CHAPTER 17

THE PHYSIOLOGY OF ESSENTIAL HYPERTENSION

Most human hypertension does not show obvious physiological abnormalities and such cases are called "essential" or "idiopathic." Rigorous experimental investigation is not often possible, and consequently an acceptable explanation for the cause of essential hypertension has not been developed.

Some physiological abnormalities have been found, but often the measured values are quite close to normal. Plasma renin activity is usually normal or low, and renin release is often sluggish. Cardiac output is elevated in some young hypertensives; flow decreases with age. Fluid volumes are normal to low. Venous compliance is decreased. Plasma catecholamines are close to normal, but the response to acute stress may be greater than normal. Baroreceptor responsiveness is attenuated. The kidney shows progressive decreases in flow and filtration and increases in filtration fraction.

Many different single causes of essential hypertension have been postulated and it has also been suggested that essential hypertension is actually a composite disease with many causes. But, less diversity is also a possibility. If inferences are drawn from animals having experimental or genetic hypertension of known origin, its possible that essential hypertension is caused by a genetic deficiency in renal function that is amplified by excessive sodium intake and/or overactivity of the sympathetic nervous system. Recalling the efficacy of the Goldblatt clamp in elevating blood pressure, an usually high afferent renal resistance might be the real culprit.

EPIDEMIOLOGICAL vs PHYSIOLOGICAL CONSIDERATIONS

Hypertension is said to exist when systolic and/or diastolic pressures pass above arbitrarily defined upper limits of normalcy, as discussed in a previous chapter. When there is no obvious reason for the pressure increase, essential or idiopathic hypertension is said to exist.
The epidemiology of human hypertension has also been discussed in a previous chapter. Studies comparing different societies and studies within societies have at times shown that the prevalence of hypertension can be correlated with age, obesity, sodium intake, and genetic and familial factors. None of these correlations indicate what mechanism or mechanisms might be causing the increased vascular resistance.

Essential hypertension has been most difficult to study experimentally. Arterial pressure may increase in an individual slowly and insidiously over several decades; prospective studies designed to follow such increases risk being tedious, expensive, and beyond the interests of most scientists and agencies supplying funds for medical research. Highly invasive studies are not acceptable for humane reasons. Data must be normalized to accommodate a wide range of body sizes and shapes. The proven benefits of antihypertensive therapy makes it very difficult to justify extended periods of observation using unmedicated hypertensives.

Theoretically, the most attractive experimental approach is the longitudinal study. Repeated measurements are made on a single individual and time-dependent features in the progression of hypertension are captured. Two less demanding alternatives are cross-sectional and retrospective studies. In the cross-sectional approach, measurements are made on many different subjects with the assumption that isolated measurements using many subjects will eventually show the same trends as multiple measurements using a few subjects. In the retrospective approach, subjects with fully established hypertension are identified and past medical records are reviewed to see if any striking features emerge. All of these experimental strategies have contributed to our meager understanding of the physiological aspects of essential hypertension.

PHYSIOLOGICAL STUDIES

The renin-angiotensin system

The mean values of plasma renin activity (33), (121), (372) and plasma angiotensin II concentration (58), (334) are normal in essential hypertension, but the distribution is broad enough so that single individuals may show high, normal or low levels. Schalekamp and colleagues