

ing one to replace the Council, Sir Paul has no doubt been influenced by his experience in business. He is critical of the Representative Body's responsibility for every detail of B.M.A. policy. Its proper role, he maintains, should be to lay down the broad lines of policy, with the Council's successor—the small central executive—having the function of executing the policy in detail. These small committees, some elected by the R.B., others based on a particular craft, would have much power, and their members would have to devote ample time to them if they are to work properly. In a profession which is as heavily committed in its work as are doctors this means that many more doctors than at present would have to be prepared to play a serious part in medicopolitics both locally and nationally. If this led to greater knowledge among doctors of how the Service ticks this would be a gain. The risk is that a situation could develop where the B.M.A. was run almost entirely by professional committeemen supported by the secretariat.

The B.M.A. and the profession owe Sir Paul Chambers a debt of gratitude for his work on their behalf, particularly as it was done without any financial reward. Certainly his wide experience of business management, the obviously painstaking study he has made of other representative organizations, and his extensive consultation within the profession have ensured that his conclusions are solidly based on informed opinion. In presenting his report to the Council Sir Paul said that he had quite deliberately omitted a summary, for he wanted all doctors to read the report as a whole. He feared that summarizing it would lead to hasty judgements from a failure to appreciate the arguments underlying his proposals. The Council is to debate the report at a specially convened meeting in July, when the views of the Association's various standing committees will be available. A Special Representative Meeting will be held in November, at which the profession at large will consider Sir Paul's report and the Council's advice. As well as being published in the *B.M.J.* the report is being sent by the B.M.A. to non-members.

With the new shadow authorities for the reorganized N.H.S. being set up in 1973 the Association has none too much time to decide whether the hair shirt it bespoke in 1970 is the right fit and fashion for the rest of the century.

¹ *British Medical Journal Supplement*, 1970, 2, 206.

² *British Medical Journal Supplement*, 1957, 2, 14.

³ *British Medical Journal*, 1968, 1, 534.

⁴ *British Medical Journal*, 1903, 2, 104.

T₃ Thyrotoxicosis

The thyroid gland secretes two hormones, thyroxine (T₄) and triiodothyronine (T₃). Thyroxine is strongly bound to serum proteins—about 15% to thyroxine-binding prealbumin (TBPA), 75% to thyroxine-binding globulin (TBG), and 10% to serum albumin.¹⁻³ Approximately 0.05% is "free" or unbound.⁴ T₃ is much less strongly bound to proteins than T₄, so that a much greater proportion will be free. The biological potency of T₃ is three to four times greater than that of T₄, but the duration of the effect is shorter.⁵ In normal persons T₄ and T₃ are present in the blood in a ratio of about 50 to 1, and both hormones contribute to the maintenance of a normal metabolic rate. In some individuals T₃ is the only thyroid hormone secreted.⁶⁻⁸

In hyperthyroid patients both hormones are usually

increased and the ratio of T₃ to T₄ is higher, though the concentration of T₃ does not equal that of T₄.⁹⁻¹⁰ There is recent evidence to suggest that hyperthyroid patients pass through a stage of high circulating levels of T₃ before developing overt thyrotoxicosis.¹¹ Furthermore, high T₃ levels may persist in thyrotoxic patients rendered euthyroid with treatment. Such patients have persistent raised serum concentrations of the long-acting thyroid stimulator, and their radioactive iodine uptakes fail to show suppression with triiodothyronine.¹² In hypothyroidism the serum T₃ concentration may be within the normal range, and indeed there seems to be sequential failure in the production of first T₄ and then T₃ as hypothyroidism becomes more severe.¹³ It has been shown that in about one-half the patients with low serum T₄ concentrations after iodine-131 therapy a normal metabolic rate is maintained by adequate or raised T₃ concentrations.¹⁴

Though no thyrotoxic patients have been encountered with an increase in serum T₄ but no increase in serum T₃, clinical thyrotoxicosis does occur with normal serum T₄ concentrations and an increase in that of T₃.¹³⁻¹⁹ This condition has been called T₃ toxicosis, though the term T₃ thyrotoxicosis is more appropriate. Most of these individuals have toxic nodular goitres, but more recent reports have included many examples of classical Graves's disease.²⁰

Diagnosis in these circumstances presents certain difficulties. The radioactive iodine uptake may not be outside the normal range, as the formation of T₃ requires less iodine than T₄. The serum concentrations of protein-bound iodine and thyroxine will be normal as well as the T₃ resin uptake provided there is no abnormality of thyroxine-binding globulin. However, in none of the patients in whom T₃ suppression tests have been carried out has the radioactive iodine uptake been suppressible by T₃.¹⁴

Though the concept of T₃ thyrotoxicosis is not new, few cases have been reported. Now that methods of measuring T₃ are becoming available,¹⁵ more cases are likely to come to light. Eight further examples have recently been reported from the Mayo Clinic,²¹ and an even larger series of 40 patients has now been published from the New York School of Medicine.²⁰ Clinically most of them were only mildly thyrotoxic. The conventional laboratory investigations did not confirm the clinical diagnosis of hyperthyroidism. All these patients had normal values for total and free T₄ and normal levels of thyroxine-binding globulin. It was not until raised serum levels of T₃ were obtained that the clinical diagnosis received adequate explanation.

