MEDICAL PRACTICE

Occasional Review

Grades of Hypothyroidism

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Summary

Seventy-nine patients with hypothyroidism and autoimmune thyroid disease were studied, and allotted to one of four categories on the basis of clinical and biochemical features. Firstly, patients with overt hypothyroidism had obvious clinical features of hypothyroidism and abnormal results from routine tests of thyroid function. Secondly, those with mild hypothyroidism, however, had minor and non-specific symptoms, but the routine measurements of circulating thyroid hormone concentration generally lay within the normal range, although they were significantly lower than those seen in subclinical hypothyroidism or in normal subjects. The serum concentration of thyroid-stimulating hormone (TSH) was raised in this group and their symptoms resolve with treatment. Thirdly, patients with subclinical hypothyroidism were asymptomatic, had a raised serum TSH concentration, but all other measurements of thyroid function are indistinguishable from those recorded in people with autoimmune thyroid disease without disturbance of thyroid function and in normal subjects. Lastly, subjects with circulating thyroid antibodies, normal indices of thyroid function, and a normal serum TSH concentration were indistinguishable biochemically from normal subjects.

Thus hypothyroidism is a graded phenomenon, the most valuable features for defining the individual grade being the clinical manifestations, the serum TSH concentration, and the presence of circulating antibodies to thyroid tissue.

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Introduction

The clinical syndrome of hypothyroidism was first described by Gull in 1874.[1] It was widely accepted during the early years of this century that hypothyroidism was an "all or none" phenomenon. Nevertheless, the advent of more precise diagnostic techniques, which enable different aspects of thyroid function to be measured, have shown that hypothyroidism is a graded phenomenon. Hypothyroidism may therefore be defined as those conditions which result from suboptimal circulating levels of one or both thyroid hormones. This definition though conceptually satisfactory poses problems in the practical definition of the various stages of thyroid failure. These problems arise in defining the suboptimal levels of circulating thyroid hormone for an individual. The laboratory techniques available for assessing thyroid function fall naturally into three groups, and the use of each group of techniques allows different aspects of impaired thyroid function to be defined.

The three groups of techniques are (1) tests of thyroid function, including direct and indirect measurements of circulating thyroid hormone concentrations and measurement of thyroid radioiodine uptake before and after administration of thyroid-stimulating hormone (TSH); (2) tests of peripheral tissue function, including measurement of the duration of the ankle tendon reflex, measurement of serum lipid concentrations, and inspection of the standard 12-lead electrocardiogram; and (3) tests of hypothalamic-pituitary function, including measurement of the basal serum TSH concentration and the rise in concentration after administration of thyrotrophin-releasing hormone (TRH). A classification of the various

grades of hypothyroidism has been proposed[2] on the basis of the clinical and laboratory features of hypothyroidism, and the present communication assesses the validity of this classification.

Patients

Seventy-nine patients were examined in detail and allotted to one of four groups on the basis of the clinical and laboratory features.

Group 1: Overt Hypothyroidism (21 patients).—These patients had the well known features of hypothyroidism—namely, lack of energy, cold intolerance, acroparaesthesiae, weight gain, constipation, and hoarseness of the voice. Confirmation of the diagnosis presented no problem. This state results from a major degree of thyroid failure.

Group 2: Mild Hypothyroidism (19 patients).—These patients, with minor but non-specific symptoms suggestive of hypothyroidism, were investigated and found to have evidence of mild thyroid failure. The only presenting symptom in 11 cases was fatigue. In the remainder complaints were of either dryness of the skin or constipation or hair loss as isolated symptoms. The possibility of a minor degree of thyroid failure was raised in the earlier patients in this group by the presence of a family history of thyroid or other organ-specific autoimmune disease or by the finding of either a small, firm goitre or vitiligo on routine clinical examination. Thyroid function was investigated routinely in later patients, who presented with either fatigue or constipation which could not be explained. Conventional tests of thyroid function often gave equivocal results, but the serum TSH concentration was found to be raised and the symptoms remitted after treatment with thyroid hormone.

