THYROTOXICOSIS

The Present Position

By J. W. LINNELL, M.D.(Camb.), F.R.C.P.(Lond.)

Consulting Physician Metropolitan Hospital; Honorary Consultant, L.C.C. Thyroid Clinic, New End Hospital, Hampstead

and

RAYMOND GREENE, M.A., D.M.(Oxon.), M.R.C.P.(Lond.)

Physician, Metropolitan Hospital and Royal Northern Hospital; Consulting Physician, L.C.C. Thyroid Clinic, New End Hospital, Hampstead

One of the authors of this paper is a physician who, for the last 25 years, has been interested in the subject of thyrotoxicosis. Associated for most of this time with thyroid surgeons, his outlook is chiefly that of a clinician. The other is likewise a physician but, being primarily an endocrinologist, his interest has been chiefly in the experimental and theoretical aspect. Now working side by side at the L.C.C. Thyroid Clinic on the practical problems which constantly arise and make thyrotoxicosis such a fascinating study, we venture to put forward our views on its present position with the knowledge that we are merely touching the fringe of a vast region, much of which is still unexplored.

Aetiology

It must be admitted that we are still very ignorant of the causes of thyrotoxicosis. Two lines of enquiry seem to offer hopes of ultimate solution—the one, a combined geological, genetical, statistical and pathological investigation of the relationship between thyrotoxicosis and either pre-existent thyroid abnormality or familial tendency thereto—the older line of investigation with which the name of McCarrison is most prominently linked; the other, a newer psychological and neurological approach, in which prominence is given to psychological disturbances and their possible repercussions on the hypothalamus, ‘the conductor,’ as Sir Walter Langdon-Brown put it, ‘of the endocrine orchestra,’ but also of the autonomic nervous system.

Geographical and Geological Clues

There is no doubt that thyrotoxicosis occurs more commonly in those areas in which simple endemic goitre is most prevalent. In England there was formerly a ‘goitre belt’ extending, in rural areas, from Cornwall, north-eastwards through Somerset into Oxfordshire between the Cotswolds and the Chilterns, through Buckinghamshire to Northamptonshire and thence northwards to Derbyshire and up the Pennine Chain (Stocks, 1928). There are offshoots from Wiltshire to the Isle of Wight, across Herefordshire, into South Wales and across Cheshire into North Wales. The incidence of goitre in this area is less obviously excessive than in years gone by, a change which is usually attributed to the improved distribution of fish, with its high iodine content, and to the betterment of the water supply, for contaminated water is a bad waster of iodine (McCarrison, 1928). Other endemic areas have been described. Of these the most important are, in Europe, the Alps; in Asia, the Himalayas and the Punjab plains, where the older Aravalli rocks penetrate the later alluvial deposits; in Africa, the Sudan, Egypt and Sierra Leone; in America, the basins of the Great Lakes and the St. Lawrence; and in Australasia, the mountainous areas of New Zealand.

The cause of the goitrousness of these areas is generally admitted to be, at least in part, iodine deficiency. This may be an absolute deficiency in the soil and therefore in the water, or a relative deficiency due to contamination of

the water, or to the presence in the soil and therefore in the water of undue quantities of fluorine, which is capable of displacing iodine and may possibly thus induce a physiological deficiency (Wilson, 1941). All such cases of deficiency may be overcome by so simple a factor as a weekly call to an isolated village by a fishmonger's van (Young, quoted by Wilson).

Given, however, the fact that thyrotoxicosis is often superimposed on a simple goitre, we are left with the question of why some simple goitres become toxic while others do not. One of us is intimately acquainted with the formerly highly goitrous area where Buckinghamshire borders on Northamptonshire and Bedfordshire, where recent surveys have shown a high incidence of fluorosis. Here the deaths certified as due to thyrotoxicosis in the period 1913-19 were only 10 to 12 per million (compared with 22-24 in West and Central Wales) and, in 1936, 40 to 50 per million (compared with over 100 in West and Central Wales). Yet a noticeable proportion of the older villagers have enormous and obviously harmless goitres. Though a large proportion of simple goitres eventually become toxic, they do not all do so, a matter to which we shall refer again later. Moreover, thyrotoxicosis may be 'primary,' arising not upon the soil of a simple goitre, but of a gland apparently normal. Further clues must be sought.

