several speakers thought, was the creation of a new career grade, a permanent post at a lower level than a consultant. But this is a contentious matter, and even the conference could reach no conclusion on it. On the other hand, some speakers considered that doctors selected for specialist training should be chosen much earlier than at present and required a more formal course of training—for example, on the lines suggested by Professor G. A. Smart. Sir Robert Aitken considered that these new training schemes would be best administered by a small regional committee composed of a representative from both the universities and the Colleges.

Several of the ideas produced at this conference were brought out into the open for the first time. Unlike some of those canvassed at other conferences, most of them could be put into effect without too much difficulty or expense. Some people may argue that the present, with its shortages of doctors and money, is not the time to do this. Many doctors who have emigrated, however, have emphasized the lack of proper training in Britain as one of the main reasons for their going. Organized schemes of vocational training might do more to halt the "brain drain" than many people would think possible.

Early Diagnosis of Retinoblastoma

It would be difficult to imagine a more tragic malignant disease of childhood than retinoblastoma, for even if the threat to life is not realized the daunting sequel of partial or total blindness remains. But early diagnosis gives a chance of successful treatment.

The disease is congenital in origin, the tumour arising from the nuclear layers of the retina, and it usually develops from multiple foci in the eye.[1] On the average it occurs about once in 25,000 births, and has been considered to be bilateral in 20 to 30% of cases,[2] but as the survival period of patients with unilateral disease improves it is becoming clear that these figures need some revision. In a recent survey of cases seen in the Department of Pathology at the Institute of Ophthalmology, London, A. Taktikos[3] found the incidence of bilateral cases to be as high as 39%.

It is now apparent that the affliction need not be so threatening to life as was once thought. Though it frequently robs a child of one eye, the prognosis for life is improving as new methods of treatment are evolved. Whether or not prognosis is related to the degree of histological differentiation of the tumour cells remains an open question. Taktikos[3] found no such relationship, whereas D. H. Brown[4] found a direct relationship between prognosis and the degree of differentiation. On the other hand, there is no disagreement that prognosis is worse as the tumour spreads to ocular structures beyond the retina and to extraocular tissues.

Retinoblastoma may be inherited as an autosomal dominant characteristic with irregular penetrance.[5] Though most of the cases at present are sporadic, arising through spontaneous mutation, there is bound to be an increase in familial cases as patients who have recovered from the disease survive to reproduce. Thus a moral question enters the problem.

Early diagnosis is the cornerstone of successful therapy, and the ominous significance of a white pupil cannot be overstressed. More rarely, the tumour first attracts attention by an inequality of the pupils, by circumcorneal injection and lacrimation without iritis,[6] or by the presence of a squinting eye, especially if it is unable to fixate on a small light. All children so affected require a careful examination of the fundus under general anaesthesia with the pupils maximally dilated. Moreover, since the hereditary features of the disease are so tragic, the siblings of patients with retinoblastoma and the offspring of those who survive the disease should undergo similar thorough examination.[1]

The tumour usually appears as a raised yellow or pink mass in the fundus with prominent vessels on its surface. It may be nearly white in colour and there is commonly more than one tumour to be seen.

Treatment is usually directed to enucleation of the one eye in unilateral cases, for the disease is frequently far advanced when the diagnosis is made. When both eyes are involved the more seriously affected eye is usually removed and the other is treated with a view to destroying the tumour and yet preserving as much vision as possible. A treatment at present being used, especially in America,[7] is fractionated doses of *x* rays, directed through nasal and temporal portals to avoid the radiosensitive lens and trabecular meshwork, while H. B. Stallard[8] in Britain has pioneered the use of radioactive applicators sutured directly to the sclera overlying the growth. Both these methods may be augmented by giving radiomimetic drugs, and these are all that is available for the treatment of widespread metastases. The role of diathermy and photocoagulation is less well defined, but they have a place in special cases.

Now that therapy which will destroy the tumour and allow preservation of useful vision is available, all doctors have a special obligation to be aware of the possibility of retinoblastoma, and in this connexion the serious import of a white pupil in an infant or young child cannot be overemphasized.

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5 Francois, J., in *Ocular and Adnexal Tumors*, 1964, edited by M. Boniuk. Kimpton, London.
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Sweat Sodium and Blood Pressure

In a thought-provoking paper from Maracaibo, Venezuela, J. Quintero-Atencio[1] and his colleagues report that the sweat of hypertensive patients is less concentrated than that of the normal person. Not only that, but these workers go further and suggest that there is an inverse correlation between blood pressure and concentration of sodium in the sweat which even holds for blood pressures within the normal range.

The electrolyte composition of sweat is controlled by adrenal cortical hormones. Corticotrophin[2] and desoxycorti-

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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