mainly from the NHS (including two doctors) with one representative from industry. As well as enforcing the working group's own proposals, the council's major task would be to determine "the best level and method of purchase [for each supplies item] having regard to the needs of the Service, industry, and the national economy."

The supply council is a compromise between the need for central co-ordination and the 1974 principle of delegated authority. Whether it is workable remains to be seen. If it succeeds in developing a comprehensive information system so that users—clinicians among others—can make informed choices then it will benefit both doctors and their patients.[6 7] But the council's continuing task is to decide the correct level of purchase for each *item* of supplies. Nowhere does the working group say what it means by item or deal with the problem of how the council's members, who all have other jobs, will cope with the sheer size of the work load and yet remain sensitive to the needs of users. The danger is that the council will frustrate its members and alienate the rest of the NHS; it will become just another committee in an organisation that already has too many.

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Clues in Perthes's disease

We now have ample histological evidence that the underlying disorder in Perthes's disease is a healing infarct of the femoral head epiphysis.[1-3] Further, many data suggest that the epiphysis in many, if not all, instances has been implicated in more than one ischaemic episode. Repetition of the ischaemia explains the slow clinical course of the disease. Similar pathological changes have been produced in the puppy by imposing repeated ischaemic episodes on the femoral head epiphysis.[4] In man, however, the cause of the repeated infarctions appears complex.

The problems of the disorder are peculiar to the hip. They not only stem from the functional importance of that joint but also reflect a "design fault" in the anatomy of the blood supply to the head of the femur. In children between the ages of 4 and 7 years no important vessels cross the epiphysial plate and a blood supply through the ligamentum teres has yet to develop.[5] Over this period the epiphysis is nourished entirely by the lateral epiphysial vessels running along the femoral neck posterosuperiorly under the periosteum. Here they are in a position to be compressed and even occluded by an effusion in the hip joint.[6] During the early stages of Perthes's disease venography has shown delayed venous outflow from the medulla of the femoral neck and diminished filling of the capsular veins.[7] Perthes's disease occurs at an age when transient synovitis of the hip is common, and indeed in a few instances it has followed a clinically evident effusion. Yet more often the onset of the disease is silent and the precipitating cause obscure.

The disease is most common between the ages of 4 and 7; it affects one or two children in a thousand and boys four times as often as girls. One case in 10 is bilateral. It is rare in negroes. If confusion with cases of multiple epiphysial and other bone dysplasias is eliminated very little genetic predisposition is demonstrable. The disorder appears in only 1% of first-degree relatives and with no excess at all in second and third degree relations.[8] Surprisingly and inexplicably there appears to be an excess of abnormalities of the groin and genitourinary system among both patients and their near relatives.[9] Recent painstaking anthropometric studies have shown that at the time of the onset of their hip disease many affected children are retarded in skeletal maturation and growth.[10-13] As judged from hand radiographs skeletal age may remain at a standstill for three or more years, although later it catches up completely or nearly so. There is associated delay in growth, the children being smaller in every dimension except head circumference, but particularly distally in the limbs.[14] In one study[8] children with Perthes's disease appeared to suffer from an unduly high proportion of unfavourable environmental factors. Before birth malposition was some three times more frequent than normal. They tended to be born late in their family, from parents of higher than usual age, and into low-income households.

The frequency of bilateral disease has long suggested the existence of a predisposition to the disorder in at least some patients. We now have evidence that perhaps in most cases there is a general abnormality of skeletal development at the time when the hip disease starts. Many questions remain. How does delayed skeletal maturation increase the risks of the period when the blood supply to the femoral epiphysis depends exclusively and precariously on the lateral epiphysial vessels? Does the more rapid skeletal maturation seen in girls and negroes confer protection? Do the environmental handicaps act by delaying skeletal development? How important are other, perhaps hormonal, causes of impaired growth? We must hope that these new clues will lead to a fuller understanding that may allow the effective prophylaxis that is so needed for a condition often unsatisfactory to treat.

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What should he eat, doctor?

Relatives looking after a patient with acute myocardial infarction at home will almost invariably ask the doctor what he should eat. Equally commonly his answer will be to give the patient "light meals only," perhaps restricting fats and cholesterol—advice which is as bland as the diet, although recommended in many standard textbooks.[1] Even in coronary care units dietary policy tends to be vague if not non-existent. Surprisingly we still seem to be in the dark about the correct regimen to give the patient in the acute phase of the illness. The problem has been highlighted by the results of a recent survey[2] in the USA, which showed that the diet given to patients with acute infarction varied considerably from hospital to hospital, although 60% of institutions did have a "routine CCU diet." Most hospitals in Britain give such patients the ordinary ward diet, perhaps with some restriction of calories.

There are two separate aspects to the diet of the patient with

myocardial infarction: firstly, the diet in the acute phase (when the serious metabolic disturbances in the myocardium might well be influenced favourably or otherwise); and, secondly, the long-term restriction of animal fat in the hope of secondary prevention of coronary artery disease. Although clinically the role of increased circulating concentrations of free fatty acids present in the first 24 hours after myocardial infarction[3] remains controversial, experimentally they are associated with cardiac arrhythmias, increased myocardial oxygen consumption, decreased contractility, and increased size of the infarct.[4] A further rise in the plasma concentration of free fatty acids might be produced by a diet rich in cholesterol and fat, or by prolonged fasting. We know that the free fatty acid concentrations are reduced after patients with an infarction have had a normal meal[5] and this is a good reason to ensure that patients eat an adequate diet in the acute phase.