Genetic Clues

However clear may appear to be the relationship between iodine deficiency and goitre, the influence of heredity must not be neglected. The railways have brought fish to inland villages, but they have also shown the inhabitants that there are other pebbles on the beach, and have reduced in-breeding. Martin and Fisher (1945) have produced strong evidence for a single recessive gene favourable to the development of primary thyrotoxicosis, the disease actually appearing in those carriers of the gene who are subjected to, perhaps, mental shock, infection, or endocrine imbalance, as at the menopause. The evidence for a genetic factor is less clear-cut in nodular toxic goitre, though affected relations are more common than in the general population. There is a growing tendency among clinicians to minimize the significance of nodules and it may be that

future genetical research will support the essential unity of the two diseases.

Another genetic clue may be found in the fact that the maps of exophthalmic goitre mortality produced by McEwen bear a startling relationship to the ethnographical maps which show stature and pigmentation. The areas with the highest death rate from thyrotoxicosis are the areas of greatest 'brunetness,' the areas, in fact, in which the former British inhabitants of these islands have become least mixed with later blond invaders (Roberts, 1938). We have ourselves been impressed by the remarkable number of cases of thyrotoxicosis referred to New End from one small area in South Wales, though we naturally bear in mind the disturbing influence on our figures of the diagnostic acumen of the local doctor.

Endocrinological Clues

The thyroid gland is under the direct control of the anterior pituitary gland, which secretes a so-called thyrotrophic or thyrotropic hormone. Both words are unfortunately chosen, for the pituitary hormone neither 'nourishes' nor 'looks towards' the thyroid but stimulates it into activity and should, with greater propriety, be called 'thyrokinetic.' In the absence of sufficient thyrokinetic hormone (as in experimental animals subjected to hypophysectomy and in human beings with Simmonds' disease and other forms of hypopituitarism) the thyroid atrophies and the symptoms of hypothyroidism appear. Injections of the appropriate pituitary extract restore the thyroid to normal. Injections of this extract into normal animals cause hypertrophy and hyperactivity of the thyroid and many of the symptoms of thyrotoxicosis, including exophthalmos. Indeed according to some observers exophthalmos is more easily produced by pituitary extract in thyroidectomized than in intact animals, and it can be caused even after section of the cervical sympathetic chain. The histological changes in the thyroid induced by the injection of pituitary extract are identical with those found in Graves' disease. These facts suggest that the symptoms of Graves' disease are due in part to over-stimulation of the thyroid by the anterior pituitary. Two difficulties in the acceptance of this view occur. In the first place experimental work

has shown that animals develop an immunity to thyrokinetic pituitary extracts. It does not follow, however, that such an immunity would develop in the naturally occurring disease. In most of the experiments heterozoic extracts were used and in such circumstances the production of immunity would be expected. Moreover it must be remembered that these extracts are artefacts of a protein nature; the natural stimulant may have a different chemical composition and may even not be a protein at all. The second difficulty which has arisen in the acceptance of the hypothesis is the fact that undue amounts of thyrokinetic hormone have not been demonstrated with certainty in the blood of thyrotoxic patients, a negative argument which does not invalidate the hypothesis, but does postpone its acceptance.