Group 3: Subclinical Hypothyroidism (22 patients).—These patients were asymptomatic and were detected in one of several ways: (*a*) observation of a small goitre in relatives or friends visiting outpatients (6 patients); (*b*) the finding of a raised serum TSH concentration in "normal" controls (10 patients); (*c*) observation of a thyroid-related ophthalmopathy in asymptomatic subjects (5 patients); (*d*) observation of vitiligo (1 patient). Conventional tests of thyroid function showed nothing abnormal in this group but they were all found to have a raised serum TSH concentration. None of these patients had been subjected to destructive therapy to the thyroid by surgery or radioactive iodine, both of which are known to lead to raised TSH levels with and without symptoms.

Group 4: Autoimmune Thyroid Disease Without Disturbance of Thyroid (17 patients).—These subjects were derived from three sources: (*a*) observation of a goitre (6 patients); (*b*) detection of antibodies in the routine investigation of the relatives of patients with thyroid disease (5 patients); (*c*) observation of thyroid-related ophthalmopathy in asymptomatic subjects (6 patients). All these patients were asymptomatic, gave a normal response to conventional tests of thyroid function, and had a normal serum TSH concentration.

Methods

Circulating Thyroid Hormone Concentrations.—The protein-bound iodine (P.B.I.) (AutoAnalyzer) and triiodothyronine (T-3) Sephadex uptake (Thyopac-3 kit, Radiochemical Centre, Amersham)[3] were measured in all subjects and the free thyroxine index was computed from the results.[4] The serum T-3 concentration was estimated by radioimmunoassay of unextracted serum[5] from 61 subjects.

Radioiodine Studies.—TSH stimulation tests were carried out in 32 subjects in groups 2, 3, and 4. Thyroid ^{131}I uptake was measured 24 hours after a 5-μCi dose before and after two intramuscular injections each of 10 IU of bovine TSH. Results were assessed according to the criteria of Perloff *et al.*[6]

Tests of Peripheral Tissue Function.—Fasting serum cholesterol was measured in 62 subjects with the AutoAnalyzer using an isopropanol extraction.[7] Fasting serum triglyceride concentration was measured by the technique of Fletcher[8] in 69 subjects. The duration of the ankle tendon reflex was measured using a Cranlea Reflexometer in 37 subjects in groups 1, 2, and 3. A standard 12-lead electrocardiogram was obtained in 57 cases using an Elema-Schonander three-channel electrocardiograph.

Tests of Hypothalamic-Pituitary Function.—Serum TSH estimation was carried out in all subjects by radioimmunoassay.[9] The response to a 200-μg dose of intravenous TRH[10] was estimated in 48 subjects.

Thyroid Antibodies.—The serum of all subjects was examined for circulating thyroid antibodies. Thyroglobulin antibodies were estimated by the tanned red cell technique using cells supplied by Burroughs Wellcome. Takatsy plates and microtitre dilutions were used according to the method of Roitt and Doniach.[11] The serum was also examined for cytoplasmic antibodies using a standard immunofluorescence technique with FITC-conjugated rabbit antihuman gammaglobulin and by complement fixation using commercially available thyroid antigen (Burroughs Wellcome).

Results

Circulating Thyroid Hormone Concentrations.—Significant differences in P.B.I., T-3 Sephadex uptake, and free thyroxine indices were observed between groups 1, 2, and 3, although there was considerable overlap between the groups (table I, figs. 1-3). No difference was found between the

TABLE I—*Circulating Thyroid Hormone Concentrations* (*Mean* ± *S.D.*)

	P.B.I. (µg/100 ml)	T-3 Sephadex Uptake	Free Thyroxine Index	Serum T-3 (ng/ml)
Overt hypothyroidism ..	3·1 ± 1·5**	1·23 ± 0·10**	2·6 ± 1·4**	0·55 ± 0·38**
Mild hypothyroidism ..	4·6 ± 0·9*	1·15 ± 0·07**	4·0 ± 0·8**	1·08 ± 0·35†
Subclinical hypothyroidism	5·4 ± 1·3†	1·09 ± 0·07†	4·9 ± 1·2†	1·35 ± 0·59†
Autoimmune thyroid disease	5·6 ± 1·1†	1·08 ± 0·07†	5·3 ± 0·9†	1·29 ± 0·40†
Controls	5·4 ± 1·5	1·09 ± 0·07	4·9 ± 1·2	1·26 ± 0·23

Symbols refer to significance of relationship of a value with the one immediately below.
* $P < 0.05$.
** $P < 0.01$
† Not significant.

