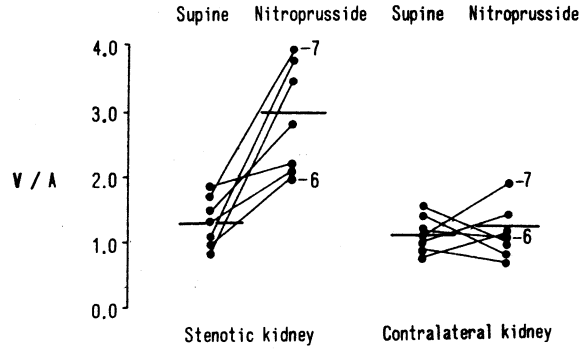
NON-RENIN-MEDIATED RENOVASCULAR HYPERTENSION

SIR,—I cannot accept the existence of non-renin-mediated renovascular hypertension as a new syndrome on the evidence provided by Dr Marks and his colleagues (March 19, p. 615). Far more substantiation is required because if this syndrome does not exist, the clinical value of divided renal-vein-renin determinations will have been needlessly questioned.

Saralasin will consistently lower the blood-pressure only when plasma-renin is raised, irrespective of whether this high renin is brought about by renovascular hypertension or by sodium depletion.[1,2] The subnormal renin in the two patients of Marks et al. would preclude a blood-pressure-lowering effect of saralasin[2] but not an initiation of renovascular hypertension by the renin angiotensin system.[3]

More important, the renal-vein-renin studies cannot exclude renin-mediated renovascular hypertension in their two patients. The low peripheral renin concentrations suggest that the renin-angiotensin system had already fulfilled the perfusion needs of the stenotic kidney by raising the blood-pressure sufficiently, and a left/right difference in renin secretion at this late stage[3] of renovascular hypertension would not be expected. Instead of measuring renal-vein renins under "slight sodium depletion", as Marks et al. did, it would seem more rational to use a test which strains the baroreceptors of the ischæmic and contralateral kidney. During the past three years we have investigated seven patients with renovascular hypertension similar to the two described by Marks et al. All were cured by surgery. The figure shows that in all of them we got false-negative results when the renal-vein was sampled with the patient supine. Only after reduction of blood-pressure to normal by sodium nitroprusside infusion[4] did we find a selective increase in renin secretion on the stenotic side; contralateral renin secretion remained suppressed. In our experience[5] stimulation of renin secretion in both kidneys indicates hypertensive damage in the contralateral kidney and permanent return to normal blood-pressure after operation would not be expected.



Renal-vein-renin studies in seven patients with unilateral renovascular hypertension done when the patient was supine and after nitroprusside-induced normotension.

Results are expressed as venous/arterial ratio of plasma-renin activity on both sides. A factor greater than 1 indicates renin secretion. Plasma-renin activity was measured by the method of Boyd et al.[8] which ensures the exclusive measurement of active renin. Renal arterial renin = peripheral venous renin.[9] Patients 6 and 7 are indicated individually.

Renal-vein-renin studies should be done during acute reduction of blood-pressure to normal. This would simulate preoperatively the expected postoperative fall in blood-pressure and thereby unmask the perfusion needs of the stenotic and contralateral kidney. Hydrallazine[6] or diazoxide[7] may provide a less elaborate means than nitroprusside[4,5] for doing these studies.

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