	0.4	0.33
Wants good job recognised by others	15.5	15.3	0.2	0.31
Fast (eating, talking, etc)	14.4	13.5	0.9	0.21
Hard driving	9.0	6.6	2.4	0.03
Expresses feelings	12.2	10.6	1.6	0.13
Few interests outside work	10.8	8.7	2.1	0.08
Ambitious	8.7	8.6	0.1	0.36
Total	174.3	155.3	19.0	0.03

*Significance level determined by one-tailed Wilcoxon matched pairs rank test.

Among the controls those in social classes IV and V had lower scores than those in classes I and II, but this gradient was not seen in the cases, in whom even those in classes IV and V had high scores.

No consistent relations existed between Bortner score and smoking habits, plasma cholesterol concentration, blood pressure, or relative weight, which had been measured at earlier screening examinations. There was no relation between the Bortner score and the time since the case subjects had had their heart attack, which ranged from 5 to 139 months (mean 55, median 57 months) ($r=0.05$).

Comment

The difference between cases and controls reached significance in only four of the individual items (as well as in the total score), but in 12 of the 14 items the subjects with CHD scored higher than the controls, which strengthens the idea that a true association between type A behaviour and CHD exists. Since the cases were studied after their infarcts, one explanation for this association might be a subsequent change in behaviour. Thus a relation might be expected between Bortner score and time since the infarct, and although this was not found, only a prospective study could determine whether the behaviour pattern or the heart attack comes first.

That there was no relation between Bortner score and conventional risk factors suggests that although type A behaviour appears to be concerned in the aetiology of CHD in Britain, it is probably not acting via associations with other known risk factors.

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