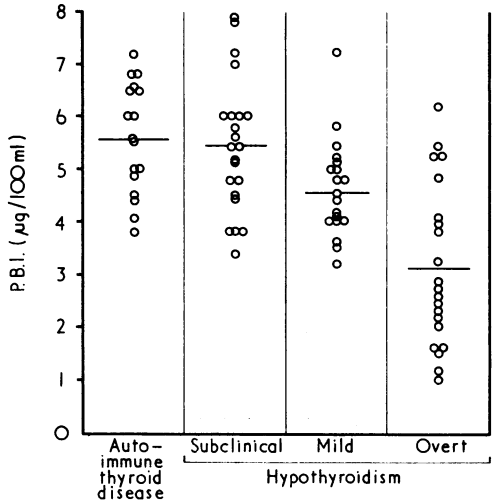

FIG. 1—Serum P.B.I. concentrations in hypothyroidism.

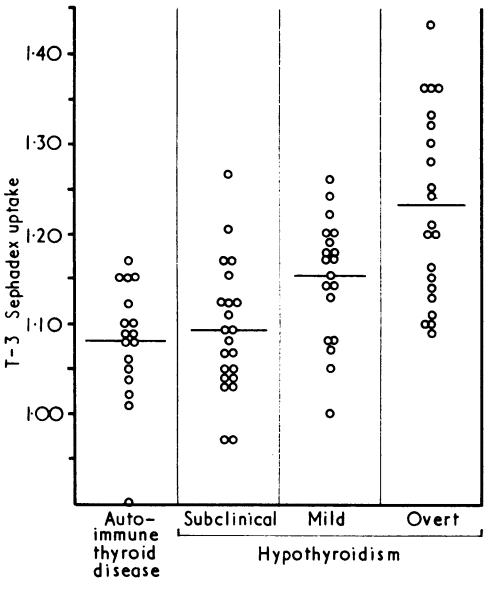

FIG. 2—T-3 Sephadex uptake in hypothyroidism.

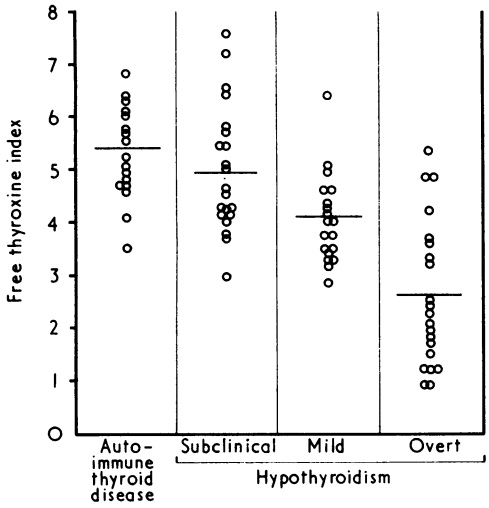

FIG. 3—Free thyroxine index in hypothyroidism.

P.B.I. and T-3 Sephadex uptake estimations in groups 3 and 4. Nor were the values for P.B.I. and T-3 Sephadex uptake observed in these two groups different from those of a control female population of a similar age group (mean P.B.I. 5·4 μg/100 ml, S.D. ± 1·5 μg/100 ml; mean T-3 Sephadex uptake 1·09, S.D. ± 0·07). It was not only evident that there was a considerable overlap between these groups but it was also observed that the P.B.I., T-3 Sephadex uptake, and free thyroxine index were consistently abnormal only in the group with overt hypothyroidism, although even in this group three subjects were shown to have normal values for all three of these estimations. These measurements fell within the conventionally accepted range of normality in at least half of the subjects in group 2. It was noted that although the P.B.I. was normal (greater than 4·0 μg/100 ml) in 16 subjects in this group the T-3 Sephadex uptake was normal (less than 1·15) in only 8 of these patients and thus appeared to be a better discriminant. The serum T-3 concentration was low in group 1, although there was some overlap with the lower end of the normal range in this group (table I, fig. 4). The serum T-3 concentration was significantly higher in group 2, only three of the subjects in this group having unequivocally low values, the remainder being in the lower part of the normal range. The level was higher in group 3 than in group 2, although this result did not achieve statistical significance. Two subjects in this group (both of whom had large goitres) had raised serum T-3 concentrations. The concentrations in groups 3 and 4 were identical (with the exception of the two patients with large goitres and raised values) and indistinguishable from that found in normal subjects (mean 1·26 ng/ml, S.D. ± 0·23 ng/ml, range 0·76-1·67 ng/ml).