There seems little reason to assume that cases of hyperthyroidism with normal serum T₄ and high serum T₃ concentrations represent a basically different variety of Graves's disease. The factors favouring T₃ production have not been fully defined. However, studies in rats that were made iodine-deficient showed a progressive increase in the ratio of T₃ to T₄ during the period of iodine deficiency,²¹ and a similar finding has been reported in man when iodine intake is restricted.¹¹ Such a shift in favour of T₃ production is teleologically sound, since T₃ has four times the metabolic activity of T₄, while utilizing only 75% as much iodine. A sixfold increase in hormonal effect for the same amount of iodine could thus be achieved, and this would obviously be advantageous in conditions of iodine deficiency. Whether this is relevant to T₃ thyrotoxicosis is uncertain.

The importance of this entity lies in the need for clinicians to be aware of the existence of a state of hyperthyroidism in which the conventional tests of thyroid function are

normal. Until serum T_3 measurement becomes more readily available, the best method of screening will be the T_3 suppression test regardless of the initial uptake value. If there is a failure of suppression in a clinical situation suggestive of hyperthyroidism, T_3 thyrotoxicosis must be seriously considered.

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Health Care of Paraplegics

Paraplegics are a particular group of patients for whom the health care has undergone revolutionary changes since the second world war. The change has been largely due to the emphasis given to three main factors by Sir Ludwig Guttmann and others working with them. These are, firstly, a regimen designed to avoid complications such as pressure sores and urinary infection; secondly, social and psychological rehabilitation; and, thirdly, removal from hospital as soon as possible to prevent institutionalization.

Almost all paraplegics spend the acute phase of their disablement in hospital and at the present time most of them will receive their hospital care in one of the special units similar to the National Spinal Injuries Centre at Stoke Mandeville. Therefore their medical and social needs are capable of expert assessment, and if on discharge these needs are not met it must be due to either a failure of communication between hospital and community or a lack of community care services. R. H. Johnson and G. S. Johnson¹ recently suggested that the unsatisfactory level of social readjustment found in their survey in the west of Scotland was partly due to the absence of the statutory requirement on local authorities in Scotland to keep a register of disabled persons, but this argument may be questioned. Experience, especially with "at risk" registers, has shown that registration is effective only if the information is passed on to the operational level for action. It would seem much more important for the hospital to prepare a check list of the patient's medical and social needs and potentials and pass

this on to his general practitioner for him to relay to the health visitor. It is the health visitor who is the most likely community care professional to be in regular contact with the paraplegic and in a position to summon appropriate medical or social services when required. It would also seem useful if the health visitor could visit the patient in hospital before discharge. From Johnson and Johnson's paper it would appear that in Scotland communications are still insufficient, as was found some years ago by A. Forder and colleagues² in the Liverpool area.

Opportunities for employment are not yet so good as they should be for disabled persons, and it is pleasing to learn that some reorganization is proposed. It would be helpful if the posts of disablement resettlement officer were made as specialist appointments and if the incumbents would remain in this work as a career. At present these posts are on the promotion ladder of the Department of Employment, and the staff change frequently. Here also the communication link by personal interest is vital and could be the responsibility of the health visitor. The tendency in getting a disabled person into employment is for both the patient and his professional advisers to give up too easily. An objective agreed by the hospital, the patient, and the community care team is needed, and they will then be able to pursue it with greater determination.

Most local authorities are more aware than they used to be of the considerable needs of the disabled, and are striving with the help of voluntary bodies to develop services to cope with them. It is timely that the need for employment should be emphasized as this is a vital factor in social rehabilitation, particularly for any young disabled person.

¹ Johnson, R. H., and Johnson, G. S., *British Medical Journal*, 1972, 1, 779.

² Forder, A., et al., *Social and Economic Administration*, 1969, 3, 3.

Trapped Nerves

The entrapment syndromes occur when a peripheral nerve lying in a confined space suffers chronic local pressure from neighbouring tissues. The injury may be direct to the nerve trunk or through damage to its blood supply, and the clinical features are determined by the type of nerve involved. With a predominantly sensory nerve paraesthesiae and pain may occur. Pain is localized to the area of distribution and may be sharp or burning. Paraesthesiae can be associated with hypoaesthesia, hyperaesthesia, or contact dysaesthesia. Later the skin texture may alter. When a predominantly motor nerve is affected the pain is dull and less well localized, and atrophy of the muscle supplied may follow though the patient is unaware of weakness. In a mixed nerve the sensory component often predominates until weakness and wasting are advanced. Electromyography, by showing partial denervation of a particular muscle or slowed conduction velocities over the compressed segment of nerve, can provide useful confirmatory evidence.

In chronic lesions of peripheral nerves opinions vary considerably about invoking entrapment as the mechanism. The median nerve in the carpal tunnel and the ulnar nerve at the elbow are familiar and generally accepted examples. The tarsal tunnel syndrome from compression of the posterior tibial nerve at the ankle, and entrapments of the ulnar nerve at the wrist and the median at the elbow, are less