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FAILURE OF SALT TO MOBILISE RENAL DOPAMINE IN ESSENTIAL HYPERTENSION

SIR,—On several occasions over the past twenty years it has been noted that patients with essential hypertension have low urinary dopamine outputs.[1,2] These observations aroused little interest since, at the time, dopamine was regarded simply as a precursor in the biosynthetic pathway for adrenaline and noradrenaline. Goldberg et al.[3] demonstrated later that dopamine has a specific receptor system in the periphery and that activation of renal dopaminergic mechanisms leads to kidney vasodilatation and natriuresis. Recent studies have suggested that dopamine is formed in the kidney by the action of renal tubular dopa decarboxylase on L-dopa.[4,5] Oral salt loading in the rat and man produces a brisk rise in urine dopamine output[6] whereas volume overload with fludrocortisone tends to depress urine dopamine concentration.[7] These and other studies have led to the concept that dopamine may be an intrarenal natriuretic hormone.[8]

Under metabolic balance conditions we maintained five male patients with essential hypertension on a 20 mmol sodium, 50 mmol potassium diet for a week (table). No patient had ever received

DETAILS OF PATIENTS AND CONTROLS AND URINARY DOPAMINE EXCRETION ON LOW AND HIGH SALT DIETS

—	Age (yr)	BP (mm Hg)	Plasma creatinine (μmol/l)	24 h urinary dopamine (μmol)	
				Low salt	High salt
Controls					
A	21	110/60	87	1·8	3·1
B	22	120/75	87	1·0	1·3
C	22	120/80	120	2·1	2·9
D	21	120/70	65	2·2	3·5
E	20	140/80	73	2·1	2·6
F	22	140/80	73	1·4	2·7
Mean±SEM	21·3±0·3	125±5/74±3	84±8	0·8±0·1	2·7±0·3
Hypertensive					
1	54	180/110	90	1·2	1·0
2	44	170/100	83	0·8	0·6
3	60	170/100	107	0·4	0·9
4	30	160/100	109	0·7	0·2
5	42	160/100	135	1·1	1·1
Mean±SEM	46±5·2	168±4/102±2	105±2	0·8±0·1	0·8±0·2

hypotensive or diuretic therapy. After one week of low salt intake the patients were changed to a diet containing 220 mmol sodium and 50 mmol potassium for a further 7 days. 24 h urine free dopamine after 5 days on low salt was 0·8±0·1 μmol (mean±SEM) and after two days on the high salt regimen the 24 h urine dopamine was not significantly different (0·8±0·2 μmol). In contrast six male normotensive individuals studied under identical conditions had a mean 24 h urine dopamine of 1·8±0·2 μmol after five days of low salt, whereas after two days of salt loading this had risen to 2·7±0·3 μmol ($p<0·05$; Wilcoxon signed rank test). The urinary dopamine output for the low salt period was higher in the normotensive group than in the hypertensive patients ($p<0·05$, Wilcoxon). Because of the small numbers we cannot say whether all hypertensive patients will show a reduced, or absent, dopamine response to a salt load. We are now investigating younger hypertensive patients and age matched controls, although we have no evidence that age alters basal dopamine output or the renal response to salt loading (unpublished).

Failure of the kidney to generate dopamine on salt load could be a cause, or a consequence, of essential hypertension and might lead to inappropriate vasoconstriction and inefficient excretion of the sodium ion; both these secondary problems resulting in a rise in arterial pressure which would be limited by the pressor natriuretic mechanism.[9] Failure to generate renal dopamine may have a genetic basis and could be one factor which might explain the variable prevalence of essential hypertension in different racial groups.

Dopamine, once regarded as an unimportant metabolic precursor of adrenaline and noradrenaline, may come to occupy a central place in the control of renal salt handling. Disorders of its renal production may contribute to the genesis (or maintenance) of raised blood pressure. Kidney specific dopamine agonists, such as γ-glutamyl dopa[10] may be effective in the treatment of the hypertensive low dopamine disorder.

C. M. PERKINS
I. F. CASSON
G. F. COPE
M. R. LEE

University Department of Medicine,
Leeds General Infirmary,
Leeds LS1 3EX

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CORONAVIRUS-LIKE PARTICLES IN DIARRHOEA STOOLS

SIR,—We find the letter by Dr Dourmashkin and his colleagues (Nov. 1, p. 1250), entitled Are Coronavirus-like Particles Seen in Diarrhoea Stools Really Viruses? rather disturbing. The title gives the impression that there is some real evidence against the conclusion that the particles referred to as coronaviruses are real viruses. However, the evidence, produced from a single case of diarrhoea, is far from convincing. Both negative staining and processing for thin sectioning cause different but significant shrinkage, and to compare dimensions from specimens treated by either of these methods is difficult. We are also led to believe that fig. 1 and fig. 2 are comparable, but the negatively stained coronavirus is reproduced at twice the magnification of the sectioned structure. Interpretation of results obtained by electron microscopy from faecal samples alone is very difficult, so those who appreciate the limitations and problems associated with preparative techniques used in electron microscopy will not be convinced by the evidence produced by Dourmashkin et al. The finding of several identifiable agents potentially capable of producing diarrhoea in a single sample is fairly common and cannot be used to dismiss a particular agent as a cause of diarrhoea. It is regrettable that Dourmashkin et al. did not take into account the excellent work carried out by Australian,[1] British[2] and Indian workers[3,4] over the past few years on epidemiological and cultural aspects of faecal coronaviruses, when considering the viral nature of these particles. We find evidence of coronavirus replication[2,5] in cell or organ culture more convincing than association of structures obtained from thin sections of faecal material.

A. CURRY
W. K. PAVER

Public Health Laboratory,
Withington Hospital,
Manchester M20 8LR

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