mm/s) of the aortic valve and the endocardial lines at the tip of the mitral valve to measure the short axis dimension of the left ventricle. Aortic pressure was monitored simultaneously and displayed together with the echocardiogram.

Before occlusion of the LAD, the beat time was 340 ± 46 ms, pre-ejection period 60 ± 14 ms (PEP: Q to beginning of aortic valve leaflet separation), total electromechanical systole 150 ± 37 ms (TEMS: Q to aortic valve leaflet closure), left ventricular ejection time 90 ± 21 ms (LVET: beginning to end of aortic valve leaflet separation) and the peak opening velocity of the aortic valve was 62 ± 18 cm/s.

These measurements were repeated after giving Nifedipine intravenously (1 µg/kg body weight) over a period of 2 min. A prolongation of TEMS to 174 ± 33 ms (mean ±SD) was first noted 30 s after the start of injection when heart rate was still unchanged. The ratio PEP/LVET decreased from 0.4 to 0.3. These changes returned to previous values after 3–5 min after the end of injection.

The snare on the LAD was tightened and the effect on TEMS, PEP/LVET and the other echocardiographic indices were measured both with and without the application of Nifedipine. Throughout all these procedures echocardiographic findings were correlated with regional myocardial perfusion using 131I-Au. The drug has specific effects on the electromechanical events and echocardiographic indices suggesting improvement in cardiac function accompanied by changes in regional myocardial perfusion. Thus echocardiography can be used to monitor the action of Nifedipine in the heart with limited blood supply in close correlation to the beginning of improvement of regional myocardial perfusion.

86. PLASMA NORADRENALINE, PHYSICAL ACTIVITY AND SYSTOLIC BLOOD PRESSURE IN HYPERTENSION

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Plasma noradrenaline (NA) probably reflects activity of the sympathetic nervous system but attempts to correlate plasma NA and systolic blood pressure (SBP) have proved controversial.

We have investigated the relationship between plasma NA, physical activity and SBP in 8 untreated subjects (3 females, 5 males; mean age 42 years, range 23–52) with mild to moderate hypertension and no evidence of target organ damage. Plasma NA was measured by a sensitive and specific radio-enzymatic method (Henry, Starman, Johnson & Williams, 1975, Life Sciences, 16, 375). Blood pressure was measured continuously from a fine brachial artery cannula and recorded on a portable tape-recorder (Littler, Honour, Sleight & Stott, 1972, British Medical Journal, III, 76). Dietary sodium intake was standardized (no salt added to diet for 3 days, then 100 mmol daily for duration of BP recording). Blood samples from a forearm venous cannula were taken during various activities over a 24 h period.

Plasma NA was low during sleep, 0.28 ± 0.03 µg/l (mean ± SEM) and increased to 0.48 ± 0.08 µg/l lying awake, 0.66 ± 0.05 µg/l standing, 1.15 ± 0.14 µg/l during walking and to 2.97 ± 0.53 µg/l during vigorous, submaximal bicycle exercise. A linear relationship was found between log NA and SBP. This apparent when mean values of NA and SBP for the whole group were correlated with mean arterial pressure (r = 0.99, P < 0.001) and when each separate value for NA and SBP was plotted (r = 0.62, n = 106, P < 0.001). The relationship also held when log NA and SBP from individual patients were plotted (P ranged from < 0.05 to < 0.001); the slope of these regression lines varied considerably between individuals.

These observations support the hypothesis that circulating plasma levels of NA reflect the level of sympathetic activity; they suggest that interindividual differences, presumably in NA release and clearance, may confound attempts to interpret plasma NA in groups of normotensive and hypertensive subjects.

87. PLASMA AND INTERSTITIAL FLUID VOLUMES IN PRIMARY HYPERTENSION

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Knowledge of the fluid and electrolyte status of patients with primary hypertension is important in establishing aetiological mechanisms. Some investigators have found normal or increased extracellular water and normal plasma volume which suggests renal retention of fluid may play a causal role. However, other groups have demonstrated a reduction in plasma volume in primary hypertension which appears to be related to the height of the blood pressure (Tarazi et al., 1969, Circulation, 50, 357).

Plasma volume was measured, by a dilution method using Evans Blue, in 68 male caucasian subjects: 41 with uncomplicated primary hypertension (casual blood pressure >140/90 mmHg) and 27 with normal blood pressure. Plasma volume was significantly reduced in the hypertensive patients using either weight, height or body surface area as reference, although the latter correlated best with the observed plasma volume in the normotensive subjects (r = 0.92). The mean plasma volume of the hypertensive patients, expressed as a percentage of the value predicted by body surface area, was 93.9 ± 1.2% (mean ± SEM). The plasma volume was reduced significantly in all subjects with blood pressures exceeding 140/90 mmHg, although the relationship between the reduction in plasma volume and blood pressure was not linear.

 Extracellular water, calculated from the radioactive bromine space (82Br) with a 6 h equilibration and plasma volume, in 27 hypertensive subjects was significantly lower than that predicted by height and weight: 17.5 ± 0.34 litres versus 18.0 ± 0.29 litres (mean ±SEM) (P < 0.025). Interstitial fluid volume (ECW - 0.92 × plasma volume) was also reduced although this did not reach statistical significance: 14.8 ± 0.31 litres versus 15.2 ± 0.24 litres (mean ±SEM) (P < 0.1, > 0.05). However, this study did demonstrate a close relationship between the reduction in plasma volume and interstitial fluid volume with increasing blood pressure.

The finding that interstitial as well as plasma volume decreases with increasing blood pressure suggests that the diuresis and natriuresis resulting from renal perfusion pressure are responsible, and therefore that fluid volume changes are a secondary phenomenon in primary hypertension and not causal. This is analogous to animal models of hypertension where a normal kidney is exposed to elevated blood pressure.

88. THE EFFECT OF HEREDITY ON THE PATTERN OF ARTERIAL PRESSURE CONTROL IN NORMAL MAN

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Since the children of hypertensive parents are much more likely to develop hypertension than the children of normotensive parents, arterial pressure has been studied in 34 normotensive young men (aged 18-26 years) in relation to their parents' pressure under disparate conditions in an attempt to define patterns of pressure response which might have prognostic significance.

Pressure was measured repetitively with a sphygmomano­meter by a single observer (i) during quiet supine recumbency for 30 min (basal BP), (ii) following an 85° tilt for 15 min (tilt BP) and (iii) following a large intravenous fluid load (26.7 ml/kg