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HYPERTENSION AND RENAL DISEASE.

By O. L. V. DE WESSELOW,
M.B.OXON., F.R.C.P.LONDON.
Senior Physician to Out-patients, St. Thomas' Hospital, London.

I PROPOSE to discuss to-night two types of hypertension :—

(1) The transitory, seen in acute glomerulonephritis and in eclampsia, and subsiding with recovery.

(2) The permanent, so-called essential hypertension which, when once established, persists. If we accept the German nomenclature, this type of raised blood-pressure may remain "benign," or may pass over into the "malignant" form, and end, unless the patient's life is terminated by cerebral hæmorrhage, cardiac failure or intercurrent disease, in contracted kidney and uræmia.

Let us first consider the factors which may produce a rise in blood-pressure. Briefly, they are : (i) increased cardiac output—the peripheral resistance remaining constant; (ii) increased blood volume—plethora, or increased blood viscosity with increased frictional resistance; (iii) increased peripheral resistance, this resistance lying in the smaller arteries and arterioles.

We may dismiss increased cardiac output and increased blood viscosity; in neither of these two types of hypertension do they play a rôle. Increased blood volume I shall discuss briefly at a later stage, but I shall assume that there is a general agreement that these two forms of hypertension result from an increase in the peripheral resistance, in a narrowing of the channel through which the blood is being forced.

Under normal conditions narrowing of one arterial region is compensated for by widening of another. An actual narrowing of the whole channel is conceivable as the result of : (i) a generalized anatomical change in the arteries ; (ii) a generalized arterial spasm ; (iii) a lack of interplay