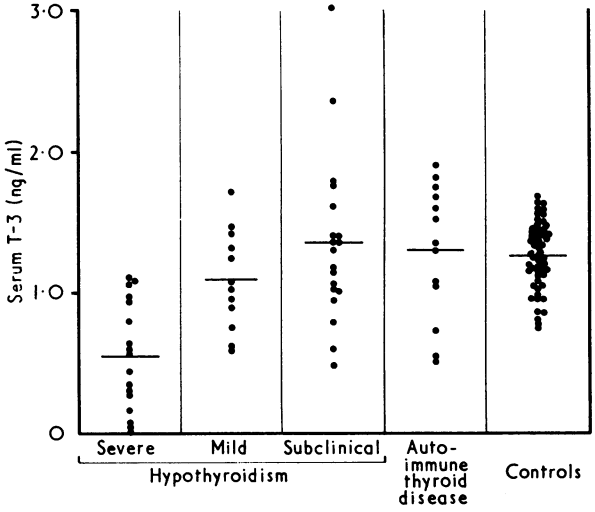

FIG. 4—Serum T-3 concentrations in hypothyroidism.

Radioidine Studies.—An impaired thyroid radioiodine response to TSH was found in four out of eight subjects in group 2 and three out of 13 in group 3) but not in any of the subjects in group 4 (table II).

TABLE II—*Other Tests of Thyroid Function*

	TSH Stimulation Test % Abnormal	E.C.G. % Consistent with Hypothyroidism	Ankle Tendon Reflex (msec) Mean ± S.D.
Overt hypothyroidism	—	60	421 ± 123
Mild hypothyroidism	50	39	303 ± 61
Subclinical hypothyroidism	23	28	308 ± 35
Autoimmune thyroid disease	0	17	—

Tests of Peripheral Tissue Function.—The serum cholesterol concentration was significantly raised in subjects in group 1 but there was no difference in concentration between the other three groups (table III, fig. 5). The serum cholesterol in group 4 was not significantly higher than in a group of controls matched for age and sex. Similarly there was no significant difference in serum triglyceride levels between the four groups, and although the values observed in the subjects with hypothyroidism were slightly higher than those observed in controls these differences did not achieve statistical signifi-

TABLE III—*Serum Lipid Concentrations (Mean ± S.D.)*

	Serum Cholesterol (mg/100 ml)	Serum Triglyceride (mg/100 ml)
Overt hypothyroidism	332 ± 87*	158 ± 75†
Mild hypothyroidism	267 ± 46†	162 ± 30†
Subclinical hypothyroidism	275 ± 85†	159 ± 85†
Autoimmune thyroid disease	258 ± 57†	131 ± 73†
Controls	231 ± 48	103 ± 52

*$P < 0.05$.
†N.S.

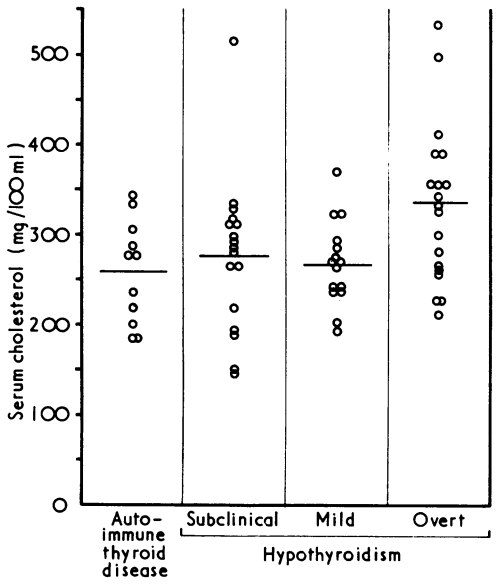

FIG. 5—Serum cholesterol concentrations in hypothyroidism.

cance. The duration of the ankle tendon reflex was found to become longer as thyroid failure became more severe but very considerable overlap was found between the groups. Electrocardiographic abnormalities consistent with hypothyroidism were found with increasing frequency as the degree of thyroid failure increased, but even in subjects in group 1 electrocardiographic abnormalities were not always observed (table II).