The relationship of the thyroid to the anterior pituitary has become clearer through recent experiments with 'goitrogenic' substances such as thiouracil. The administration of such drugs to animals has been shown to interfere with the synthesis of thyroxine by the thyroid. The drugs are not antidotes to thyroxin, for they do not interfere with the action of thyroid in the treatment of myxoedematous patients. The deficiency of thyroxine in the blood causes an increase in number of the basophil cells of the anterior pituitary with a vacuolation exactly similar to that which occurs in experimental animals exposed to cold. So great is the increase in number and activity of the basophil cells that the acidophil cells are almost crowded out. This effect is only temporary, and may be inhibited by the simultaneous administration of thyroxine and thiouracil but not of sodium iodide and thiouracil. The last link in the chain of events connecting the pituitary and thyroid glands is thus forged. The thyroid activity is controlled by the secretion of the basophil cells of the anterior pituitary, which are themselves controlled by the level of circulating thyroxine.

The thyroid is intimately linked with other glands than the pituitary. The islets of Langerhans may be called upon to deal with the hyperglycaemia which often accompanies excessive thyroid activity. The relationship between the thyroid and the gonads is shown by the enlargement of the former so common in girls at puberty and in women during

pregnancy and the premenstrual phase; menstrual function may be affected in both hyperthyroidism and hypothyroidism; and in men myxoedema is often associated with depression of the sexual function and of the production of sperm. In the response of the body to cold the thyroid works in partnership with the adrenal medulla and there is evidence that its activity is in some circumstances inhibited by the adrenal cortex. The function of the thyroid and the parathyroids are complimentary in respect of calcium and phosphorus metabolism. None of these interrelationships, other than that between the thyroid and the anterior pituitary appear to be of significance in the aetiology of thyrotoxicosis, unless perhaps the many points common to thyroid crisis and adrenal crisis may point to a relationship of greater significance than has hitherto been thought. Nevertheless they are important in explaining many symptoms which, escaping the 'classic' list, escape also adequate treatment. There is no disorder in which it is more necessary for a physician to be a *holist**; to remember that the patient is more than the sum of his parts, and that concentration on his thyroid gland will often leave much of his illness still untreated.

Neuropsychiatric Clues

It is well known that the beginning of thyrotoxicosis can often be related to mental disturbances. 'The worry and fatigues associated with the nursing of a near relative through a long illness and the silent struggle with financial difficulties are frequent factors. Perhaps the most potent forms of mental disturbances are those associated with the emotions, the unhappy marriage with its continued irritations and inhibitions, the broken engagement, and the fear of undesired pregnancy' (Fraser and Dunhill, 1937).

It is through the hypothalamus and the autonomic nervous system under its control that emotions find their somatic expression, and it is therefore relevant to ask what clues there may be which link it with thyroid overactivity. Many of the symptoms of thyrotoxicosis—those of autonomic imbalance—may be produced by electrical stimulation of the hypothalamic nuclei or mimicked by encephalitis in this region of the brain. Experi-

* *Holist* : One who believes that the whole may be greater than the sum of the individual parts

mentally thyroxine in excess has been found to produce toxic changes in the brain stem. Narcotics acting on the hypothalamus have little influence on the B.M.R. of normal people or on their reaction to insulin or on their blood cholesterol, whereas in sufferers from Graves' disease the drop in the B.M.R. may be as great as 50 per cent. and the insulin resistance and blood cholesterol show greater changes (Fenz, quoted by Boon, 1938). Such results might be due to a narcotic effect on hypothalamic centres thought to control thyroxine production or to a reduction of the sensitivity of these centres to thyroxine. It is certainly within the bounds of probability that thyroxine acts on the hypothalamus as well as on the peripheral nervous system. Boon (1938) has suggested that in thyrotoxicosis a hypersensitive hypothalamus, reacting to a normal blood-thyroxine, causes, via the carotid plexus and the pituitary, a still greater output of thyroxine, thus initiating a vicious circle. On this hypothesis it is easy to explain both the influence of mental strain in initiating the disease and the occasional cures produced in early primary cases by rest, psychiatric methods or thiouracil. It is, on this hypothesis, improbable that a permanent cure could be produced in long-standing cases by any known means other than thyroidectomy.

