costerone[3] lower both the concentration of sodium in sweat and the rate of sweat formation, while spironolactone antagonizes these effects.[3] A low concentration of sodium in the sweat is therefore predictable when hypertension is associated with increased secretion of aldosterone, as in Conn's syndrome[4] and in some patients with severe renovascular hypertension.[5-7] On the other hand, secondary aldosteronism has not been found in cases of uncomplicated hypertension of undetermined cause, in which a reduced sweat sodium would be unexpected.

The electrolyte composition of sweat is also much influenced by the rate of sweating. At low rates of production sweat is very hypotonic, with a sodium concentration as low as 20-30 mEq/l., and as the rate of sweating rises its sodium content rises until at very high rates sweat is almost isotonic.[8] However, salt depletion arising during heat exposure results in a fall in both the rate of sweating and the concentration of sodium in it, and this fall is accompanied by an increase in urinary aldosterone excretion, which can be further augmented by restriction of dietary salt.[9]

Now Maracaibo is near to the equator and at sea level, and in addition the hypertensive patients were with few exceptions being treated with thiazide diuretics. It is not unreasonable to assume that they were salt-depleted in comparison with the control subjects, and the results may well be different when the work is repeated in a temperate climate and with untreated hypertensive patients. This needs doing, but a more interesting implication now emerges from this study.

Evidence exists for regulation of aldosterone secretion through the renin–angiotensin system[10 11] and for an inverse relation between plasma renin and plasma sodium.[12] Raised secretion of aldosterone found in patients with severe renovascular hypertension has been associated with hyponatraemia and with increased plasma renin in the limited number of patients in whom this has so far been measured.[11] An increase in plasma renin occurs also in the hyponatraemia and secondary aldosteronism associated with heart failure treated with diuretics. An exception is Conn's syndrome, in which hypertension and hypernatraemia due to a primary increase in secretion of aldosterone are associated with depressed levels of plasma renin.[11]

The estimation of sodium in sweat is simpler than assay of renin in the plasma. It might even be a more sensitive indicator of renin–angiotensin activity than direct assay itself. Clearly the sweat sodium deserves further study.

Rehabilitation and Strokes

Recent improvements in the treatment of the lethal accompaniments of a cerebrovascular stroke have led to a larger number of survivors than formerly and make the problem of their rehabilitation more pressing. Some degree of rehabilitation is possible and desirable in almost all cases. The goal to be aimed at, though obviously not always attainable, is physical, and, if possible, economic independence. The degree of physical disablement—that is, the amount of brain damage and the extent of secondary skeletal changes as well as concomitant disease not primarily related to the stroke—is one important factor in deciding how much rehabilitation is feasible. However, the psychological make-up of the patient may be almost as important. Few cases are beyond help. In the great majority the possibility of rehabilitation must be explored to the full.

Since the secondary effects of immobility on joints and muscles are a special hazard for the patient who has had a stroke, the sooner rehabilitation begins the better. Some strokes are so mild that encouraging the patient to voluntary activity and mobility is all that is required. Here the family doctor, the district nurse, and relatives and friends have a most important role. Indeed, for the more severely affected patient leaving hospital the family and the family doctor can make all the difference to how nearly the final goal of independence is reached. Staying in bed may be bad treatment, unless there are other indications for it. In fact, in maintaining independence for the patient with established hemiparesis or paraparesis bed is the enemy. If intercurrent illness occurs simple supervised mobility, such as intelligent relatives can give to the paralysed part, and the minimal possible stay in bed are essential. If flexor or extensor spasms, muscle contractures, or joint stiffness appear to be developing, the expert help of the physiotherapist should be sought without delay. The timely use of analgesics to allow active or passive movement which is otherwise painful will bring benefit later.

The more severely afflicted patient will probably be treated initially in hospital. Here again rehabilitation should be begun early. Clearly there is a time at first when the patient should be left in peace: but if he is expected to survive the acute stroke it is a shortsighted kindness to leave him for long without some physiotherapeutic assessment and simple treatment. This is increasingly recognized in the management of strokes as such. It is sometimes disastrously forgotten when the patient with a stroke is admitted with some unrelated acute condition. There is of course an obverse to this coin. It is unkind and wrong to lavish physiotherapy on a dying patient. The important decision, which has to be made early, is the prognosis for survival.

Formal physiotherapy is the backbone of initial and continued rehabilitation, but it is not all. Occupational therapy has a part to play, especially in re-educating the patient to make fine movements or when some extra psychological stimulus is needed. Here the co-operation of the doctor in hospital or at home is important in guiding and adapting the particular form of occupational therapy employed. Encouraging the patients to do for themselves is also an essential part of the rehabilitation process. Sisters and ward staff in hospital can make or mar this, just as relatives, doctors, and district nurses or health visitors can in the home.

Finally, no amount of physiotherapy or occupational therapy is a substitute for the activities of everyday life, so these must be made the vehicle for rehabilitation as soon as possible. While there is often a case for a short period of domiciliary treatment either just after discharge from hospital or to try to avoid admission to hospital, this should always aim at channelling rehabilitation into normal activities. If there is any doubt about return to work or household activity it is always best to try and see. Ideally the attempt should be by way of graded activity, and the social worker can often help over this. The time factor here too must be borne in mind: the longer the patient is away from his usual routine, whether of work or daily living, the more difficult and time-consuming will be the return. In the patient's and the community's interests a little practice is worth a lot of speculation here. But practice must be realistic, and if the patient obviously cannot manage the task set, a strategic change of objective should be introduced early. An unrealistic optimism about the patient's performance brings only discredit to the process of rehabilitation and its proponents