Pointers

Londonderry Clinical Meeting

Release of Noradrenaline: Professor J. H. Burn discusses the probable mechanism of the release of noradrenaline from sympathetic post-ganglionic fibres (p. 197).


Activity of Steroids: Drs. J. S. Jenkins and P. A. Sampson found conversion rates of cortisone and prednisone to the biologically active steroids were approximately the same in normal subjects and in four patients with liver disease (p. 205).

Blood Pressure and Bladder: Mr. J. J. G. Szasz and Dr. H. M. Whyte from Australia observed that distension of the bladder and voluntary contraction of the sphincter in the non-distended bladder produced a rise in blood pressure (p. 208).

Provocation of Multiple Sclerosis: Professor Henry Miller and colleagues report nine cases where vaccination or inoculation apparently produced this effect (p. 210). Leader at p. 192.

Anaemia in Renal Failure: Dr. A. B. Shaw submits evidence for a reduced red blood cell survival rate running in close parallel with the level of blood urea (p. 213).

Cystinuria: Dr. J. C. Crawhall and colleagues observe no untoward effect on foetus and dissolution of renal calculi in a patient treated throughout pregnancy with d-penicillamine hydrochloride (p. 216).

Self-inflicted Lesions: Dr. Ian D. Kitchin and colleagues describe artefact ulcers and bone lesions produced by elastic bands (p. 218).

Chloroquine Neuromyopathy: Arising after treatment for arthritis and associated with ocular lesions (p. 219).

Behçet’s Disease: Report of two cases with retinal vascular lesions (p. 220).

Glass-fibre Itch: Unusual mode of contamination (p. 221).

Scabies: Dr. Alan Lyell discusses its diagnosis and treatment in his Current Practice article (p. 223). Leader at p. 193. Letters at p. 245.

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Pertinax: Without Prejudice (p. 240).


Potassium Deficiency in Ambulant Patients

Though hypokalaemia is well recognized as a complication of a wide variety of medical and surgical disorders, it may also develop insidiously in patients who are ambulant and reach a severe degree before being diagnosed. A history of tiredness, muscle weakness, and perhaps polyuria and nocturia should suggest the possibility of hypokalaemia in a clinical situation where it might develop.

Potassium is largely an intracellular ion, and the extent to which it may be lacking is not easily measured. The plasma concentration of potassium is controlled by a number of factors in addition to the amount of potassium in the body, and though deficiency is often accompanied by a low level in the plasma the relationship between the degree of deficiency and the plasma level is variable. The electrocardiograph may occasionally show changes, but they correlate poorly with the body’s total deficiency of potassium. More precise diagnosis depends on the measurement by isotope tracers, whole-body counting, or by muscle biopsy, but none of these techniques is generally available.

Perhaps the commonest cause of potassium deficiency in the ambulant patient is the prolonged use of diuretics, often the benzothiadiazines, but also ethacrynic acid or frusemide. These drugs increase the amount of sodium reaching the distal tubules, and it is there available to exchange with potassium. If in addition there is some inhibition of carbonic anhydrase, the hydrogen ion concentration in the tubular cells is decreased and potassium excretion is enhanced. It is especially if this situation is combined with a low intake of potassium that deficiency will develop. These diuretics are often given for the treatment of congestive cardiac failure and less often for cirrhosis of the liver and the nephrotic syndrome. In all these conditions there may be increased production of aldosterone and thus an even greater tendency to lose potassium. Sometimes, particularly in the nephrotic syndrome, diuretics are combined with corticosteroids, and this is a potent potassium-depleting combination.

A practical problem is to decide which patients on diuretics require repeated measurements of plasma potassium and treatment with potassium supplements. This is particularly relevant to the treatment of patients on digitals, for shifts in the concentration of potassium in the plasma will alter the susceptibility of the heart to the action of the digitals. There is no easy solution to the problem, but in general, if the patient is taking a relatively small dose of a diuretic and is on a good mixed diet, potassium deficiency is unlikely to develop, particularly if dosage is intermittent and not continued for more than a few months. Those taking larger doses of diuretics, especially if they are on a restricted diet, will require potassium supplements. Until recently potassium chloride in enterico-coated tablets was the preferred form of oral potassium therapy. However, in 1964 D. R. Baker and colleagues reported ulceration of the
small intestines from them, and this has now been confirmed. Animal experiments suggest that it is the potassium chloride rather than the diuretics which is responsible. The combination of potassium chloride with a diuretic in a single preparation would seem to offer no advantage except convenience, for some such preparations can also cause intestinal ulceration, and they do not allow the dose of potassium to be adjusted to the needs of the patient.