Symptomatology

To discuss in full the symptoms and signs of thyrotoxicosis hardly comes within the scope of this paper. They have been described on many occasions by first-rate authorities, and comparatively recently an excellent paper dealing with the heart in toxic goitre by Papp (1945) was published in this journal. We shall therefore content ourselves with drawing attention to the increasing importance which is being attached, both in America and this country, to the large group of patients with nodular goitres so little toxic that they are accounted by the profession in general as non-toxic. In this country their importance has been particularly stressed by workers at the L.C.C. Thyroid Clinic, and in America by several well-known workers at different clinics, whose views have been admirably summarized by Cole, Slaughter and Rossiter (1945). For many years there has been a strong belief at the L.C.C. Thyroid Clinic that not only do

the great majority of all such goitres eventually become toxic but that by the time early middle life is reached goitre, without some evidence of toxicity, is comparatively uncommon. The evidence, however, is, as a rule, only to be obtained through most careful observation since it may amount to no more than a few of the following symptoms and signs; a constant feeling of lassitude, occasional attacks of palpitations, a certain degree of unwanted emotional instability, irritability, and nervousness, generally noticed by relatives and friends rather than by the patient herself, loss of weight, irregular sweatings and feelings of heat, a slight, sustained increase of the resting or, better, sleeping pulse-rate, a fine tremor of the fingers, moist palms, and the suspicion of a stare due to retraction of the upper eyelids. The fact that their onset is generally insidious and that they are liable to phases of remission as well as exacerbation does not aid their discovery. It may well be thought that when such a goitre patient is the subject of neuro-circulatory asthenia or an anxiety state, or has arrived at the climacteric, it must be a matter of the greatest difficulty to tell whether she is thyrotoxic. This cannot be denied, yet though diagnostic mistakes are bound to occur, they become progressively fewer with increasing experience. When any reasonable doubt exists, help may be found in coming to a decision in the improvement or lack of improvement resulting from a three weeks' course of thiouracil judiciously administered. It may be argued that all that is needed is an estimation of the basal metabolic rate. Unfortunately, the fact of the matter is that the estimation of the basal metabolic rate is a highly fallible procedure. Not only is it inaccurate in many hands, but even when the value given is a true one it can give no indication of the level before the beginning of the disease. The increase in oxygen consumption represented by a rise of 20 per cent. is significant, but if this rise is from —20 per cent. to normal, the significance will be missed. In the slightly toxic patient whom we are considering, the rise, which must in every case be present, may be even less than this and its estimation is thus of little value. In view of a growing belief that all nodular goitres should be removed without undue delay, irrespective of their being toxic or not

on account of their possessing potentialities for evil in several directions, mistakes may prove less harmful in the future than they have done in the past.

By the terms of reference imposed on us by the title of this paper we are precluded from considering possible complications other than those which are thyrotoxic in origin. These are, however, of sufficient importance. We have every reason to believe that it is still far from generally known that after acute rheumatism, toxic goitre is the most fruitful cause of auricular fibrillation; that many cases so caused derive from goitres so slightly toxic that they are apt to be looked on as non-toxic is much less widely appreciated; that auricular fibrillation can occasionally be the first detectable sign of toxic change is probably known to few. Fibrillation caused by thyrotoxicosis is nearly always paroxysmal at first and only later becomes established, which is fortunate since established fibrillation, in the absence of treatment of the causal thyrotoxicosis, leads on inevitably to congestive failure. It is well worth remembering that a slightly toxic nodular goitre, the presence of which is often unrecognized, can be responsible for, or share in the development of fibrillation in a patient who is suffering from rheumatic heart disease, or who is the subject of hypertension. Instead of fibrillation, auricular flutter or auricular paroxysmal tachycardia can be caused by the mildly toxic as well as the frankly toxic goitre, but these are, in our experience, rare complications.