Tests of Hypothalamic-Pituitary Function.—There was a clear and significant difference in serum TSH concentration between all four groups (table IV, fig. 6), although there was some overlap between individual members of groups 1, 2, and 3. The values recorded in group 4 were by definition normal. The 20-minute serum TSH level after TRH also showed a graded response through the groups. The response was normal in all 11 members of group 4 tested.

Thyroid Antibodies. Circulating antibodies to thyroid tissue were found in all subjects in group 4 and in 85% or more of the subjects in each of the other three groups (table V). Cytoplasmic antibodies were found more frequently than thyroglobulin antibodies in all groups. The frequency of thyroglobulin and cytoplasmic antibodies was significantly higher than in a control population drawn from the local community matched for age and sex.

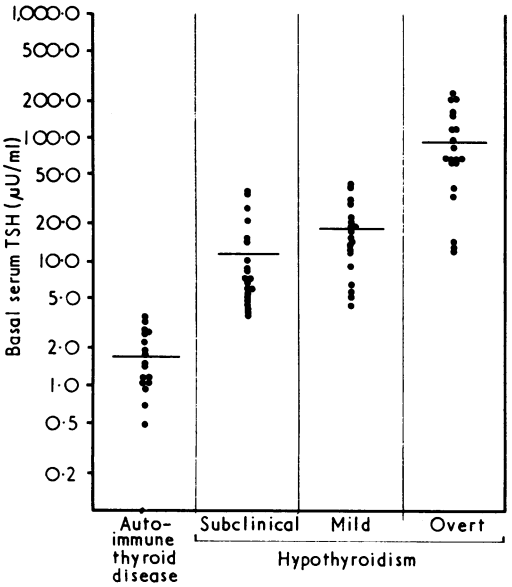

FIG. 6—Serum T S H concentrations in hypothyroidism.

TABLE IV—*Serum TSH Levels Before and 20 Minutes After 200 μg of TRH Intravenously (Mean ± S.D.)*

	Serum TSH (μU/ml)	Serum TSH (μU/ml) 20 min After TRH
Overt hypothyroidism	91·0 ± 63·1*	169·0 ± 98·1**
Mild hypothyroidism	17·9 ± 11·1*	63·3 ± 23·9†
Subclinical hypothyroidism	11·2 ± 9·7**	40·8 ± 18·3**
Autoimmune thyroid disease	1·77 ± 0·86	13·2 ± 4·7

* $P < 0.05$.
** $P < 0.01$.
† Not Significant.

TABLE V—*Thyroid Antibodies*

	Auto-immune Thyroid Disease	Sub-clinical Hypo thyroid-ism	Mild Hypo-thyroid-ism	Overt Hypo-thyroid-ism	All	Controls (150)
Tanned red cells	71%	47%	74%	43%	58%	1·3%
Cytoplasmic	100%	65%	75%	83%	80%	1·3%
Complement fixation test	92%	65%	58%	80%	76%	2·0%
Any	100%	85%	95%	86%	89%	3·3%

Discussion

This study showed very clearly that hypothyroidism is a graded phenomenon. It became evident that the most valuable observations for defining the grades of thyroid failure and identifying subjects with thyroid failure were the clinical features, the serum TSH concentration, and the presence of circulating antibodies to thyroid tissue. It was also possible to review critically many of the other tests used for the diagnosis of hypothyroidism.

The clinical features of overt hypothyroidism are well known and confirmation of the clinical diagnosis rarely presents a problem since the results of conventional tests of thyroid function are rarely normal in this group.