Effervescent potassium B.P. is satisfactory so far as replacing potassium is concerned and is fairly palatable, but it is in the form of potassium bicarbonate, and this exacerbates rather than corrects any associated chloride deficiency produced by the diuretic. The resulting alkalosis will further increase loss of potassium through the kidneys and so prevent correction of any deficiency. The alternative is to use the proprietary Slow-K, which is a slow-release preparation containing 600 mg of potassium chloride per tablet (8.0 Meq potassium) and appears to be safe and satisfactory. The dose of supplementary potassium will depend on the degree of deficiency, the daily loss, and the amount of potassium already available in the diet. It will usually be in the region of 4.0 g potassium chloride (52 Meq potassium) per day, and there is little danger of hyperkalaemia developing, provided renal function is normal.

It has been recommended that supplementary potassium should be given on the days when the patient is not taking a diuretic, as it is then retained rather than promptly excreted, and this is satisfactory for many patients receiving diuretics. But some patients with severe cardiac failure are in a state of potassium deficiency in which they are unable to retain potassium even if adequate amounts are given.

The clinical picture of the patient with chronic congestive cardiac failure who has had a prolonged course of diuretics and who ultimately excretes potassium rather than sodium in response to these drugs is all too familiar. He retains increased amounts of extracellular fluid, with resulting oedema, but develops progressive intracellular deficiency of potassium with intracellular dehydration. This may be partly due to excessive production of aldosterone, but in addition it seems possible that the metabolic processes responsible for the sodium and potassium gradients between the intracellular and extracellular spaces may be defective, and thus allow potassium to leak from the cells and be excreted in the urine. The treatment of this state depends more on reversing the cardiac failure than on supplying excess potassium. These patients, however, are unlikely to be in the ambulant group discussed here. Occasionally potassium deficiency in these circumstances can be prevented by combining the benzothiadiazine with the potassium-retaining diuretic triamterene or by inhibiting the effect of aldosterone with spironolactone.

Excessive loss of potassium from the lower bowel accounts for another group of ambulant patients with potassium depletion. The colon can excrete potassium up to a concentration of 30 Meq/litre. Thus chronic diarrhoea, from whatever cause, can lead to severe potassium depletion. One class of patients in whom the cause of potassium deficiency may be obscure are those whose diarrhoea is due to excessive consumption of laxatives, especially of the anthraquinone group, over long periods. As they may be taken secretly, the cause may be far from obvious. Neoplasms of the colon may cause excessive potassium loss. These are usually villous papillomata, when the potassium is presumably excreted by the growth. Removal of the precipitating cause and giving supplementary potassium will quickly correct the depletion.

Repeated vomiting over a long period can cause loss of potassium. This is due partly to loss of potassium in the vomit and partly to the resulting alkalosis necessitating loss of potassium in the urine. Such patients may be ambulatory and show little clinical evidence of potassium deficiency until operation, when marked signs of depletion may develop.

Excessive potassium loss in the urine may be a feature of pyelonephritis, of a variety of congenital renal tubular defects, and prolonged osmotic diuresis, though the primary disease has usually overshadowed the potassium deficiency. Primary hyperaldosteronism also leads to excessive urinary loss of potassium and may accompany attacks of muscular weakness. Similarly, hypokalaemia may be a feature of Cushing's syndrome. Finally, a fall in plasma potassium associated with muscular weakness is a characteristic feature of the hypokalaemic type of periodic paralysis, whether hereditary, sporadic, or associated with hyperthyroidism. Potassium depletion does not occur in these conditions, and the fall in the plasma level represents merely a change in distribution between intra- and extracellular potassium.

Potassium deficiency should always be remembered, though it is rarely confirmed, in patients with muscular weakness or fatigue. Associated clinical features may make the diagnosis easy, but occasionally the cause may require an extensive search before it is elucidated.

Precipitants of Multiple Sclerosis

When a disease is as unpredictable in its behaviour as multiple sclerosis it is not surprising that a variety of causes have from time to time been thought to precipitate relapses or even to promote the first attack. The very multiplicity of these might argue against most of them having any specificity, and in fact onset and relapse are more often than not unrelated to any recognizably cause. But some factors do appear to have a more than coincidental relationship in certain patients, even though relatively few, and they may be divided roughly into four groups—traumatic, infective, hormonal, and immunological.

M. Keschner suggested a relationship between severe cranial and spinal trauma and the onset of the disease or its relapses, and D. McAlpine and N. Compston found a history of trauma (with which they class dental extraction) within