COMMUNICATIONS

1. BODY FLUID DISTRIBUTION AND PRESSURE IN EXPERIMENTAL HYPERTENSION

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Ledingham has produced evidence that in experimental renal hypertension (ERHT) the elevated blood pressure is due primarily to a rise in cardiac output with a secondary increase in peripheral resistance (Ledingham & Pellin, 1967, Circulation Research, 20, Suppl. 2, p. 187; Ledingham, 1971, Journal of the Royal College of Physicians of London, 5, 103-134). Part of this evidence is the observation that following removal of the constricting renal artery clip the fall in blood pressure is associated with a marked decrease in cardiac output with a rise in peripheral resistance.

We have observed changes in blood pressure, venous pressure and tissue pressure (Guyton capsule), plasma volume (PV), extracellular fluid volume (ECFV) and haematocrit during the development and reversal of ERHT in the rat, in an attempt to account for these alterations in cardiac output.

The development of hypertension is associated with a rise in plasma volume, venous pressure and tissue pressure. After removal of the clip blood pressure falls rapidly; this is associated with an internal redistribution of fluids. PV falls, ECFV rises and venous pressure falls. This could account for the decrease in cardiac output. The rise of ECFV is accompanied by a fall in tissue pressure suggesting an increase in the compliance of the tissue spaces.

The part played by changes in tissue pressure in the mechanism of ERHT will be discussed.

2. THE RELATIONSHIP BETWEEN BODY FLUID COMPARTMENT VOLUMES, RENIN AND BLOOD PRESSURE IN CHRONIC RENAL FAILURE

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Blood pressure in patients with chronic renal failure appears to be dependent on (a) sodium and (b) the renin-angiotensin system. The present study was performed to investigate the role of various body fluid compartment volumes and plasma renin activity in determining mean arterial blood pressure in such patients.

Total body water, total exchangeable sodium, extracellular fluid volume, plasma volume and red cell mass were measured simultaneously by an isotope dissection technique, using tritiated water, $^{24}\text{NaCl}$, $^{38}\text{Na}$, $^{35}\text{SO}_4$, $^{125}\text{I}$-labelled human serum albumin and $^{51}\text{Cr}$-labelled red cells. From the above were also derived 'intracellular' fluid volume and residual sodium (estimated as sodium lying outside the extracellular fluid space). The isotope methods used were verified in vitro. Plasma renin activity was measured by an enzyme-kinetic method (Newton & Laragh, 1968, Journal of Clinical Endocrinology and Metabolism, 28, 1006).

Patients with terminal renal failure were studied before starting regular dialysis treatment, all hypotensive and diuretic drug therapy having been withdrawn at least 1 week previously. Any previous dietary sodium restriction was continued throughout the study. After a period of dialysis treatment, during which time an attempt was made in all patients to control blood pressure by removal of salt and water, and by dietary salt and water restriction, each patient was studied again.

Before dialysis treatment was started, mean arterial blood pressure was significantly related to blood volume ($P<0.01$), plasma volume ($P<0.02$), total exchangeable sodium ($P<0.05$) and plasma renin activity ($P<0.05$): there was no significant correlation with extracellular fluid volume.

In the second study, performed after a period of regular dialysis treatment, no correlation was found between mean arterial pressure and plasma renin activity or any of the body fluid compartment volumes.

3. AORTIC ARCH BARORECEPTOR ACTIVITY IN EXPERIMENTAL HYPERTENSIVE RABBITS

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(Introduced by H. B. BARCROFT)

The physiology of the aortic arch baroreceptors has been studied in experimental renal hypertension and in calciferol-induced medial sclerosis. The mean blood pressure of the renal hypertensive rabbits was 156.2 ± 7.7 mmHg (range 125-180), and of the calciferol group was 135.5 ± 8.9 mmHg (range 122-175). The normal pressure was 85.3 mmHg (range 65-110).

The aortic arch was isolated and perfused at non-pulsatile pressures with Kreb's Henseleit solution. Single- or few-fibre recordings were made from the baroreceptor fibres of the aortic nerves.

It was found that the threshold pressure below which the fibres were inactive was higher in the renal hypertensive group (Angell James, 1971, Journal of Physiology, 213, 429-439), and lower in the calciferol treated group (Angell James, 1971, Journal of Physiology, 217, 309-319) than in the control normal group (Angell James, 1968, Journal of Physiology, 169, 315-328; 1971, Journal of Physiology, 214, 201-223). However, in both the treated groups the increase of baroreceptor activity with increments of pressure was less than in the controls.

The diminished baroreceptor activity was associated with an increase in the stiffness in the aortic arch wall demonstrated by pressure-volume curves. There were also demonstrable histological lesions. This change in baroreceptor activity could, in part,