If we are pressed to supply proof of the truth of our assertions in regard to the danger of goitres generally considered harmless, our reply is that the proof lies in the dramatic improvement brought about by subtotal thyroidectomy; in literally scores of patients we have seen not only do all symptoms of ill-health disappear, but also auricular fibrillation—especially if it is still paroxysmal.

Treatment

There are some patients—in our experience they constitute a small minority—the subjects of primary toxic goitre, who will recover either with no treatment at all or with rest and sedatives and the removal of mental stress. Unfortunately, when faced with an individual

patient, one has no means of knowing if she belongs to either of these classes, and, again, there is no doubt that a number of such patients so 'cured' relapse later under conditions of stress, during pregnancy or the puerperium, at the menopause, as a result of an infective illness, or, occasionally, for no discoverable reason. Previous to the advent of thiouracil it was considered wise to watch the effects of a few months' rest and sedatives on any patient with primary toxic goitre, in whom the symptoms were of recent origin, before going further, but today such a course is probably seldom followed. Deep X-ray therapy, once so strongly advocated, has gradually fallen into disfavour as being useless in nodular and, at best, very undependable in primary toxic goitre. In spite of article after article by authorities insisting that iodine does not cure but only brings about a temporary amelioration of the symptoms and signs of thyrotoxicosis, so firm is the belief in its curative virtues that it is comparatively rare even now to see a thyrotoxic patient for the first time who has not been taking it on medical advice for weeks, months, or even years. That it has had, and still has, an important place in treatment immediately before, and possibly immediately after, operation, there is no doubt, and its use should, in our opinion, be restricted to these two periods. At the present moment for all practical purposes choice of treatment lies between thiouracil and surgery. Thiouracil, which was introduced by Astwood (1943) was greeted by the profession in general with almost unbridled enthusiasm as providing a safe means of cure, some well-known physicians declaring exultantly that thenceforth operation would be unnecessary save to relieve pressure symptoms or for cosmetic reasons. Ere long, however, voices were raised in many quarters both in America and this country contesting the extreme claims made for it and insisting that not only was there no convincing evidence that it cured but that it was by no means entirely harmless, since various complications attended its use in a not unimportant proportion of patients treated with it. The list of possible complications grew rapidly with increasing experience of the new drug, headache, nausea, vomiting, diarrhoea, pyrexia, splenic enlargement, various rashes, adenitis, swelling

of the salivary glands, jaundice, pains in the joints, purpura, anaemia, leucopenia, thrombocytopenia, agranulocytosis, myxoedema (which is reversible) and even heart-block being described by different workers. There is little doubt now that at first excessive dosage was the rule and that many of these complications were directly due to this, but judging by reports in the current literature, it would seem that even with the far smaller dosage in use today adverse reactions are to be expected in at least 10 per cent. of all cases treated. Most of these are, fortunately, of little importance, but some, for instance agranulocytosis, high fever, severe pains in the joints, jaundice, purpura and severe anaemia, and persistent vomiting, call for a cessation of administration of the drug. By far the most serious of the complications is agranulocytosis, which is said to occur in about 2.5 per cent. of patients treated and carries a death-rate of 25-30 per cent. Usually, but by no means always, it develops in the early weeks of treatment, appears to have little or no relation to dosage, and may come with alarming suddenness. At first it was believed that its approach could be predicted by means of frequent white blood counts, but now it is recognized that this is not the case since a certain degree of leucopenia is the commonest of findings in patients treated with thiouracil. It may be said at this point that there is general agreement that the mortality rate of agranulocytosis can be materially reduced by the administration of massive doses of penicillin, e.g., 500,000 units in the day. As regards results it can now be definitely said that good results are to be expected at first in nearly all cases treated, the symptoms and signs of thyrotoxicosis generally diminishing dramatically within a few weeks unless the patient has been given a course of iodine a short time previously, when the effect of the thiouracil will be much delayed. A very few patients are, in our experience, completely recalcitrant, and a few improve only up to a point and then either remain stationary or deteriorate. As regards later results, numbers of patients are enabled to resume and maintain their normal activities as long as treatment is continued. When it is discontinued most patients relapse sooner or later, and usually sooner rather than later. Re-

