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Sympathetic tone and relation between sodium intake and blood pressure in the general population

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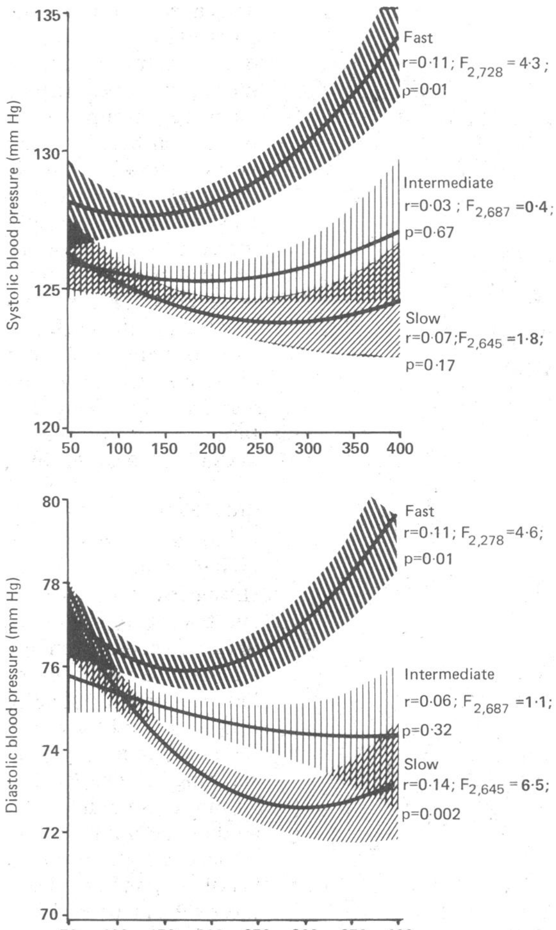
In a random sample of the population we tested the hypothesis of Poulter *et al* that sodium intake and sympathetic tone interact to determine blood pressure.[1] As in Poulter *et al*'s study, the pulse rate at rest was taken as an index of sympathetic tone and the urinary excretion of sodium as an index of salt consumption.

Subjects, methods, and results

We studied 2450 subjects aged 18 and over who were randomly selected from the population.[2] Everybody was visited twice at home and on each visit the pulse rate over one minute and five blood pressure measurements were obtained. The results were averaged for analysis.[2] Twenty four hour urine collections were analysed for sodium and creatinine. People taking antihypertensive drugs (n=296) and those who collected more or less urine than required[2] (n=73) were removed from the analysis. As in Poulter *et al*'s study,[1] the remaining 2081 subjects were divided according to sex and the two groups stratified into three according to pulse rate. Significant blood pressure covariates were traced by stepwise multiple regression. Blood pressure adjustments for age included both a linear and a quadratic term. Coincidence of regression lines was investigated by a multiple regression approach with two dummy variables identifying the pulse rate thirds.

After adjustment for sex, age, and body mass index there was a significant curvilinear relation between urinary excretion of sodium over 24 hours and both systolic and diastolic pressure in the upper third of pulse rate (n=735) (figure). In the middle third of pulse rate (n=694) the correlations between blood pressure and urinary sodium excretion were not significant. In the lowest third of pulse rate (n=652) urinary sodium excretion was again curvilinearly related to both systolic and diastolic blood pressure (figure).

The regression lines adjusted for sex, age, and body mass index were not coincident across the pulse rate thirds ($p<0.001$ for test of coincidence). Indeed, the linear and quadratic regression coefficients for urinary sodium excretion, but not the intercepts, were different in the upper third of pulse rate compared with the two others ($F_{2,2071}=18.4$ and 25·7 for systolic and diastolic pressure respectively; $p<0.001$). Additional adjustments for smoking, consumption of alcohol, and use of the contraceptive pill did not remove these differences.



Curvilinear relations between urinary sodium excretion and systolic and diastolic blood pressures (adjusted for sex, age, and body mass index) in three pulse rate thirds (fast, intermediate, and slow). For each regression line 95% confidence interval for population mean, correlation coefficient (r), F value, and associated p value are given

Comment

In our study less than 2% of the variance in blood pressure was explained by changes in the urinary excretion of sodium. Nevertheless, the finding that a raised blood pressure was associated with the combination of a fast pulse rate and a high urinary excretion of sodium is compatible with the suggestion of Poulter *et al* that a high sympathetic tone is a prerequisite for sodium to act as a pressor substance.[1] Alternatively, a high intake of sodium may facilitate the pressor effect of sympathetic stimulation. In our study smoking and consumption of alcohol were ruled out as confounding factors, but no information was available on the subjects' psychological stress and usual physical activity and therefore a possible influence of these two factors cannot be excluded.

