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(*Accepted 22 November 1978*)

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Gastric secretion in peripheral vascular disease

An association has been reported between arteriosclerotic cardiovascular disease and peptic ulcer.[1] Both gastric and duodenal ulcers occur more frequently than expected in necropsy studies and retrospective reviews of patients with abdominal aortic aneurysms. It has even been suggested that elective vagotomy should be carried out at the same time as aortic reconstruction. Duodenal ulcer has also been associated with "occlusive" peripheral vascular disease.[2] We therefore investigated basal and maximal acid output in patients with occlusive peripheral vascular disease who had no known peptic ulcer.

Patients, methods, and results

We studied a series of 71 consecutive patients who presented between March 1977 and January 1978 to the vascular unit with occlusive arteriosclerotic peripheral vascular disease. They were under the age of 80, their claudication or rest pain was sufficiently severe to warrant translumbar aortography, and they were fit for general anaesthesia. Eleven patients (16%) had already been treated for peptic ulcer (one gastric, 10 duodenal), and they were not studied further. Fourteen of the remaining 60 were unwilling to swallow the tube and in four the test was technically unsatisfactory. Finally, 42 (32 men and 10 women) underwent satisfactory acid secretion studies. All the patients were Caucasian. Their mean age was 59·3 years—men 58·6 (range 41-69), women 61·4 (range 44-77). Their mean weights were: men 69·1 kg, women 60·8 kg. Distribution of the ABO blood groups was in the accepted British range. Only one man and one woman were non-smokers. Three men and two women were diabetic. Eight men and one woman had had myocardial infarcts. Ten men (31%) and nine women (90%) complained of dyspepsia (the distribution of dyspepsia was the same in those patients who could not tolerate the test as for those who completed it).

The acid secretory state of the 42 patients was established by a standard basal-pentagastrin gastric function test. After an overnight fast a nasogastric tube was passed. Basal acid output (BAO) was measured over 30 minutes and was followed by a 90-minute intravenous infusion of pentagastrin (6 μg/kg/h). Peak acid output (PAO) was calculated in mmol/h by trebling the highest 20-minute acid output. The results have been compared with previous measurements of basal and maximal acid output by one of us (JHB) in 31 people without dyspepsia[3] and 60 patients with duodenal ulcer[4] aged 30 years or older. Significant differences were calculated by Student's *t* test for peak acid output and by the non-parametric Mann-Whitney U test for basal acid output because these measurements were not normally distributed.

In both men and women with peripheral vascular disease basal acid output was significantly higher than it was in normal people and not statistically different from output in patients with duodenal ulcer (table). Peak acid output in women with peripheral vascular disease was also significantly higher than in normal women and not significantly different from output in women with duodenal ulcer. The peak acid outputs in the men in our study were significantly higher than in normal men but significantly lower than in men with duodenal ulcer.

Comment

Patients with peripheral vascular disease without known ulcer disease may be acid hypersecretors almost in the duodenal ulcer range, but we cannot explain this association. Smoking may cause peripheral vascular disease but probably does not produce either duodenal ulcers or gastric hypersecretion.[5] It is impracticable to measure gastric secretion in all patients undergoing vascular operations, but we are currently assessing the prophylactic use of cimetidine.

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(*Accepted 17 November 1978*)

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Difficulty in stopping lithium prophylaxis?

Textbooks and articles about lithium prophylaxis for recurrent manic-depressive psychosis tend to imply that, once begun, prophylaxis should continue indefinitely.[1 2] Little has been written about the indications for stopping lithium or when and how to do this. Our experience in a lithium clinic suggests that attempts to stop the drug can result in unexpected clinical difficulties. This paper reports the development of an acute confusional state associated with stopping lithium.

Case report

A woman, now aged 58, with a bipolar manic-depressive psychosis required six hospital admissions between 1964 and 1971. Lithium prophylaxis was begun in 1971. She was given lithium carbonate, between 800 and 1600 mg/day, by mouth. Serum lithium concentrations always remained within our "therapeutic range" of 0·5-1·5 mmol(mEq)/l during treatment. In November 1977 she asked to stop taking lithium. She had been well for six years, had not needed hospital admission during this period, and no

Comparison of basal (BAO) and peak acid outputs (PAO) in patients with peripheral vascular disease (PVD) with published data in normal subjects[3] and patients with duodenal ulcer[4]

	BAO (mmol/h)				PAO (mmol/h)			
	No	Median	Range	P	No	Mean	Range	P
Men:								
Normal	16	0·6	0-3·8	<0·00003	16	18·7	0·3-45·0	<0·02
PVD	32	1·8	0-10·6	NS	32	32·3	1·8-69·3	<0·005
DU	41	2·8	0·1-17·9		43	41·8	15·0-66·6	
Women:								
Normal	15	0·2	0-2·7	<0·025	15	8·0	0·4-15·8	<0·001
PVD	10	1·5	0-7·6	NS	10	29·8	10·8-52·5	NS
DU	17	1·9	0-5·7		17	32·4	18·8-82·6	