missions, lasting for months, however, are possible in a considerable proportion of cases where treatment is maintained for six or more months prior to its discontinuance, and there is no doubt that in a certain number of patients where perhaps in any case a spontaneous cure would have resulted, thyrotoxic symptoms and signs have been kept in abeyance till it has occurred. It is even possible, should the hypothesis of the vicious circle described in an earlier part of the paper prove true, that an occasional patient may, by prolonged induced hypothyroidism, be given an opportunity to escape from the circle; but of this we have so far seen no evidence.

As regards the effect of thiouracil on the size of the goitre this, in the case of a nodular toxic goitre, is practically nil; a primary toxic goitre may likewise be unaffected or it may decrease, sometimes to the point of disappearing, or it may increase, sometimes with alarming rapidity. Exophthalmos may remain stationary, decrease or increase. Auricular fibrillation disappears in a certain number of cases, especially if the treatment be persisted with. As regards the prospect of permanent cure, a matter to which we have already referred, in spite of the optimism of Meulengracht (1946) and others, the general opinion in expert circles is that thus far there is no convincing evidence that it occurs. Even the *Lancet* (1946), a confirmed adherent to thiouracil, considers that it is still too soon to dogmatize or to speak of a permanent cure. The duration of the treatment necessary to keep a patient in reasonable health is, therefore, unpredictable. Nor is this the whole story, for throughout the course the patient must, ideally, keep in close contact with her doctor, since not only agranulocytosis, but some other complication may appear without warning, at any stage, even if they usually appear—when they do appear—in the first few weeks of treatment. Especially should she be instructed to cease taking the drug and report at once to her doctor, should a sore throat, fever or a rash develop.

So much we believe can be said in all fairness of thiouracil in treatment, whether with the idea of cure or of remission of symptoms. Those who are still enthusiastic—and they are many—should, we think, read the report to the Council on Pharmacy and Chemistry made

by Van Winkle Jr. and his associates (1946), before making up their minds finally about it. It is the result of a survey of no less than 5,745 patients treated with thiouracil for various periods, and its conclusion is that on the basis of the available information it can only be recommended for use in pre-operative treatment and where operation is contra-indicated. To this conclusion we had ourselves come before the publication of this report. Not only had we had in mind the danger of complications, the uncertainty which prevails as to the length of treatment necessary and the improbability of cure, but the expense and tedium it causes the patient and the worry and anxiety the doctor, especially if the patient be careless or ignorant. Finally we had been influenced by the fact that a number of patients come to us not only with auricular fibrillation but congestive failure, that then time is often of supreme importance, and thiouracil too slow and uncertain in its action. Thus, in spite of our being physicians, it had not been long before we were convinced that surgery still held pride of place in the treatment of thyrotoxicosis. That thiouracil had an important place, too, we did not doubt, but generally speaking, only in preparation for safer operation, and not as a method of cure. As a matter of fact there is no need for its use in the preparation of the large majority of thyrotoxic patients, iodine being quite sufficient, but there are a considerable number of patients so toxic that they cannot be rendered sufficiently safe for operation by iodine and for whom operation by stages—a burden on patient and surgeon alike—would have been accounted necessary a short time ago; now, however, by the judicious use of thiouracil the large majority of them can achieve such a degree of pre-operative improvement that they can be operated on with little danger. In this connection it is well to remember that its effect on the gland substance is to make it extremely vascular and friable so that the operation becomes most difficult and trying unless iodine is given during the last two or three weeks to devascularize it to some extent. It is also our experience that thiouracil causes adhesions to form between the capsule of the gland and the surrounding tissues. In view of these facts, contrary to the practice that obtains in

many of the well-known American Clinics, we tend more and more to discontinue thiouracil once the gross thyrotoxic symptoms are controlled, switch over to iodine for a fortnight or so, and then operate. Thus far we have had no reason to regret our adoption of this procedure.

