Morton, 1974, in press). Plasma AVP levels (measured as pmol l⁻¹) rose significantly during fluid deprivation (5.9±0.3 to 9.6±0.2 SEM, \(P<0.001\)) and fell following an oral water load (5.0±0.6 to 3.8±0.5 SEM, \(P<0.02\)). There was no significant change in plasma AVP levels following an acute haemorrhage (500 ml in 10 min) nor after assuming the upright posture. Throughout the wide range of physiological studies there was a good correlation between plasma AVP levels and concurrent urine osmolality \((r = 0.69, P<0.001)\).

Thirty patients with hypertension (diastolic blood pressure > 100 mmHg) were studied under standard conditions of overnight recumbency and fluid deprivation. Plasma AVP in twenty-one patients with benign 'essential' hypertension was significantly lower than in fourteen normotensive subjects under the same conditions (3.8±0.04; 5.4±0.15 SEM, \(P<0.001\)) whereas plasma AVP was elevated in nine hypertensive patients in the malignant phase (8.2±1.2 SEM, \(P<0.01\)) although there was some overlap between the groups. There appeared to be no association between high levels of plasma AVP and the presence of hyponatraemia. Ten patients with cirrhosis of the liver (four with ascites) were studied under identical conditions. Plasma AVP levels were widely distributed, being elevated in five patients and normal in the remainder. There was no tendency for these patients with ascites to have higher levels of plasma AVP and again there was no association between high AVP levels and the presence of hyponatraemia. Of three patients with the nephrotic syndrome, two had distinctly elevated levels of plasma AVP (8.3, 9.3) while one patient had normal levels (5.3). One patient with pituitary diabetes insipidus showed no tendency to elevate plasma AVP levels on chlorpropamide therapy, despite a 25% reduction in urine flow. A patient with nephrogenic diabetes insipidus induced by lithium therapy had high basal levels of plasma AVP (6.6, 7.0); rising predictably during AVP infusion without change in a low urine osmolality.

13. THE INCORPORATION OF \(^3\)H-LYSINE INTO CARDIAC MYOFIBRILS IN CARDIAC HYPERTROPHY IN THE RAT, WITH A DESCRIPTION OF A NEW TECHNIQUE OF PREPARATION OF MYOFIBRILLAR PROTEINS FOR ISOTOPIIC COUNTING

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Cardiac hypertrophy was induced in the rat by abdominal aortic coarctation. Intraperitoneal \(^3\)H-lysine was given 4 h prior to sacrifice in rats with hypertrophy, sham operated rats, and normals. Cardiac myofibrils were prepared and characterized by succinate dehydrogenase and the inhibiting effect of sodium azide on Mg⁺⁺ ATP-ase. The incorporation of isotope into the whole myofibril was measured and correlated well with the onset and degree of hypertrophy. The same myofibrils were subjected to vertical slab, polyacrylamide gel electrophoresis in the presence of S.D.S., using a Tris borate buffer system, pH 7.2. This revealed clearly defined bands of myosin heavy chain, actin and tropomyosin which were selectively removed by staining of lateral gel section with Coomassie Blue. The gel slices were then eluted with a 0.5 M ammonium carbonate buffer and the protein solutions lyophilized for preparation for liquid scintillation counting. By this technique the approximate yield of these myofibrillar components was 25%.

14. PARAMALIGNANT SYNDROMES IN LUNG CANCER

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The incidence of paramalignant syndromes in lung cancer was studied in a series of 280 consecutive patients with histologically confirmed disease. The group as a whole consisted of patients with early lung cancer, one-third being referred through a Mass X-ray Unit.

The commonest abnormalities were weight loss and fever. The cause of fever could usually be explained. Endocrinopathies were present in 12% of the group, the commonest being non metastatic hypercalcaemia. Neuromyopathies were uncommon compared to other series and reasons are given for this discrepancy. Other paramalignant abnormalities were individually uncommon.