While intracellular imbalance of sodium and potassium is also an important feature of acute infarction, serious abnormalities of the plasma electrolyte concentrations are uncommon. The development of cardiac failure will lead to retention of sodium and fluid and a case could be made for the prophylactic restriction of added salt. Loss of potassium through the cell wall accompanies anoxic damage and may result in electrical instability. Attempts to prevent this effect by the intravenous infusion of a potassium, glucose, and insulin regimen gave disappointing results,[6] and it is unlikely that giving potassium supplements by mouth could appreciably influence its concentration in the cells. On the other hand, hypokalaemia that develops owing to treatment with diuretics can be prevented by additional potassium.

What conclusions can we draw about diet for these patients? The main one seems to be that we are still ignorant about their dietary requirements. Possibly this is a matter of no importance, but until we know this for certain it would seem sensible to give our patients a diet rich in carbohydrate and low in fat and cholesterol with restriction of added salt. They should also avoid fasting and, if they are disinclined to eat, glucose drinks may be a helpful alternative. But this is another example of a common condition about which we know little and on which research would pay valuable dividends.

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Arginine vasopressin in health and disease

The posterior pituitary antidiuretic hormone, arginine vasopressin (AVP), normally controls the urine concentration by regulating the permeability of the collecting ducts to water. Nevertheless, a urine more concentrated than plasma can be formed without it. In the event of only a small amount of tubular fluid reaching the diluting site in the ascending limb of the loop of Henle (where more sodium is reabsorbed than water), the scope for dilution is limited, and the subsequent need for AVP is diminished. Thus the urine of patients with diabetes insipidus treated with diuretics and salt restriction becomes paradoxically more concentrated because glomerular filtration falls, proximal salt and water reabsorption increases, and less tubular fluid is delivered to the diluting site.[1]

Pituitary diabetes insipidus is rare; it may be caused by tumours in or near the pituitary, cerebral trauma, meningitis, or granulomatous deposits. Renal diabetes insipidus, the result of renal insensitivity to AVP, is rarer still; usually an inherited condition commoner in male members of affected families,[2] it has occasionally developed as a result of acquired disease such as myeloma with hypercalcaemia and Bence Jones proteinuria[3] and amyloidosis.[4]

Excessive secretion of AVP is more common than deficiency but it is much harder to define, except when it is due to ectopic production of AVP-like polypeptides by an oat-celled bronchial carcinoma (or rarely by another malignant tumour). The associated extracellular hyponatraemia is clinically important because water moves into body cells, which are now relatively hyperosmolar; the brain reacts to overhydration with symptoms of raised intracranial pressure—confusion, convulsions, and eventually coma. Ectopic secretion of AVP-like polypeptides can usually be diminished by radiotherapy or chemotherapy; parried by water restriction; or antagonised by lithium or demeclocycline,[5] which inhibits AVP-sensitive medullary adenylcyclase in the collecting ducts. Acute symptoms of cerebral overhydration demand immediate treatment with hypertonic saline.

Hyponatraemia is, however, more commonly a complication of acute illness than of carcinoma, especially in the elderly. Usually it is associated with infections of lungs or central nervous system. In such patients the urine is frequently more concentrated than plasma, suggesting "inappropriate secretion of antidiuretic hormone."[6] Though the renal disturbances induced by illness and dehydration may themselves sometimes be sufficient to explain the paradox, the plasma AVP concentration is in fact usually either high or at least detectable in these hyponatraemic patients[7] (whereas complete suppression would at first sight seem the appropriate response to their hyponatraemia). Judging the appropriateness of a plasma AVP concentration in a hyponatraemic patient is difficult, however, and the perplexed posterior pituitary responds as appropriately as it can to several conflicting claims. On the one hand, it releases AVP in response to dehydration as part of a co-ordinated response to preserve plasma volume; on the other hand, a fall in plasma osmolality inhibits AVP secretion. Preservation of plasma volume wins and AVP secretion is not completely suppressed—if at all. Normally treatment of the primary illness rapidly reverses this hyponatraemia without recourse to the measures which are necessary to combat ectopic secretion of AVP.[7]

Pain and discomfort may play a larger part in stimulating AVP release in acute illness than has been realised. That AVP is released in response to surgery has long been recognised and attributed to trauma[8] and anaesthesia,[9] but pain by itself triggers release of AVP in man[10] as in animals. Here, then, is another good reason for keeping patients as comfortable as possible—but preferably not with narcotic analgesics, which may themselves stimulate AVP release.

Inability to concentrate the urine to a normal maximum is common in real disease but is rarely of great symptomatic importance, other than contributing to nocturia. Impaired urinary concentrating ability is, however, a useful marker in clinical diagnosis. Prolonged water deprivation is the most potent stimulus to urine concentration, but it is also the least acceptable diagnostic test. For many years an intramuscular injection of vasopressin tannate in oil provided a more convenient clinical test, and this has now been replaced by intranasal synthetic deamino-D-arginine-vasopressin (desmopressin).[11,12] Intranasal desmopressin is also an excellent treatment for pituitary diabetes insipidus[13] and has potential as treatment for nocturnal enuresis.

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