Coming more closely to the important subject of surgery, the best results can, in our opinion, only be obtained through teamwork. What is needed is a surgeon who is not only a technical expert as regards the operation but has made as deep a study of thyrotoxicosis as the physician with whom he should be associated. It is almost unnecessary to say that the latter in addition to being an experienced general physician should be a competent cardiologist. The anaesthetist too plays a very important role, for there is no operation in surgery for which there is greater need of special experience on the part of the anaesthetist than subtotal thyroidectomy. The theatre staff should be specially trained and the ward nurses not only qualified to deal with the many difficulties and occasional dangers which may almost suddenly arise before and after operation, but possess sympathy and consideration of a high degree and have the ability to inspire their charges with all possible confidence. When the margin of safety is often narrow, the smallest detail may make the difference between life and death. The common belief among surgeons that any competent general surgeon without special training in thyroid surgery is technically fit to perform an operation in which a small, but still important, proportion of cases presents great difficulties to the expert, we think ill-founded. That without special knowledge of the subject of toxic goitre and cardiology, he should consider himself competent to take medical, as well as surgical, charge of the patient before and after operation, we cannot allow. It is, we believe, the occasional thyroid surgeon who is chiefly responsible for the ill repute which is still attached to the operation. Apart from the tragically high mortality rate which often attends his well-meant efforts, such undesirable results as insufficient removal of gland tissue, severance of one or both laryngeal nerves, or chronic tetany are too common. Nor is he likely to realize the immense importance a

woman, even if she be of an age at which one would think that appearances matter little, attaches to the cosmetic effect. Over and over again it has been our lot to be accused by general practitioners of encouraging 'massacre' when we have publicly advocated surgery. This attitude is understandable. An analysis of the Registrar General's returns for 1936 shows that of the deaths from thyrotoxicosis in the whole of England and Wales, over 16 per cent. were operative deaths, and we have no reason to believe that it has greatly fallen since. This figure assumes that all death certificates issued mentioned operation as a contributory cause. It is probable that doctors did not always mention it if they considered that death took place despite and not because of surgery. Our attitude is that a good method of treatment ought never to be damned for a lack of skill on the part of those who use it, provided always that there are ample opportunities for such a state of affairs to be remedied. And these there are in abundance. We fully appreciate the fact that there are far too many thyrotoxic patients in the country for more than a fraction of them to be treated at existing goitre clinics, but, as Linnell, Keynes and Piercy (1946) have recently suggested, teams can easily be built up at most hospitals of standing on the lines we have indicated by surgeons who have made themselves competent to do thyroid surgery by watching experts and studying the many aspects of the disease. There should be, and we believe, need be no second-best in thyroid surgery. And in good hands there is no more satisfactory operation in the whole realm of surgery, the vast majority of patients being able to live approximately normal lives thenceforward. There must be occasionally cases of regrowth of the gland with a recurrence of toxic symptoms but, generally speaking, not only is the tumour removed once and for all, together with the anxiety its mere presence so often causes, but with it the danger of such complications as pressure, haemorrhage into its substance and carcinomatous change. In the great majority of patients thyrotoxic symptoms disappear rapidly, to recur no more; the risk of the onset of auricular fibrillation is practically abolished; where it is present there is a good

prospect of a quick return to normal rhythm, either as a direct result of the operation or of the administration of quinidine. Where it still persists the ventricular rate can almost always be satisfactorily controlled by digitalis and—a most important point, especially today—convalescence is relatively brief. As regards mortality, this is at the present time in the region of 1 per cent. in practically all the well-known clinics of the world, and this in spite of patients of all ages, of every degree of thyrotoxicosis, with and without auricular fibrillation, with and without congestive failure on admission to hospital, being accepted as candidates for operation. With the judicious use of thiouracil in the preparation of very toxic cases this mortality rate should fall still further, though it is unreasonable to suppose that it will ever reach a vanishing point; toxic crises can occasionally follow operation, and not every patient moribund through heart failure can be saved by it. It has been said that so great is the risk of operation in elderly patients with auricular fibrillation that it should not be attempted. This is not our experience. The patient who frightens us is the frightened girl.