Subjects with a slow pulse rate might have a low sympathetic tone; if their intake of sodium increases from 50 to 300 mmol/24 h sympathetic tone might be inhibited further and the secretion of renin suppressed, resulting in a fall in blood pressure. In contrast, people with a fast pulse rate might have an enhanced sympathetic tone and less effective baroreflexes[3]; if their intake of sodium increases above 160 mmol/24 h the high sympathetic tone might prevent the inhibition of renin secretion and impair the ability of the kidney to excrete sodium,[4] thus causing the blood pressure to rise. Increasing the intake of sodium may therefore have opposite effects depending on the prevailing sympathetic tone.

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Quality of life of elderly people after surgery for benign oesophageal stricture

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Benign oesophageal stricture in elderly patients is usually treated by intermittent dilatation and drug treatment to control continuing symptoms of reflux.[1,2] But this treatment has complications.[3] We assessed the results of surgery for stricture in elderly patients by using a validated quality of life scale[4] in patients in whom endoscopic dilatation was unsuccessful or impossible because of the severity of the stricture.

Patients, methods, and results

We assessed consecutive operations performed between 1971 and 1983 for histologically confirmed benign oesophageal stricture in 124 patients (85 women, 27 men) aged 70 and over (range 70-89, mean 76; 16 patients were over 80). Sixty eight patients were alive, 44 had died, and 12 were excluded from the study because hospital records were untraceable for nine and death records unavailable for three. Surgery was performed by KM and the survivors assessed independently by BNMJ.

Twenty four patients had medical disorders that were likely to influence the outcome of surgery. Six had a previous history of carcinoma. Forty five had had a laparotomy and 13 repairs of hiatus hernia and gastro-oesophageal reflux. Seventeen were taking non-steroidal anti-inflammatory drugs.

Intraoperative guided dilatation and an antireflux procedure were performed in 79 patients transthoracically. Thirty two required resection of the stricture because it was difficult to carry out the dilatation, or because of deep penetrating ulcer, or because of scarring with considerable oesophageal shortening. A Celestin tube was inserted in one patient when a malignancy was suspected.

Twelve patients died after operation (five from cardiac causes and two from pneumonia); seven had had conservative surgery and five had had resections (not significant $0.5>p>0.1$); only one had had previous surgery on the oesophagus. Four further patients were too frail to return home and died in hospital. Twenty eight others died two months to 10 years (mean 42·5 months) after surgery. Five year mortality was 32%.

The state of health of the 68 survivors was assessed from two to 14 years (mean 65·5 months) after surgery according to the index of Fanshel and Bush (table): 41 were in groups S_A, S_B, and S_C, 13 having occasional symptoms. Minor and major disabilities were caused by heart disease, arthritis, old age, or memory impairment. The patient in group S_F had had a myocardial infarction and was expected to return to group S_A. The patients in group S_G were in wheelchairs because of arthritis. The one patient in an institution had dementia. Eight patients continued to receive drugs that were likely to cause local inflammation.

Fifty four of the 68 patients had no dysphagia. Six showed no improvement from the preoperative grade. Only two required further intermittent dilatation. Fifty two had no symptoms of reflux, but six were taking antacids, cimetidine, metoclopramide, or anticholinergic drugs alone or in combination. There was no significant difference in the incidence of dysphagia ($p<0.5$) or symptoms of reflux ($p<0.5$) between the conservative and radical procedures. Two patients had symptoms of the gas bloat syndrome. None required further surgery.

State of health of 68 survivors of surgery for oesophageal stricture according to Fanshel and Bush index[4]

Quality of life index[4]	Asymptomatic	Reflux only	Dysphagia only*	Reflux and dysphagia*
S_A: wellbeing	21	3	2 (26)	1 (1)
S_B: dissatisfaction (group that includes most of the population)	5	2	1 (8)	0
S_C: discomfort (minor symptoms that do not appreciably alter efficiency)	2	4	0 (5)	0 (1)
S_D: minor disability (reduced efficiency)	8	1	3 (16)	4
S_E: major disability (severe reduction in expected level of performance)	1	0	1 (2)	0
S_F: disabled but ambulatory (yet able to contribute to society)	1	0	0	0
S_G: confined to home	5	1	1 (4)	0 (3)
S_H: confined and bedridden				
S_I: isolated and in institution	0	0	1 (1)	0
S_J: coma				
S_K: death				
Total No (%)	43 (63)	11 (16)	9 (13)	5 (7)

*No of patients with symptoms before operation in parentheses.

Comment

Surgery relieved the symptoms of benign oesophageal stricture in 43 of the 68 patients whose