We are not suggesting that by any method of treatment the patient is always completely cured, even though her hyperthyroidism may be perfectly controlled. One cannot make a silk purse out of a sow's ear, and the thyrotoxic patient is often, from the beginning, 'poor stuff'. Moreover, in preventing, whether by medical or by surgical means, the excessive production of thyroid hormone, we are leaving largely unaffected the cause of this excess. The psychological maladjustment or autonomic imbalance remain.

We cannot leave the subject of treatment without calling attention to the fact that recently a new remedy has been tried out in America with, apparently, considerable success. Hertz and Roberts (1946) and Chapman and Evans (1946) have published reports on the effects of freshly prepared radio-active iodine administered orally in a number of cases of toxic goitre. One dose of the liquid, which is practically tasteless, has in several patients proved sufficient to effect a 'cure.' It is impossible for us to express any opinion whatever

regarding the value of the treatment, since we have had no experience of it, but if the claims of these workers are corroborated, in the near

future it may take the place of surgery to a considerable extent and oust thiouracil from the place it now occupies.

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CORRESPONDENCE

THE DOCTOR AND THE NURSE

SIR,

I have read with interest your Editorial for the July number of the Journal. My personal experience of surgeons does not incline me to agree with your observations as to their meekness, or to accept as realistic your account of the conversation between sister and surgeon which you allege is a daily occurrence. Be that as it may, you have certainly raised an extremely important practical point in regard to the training of the student nurse, viz., the failure on the part of the vast majority of doctors on a hospital staff to realize their obligations in this vital matter. Medical men in general, and surgeons in particular have been accustomed for many years to a privileged position, in virtue of which they receive the slavish attention of their various technical assistants in ward or theatre, to say nothing of the adulation of a public brought up in the tradition of the sacrosanctity of the Medical Profession. They have accepted this, like so many of the privileged classes, as an inalienable right, and at the same time (again like so many of the privileged classes), they have failed to shoulder the responsibilities upon which alone the title to privilege should rest.

There are other points in your Editorial which give opportunity for discussion, but I hesitate to trespass upon your valuable space. The duty of the doctors in playing their part in the young nurses' education is one of primary importance and you, Sir, do well to call attention to it. I hope that others may be stimulated to contribute to this correspondence and to make some effort to jolt the Honorary Members of Hospital Staffs out of their attitude of complacent self-satisfaction.

I enclose my card, and remain, Sir,

Yours faithfully,
 ATHANASIOS, M.D.

THE NURSE AND THE DOCTOR

SIR,

I have been given a copy of your Journal so that I may read the Editorial: as I work in the theatre I wonder if you would be interested in my views. I agree that it is most unsatisfactory when changes are made in the theatre staff during a 'list', but the nurse usually regrets this more than the surgeon, and we, of course, obey sister's orders. I think it is true that sister usually has our best interests at heart and cannot afford to have us going sick, that is why she avoids any individual overworking if possible. But sisters like surgeons have their likes and dislikes.

As regards the medical staff teaching the nurses, I couldn't agree more. We always enjoyed lectures from the doctors and one distinguished surgeon who used to visit us was more than popular. It is interesting, too, that he was one of those who didn't ignore us in the wards, even in our probationary days and always had time to give a reason for his demands.

Finally, whilst agreeing that the nurses' curriculum needs improving I have long wanted to make an addition to the doctor's education. Just as we benefit from the doctors' teaching I think that they would benefit from ours. Every time a newly qualified house surgeon appears for the first time in the theatre or wards, I am amazed at his (or her) ignorance of the ordinary routine of hospital practice. Could not something be done to remedy this?

Yours,
 'S.R.N.'