The recognition of minor degrees of thyroid failure presents many difficulties, since subjects with mild hypothyroidism have minor and generally non-specific symptoms. Tiredness was the only symptom present in more than half of the subjects in this group. The remainder presented with either dryness of the skin or constipation or hair loss. These symptoms are, of course, common in middle-aged women and are usually not associated with thyroid disease. Possibly these symptoms in the group 2 subjects were unrelated to thyroid failure and that the response to therapy was merely a placebo effect. However this group, who were distinguished from those with subclinical hypothyroidism (group 3) only by the presence of a single symptom, had a significantly lower P.B.I. and free thyroxine index and a significantly higher serum TSH concentration. Direct evidence that subjects in group 2 represented a different, albeit overlapping, population from those in group 3 can be provided only by a double-blind crossover trial using L-thyroxine and a placebo, which is at present under way. But, the diagnosis of mild hypothyroidism on the basis of primary thyroid disease can be confidently excluded by the finding of a normal serum TSH concentration.

Subclinical hypothyroidism has been defined as an asymptomatic state in which a reduction of thyroid activity has been compensated by an increased TSH output to maintain a euthyroid state.[2] The data presented here support this definition. These subjects all showed a serum TSH concentration which was significantly raised compared with the sub-

jects in group 4 and also significantly raised when compared with the normal population. The estimation of circulating thyroid hormone concentrations (P.B.I., T-3 Sephadex uptake, free thyroxine index, and serum T-3) gave values which were indistinguishable from those observed in subjects in group 4 and in normal subjects. However, two subjects (both with large goitres) had greatly raised T-3 concentrations, and possibly this was a part of the adaptive mechanism to developing hypothyroidism in some subjects. A trial of thyroxine therapy was carried out in six of these subfjects (group 3) but none of them reported any symptomatic change. Subclinical hypothyroidism has been defined in rather different terms by others. Bastenie *et al.*,[12-14] defined this stage in terms of circulating thyroid antibodies, whereas Fowler *et al.*[15-16] based their definition largely on raised serum cholesterol concentrations. Although most subjects in this group had circulating thyroid antibodies and some had a raised serum cholesterol concentration or both of these phenomena, neither of these findings was diagnostic of subclinical hypothyroidism. The results of the TSH stimulation test are almost invariably normal in these patients and the diagnosis can be made only by finding a high serum TSH level in an asymptomatic subject. The abnormalities of thyroid function observed in this group can be restored to normal by the administration of thyroid hormone, but the need for hormone treatment in these asymptomatic subjects remains to be proved. At present neither the frequency of their progression to symptomatic hypothyroidism nor the possible time course of such progression is known.

Circulating antibodies to various thyroid components are frequently found in symptomatic hypothyroidism. They may also be detected in a group of subjects who have no evidence of thyroid failure and who are in all respects indistinguishable from normal. The natural history of these subjects is also unknown.

Evidence has been presented that in a hospital population (1) people with hypothyroidism have an increased incidence of ischaemic heart disease;[13] (2) subjects with positive thyroid antibodies and lymphocytic infiltration of the thyroid, but without overt thyroid disease, have an increased incidence of ischaemic heart disease and a raised serum cholesterol concentration.[12 13 15 16] There is a recognized association between hyperlipoproteinaemia (Fredrickson types II and IV) and hypothyroidism.[12 13 17 18] The nature and frequency of this association and its clinical significance were not apparent from the present study. The serum choloesterol and triglyceride concentrations were raised in all groups when compared with a control group drawn from the general population matched for age and sex. The hypothesis that subclinical hypothyroidism or autoimmune thyroid disease without disturbance of thyroid function are significant risk factors for coronary artery disease remains to be tested adequately in the general population.

The present study made it possible to review a number of the investigations which are currently used in the diagnosis of hypothyroidism. The commonly used indirect measurements of circulating thyroid hormone concentration (the P.B.I. and the T-3 Sephadex uptake) showed clearly that hypothyroidism is a graded phenomenon, but there was considerable overlap between the various groups. These indices of thyroid function were almost invariably abnormal in subjects with overt hypothyroidism. In patients with mild hypothyroidism values for these tests frequently fell within the conventionally accepted normal range but were still significantly different from those observed in normal subjects. Normal values for P.B.I. and T-3 Sephadex uptake cannot therefore be used to exclude a diagnosis of thyroid failure in an individual patient. The computation of the free thyroxine index does not appear to increase the diagnostic value of these tests in hypothyroidism.

The TSH stimulation test was performed on a significant number of subjects in groups 2, 3, and 4. The response to TSH stimulation was normal in half of the subjects with mild hypothyroidism in whom this investigation was carried out. Similarly a normal response was found in 77% of subjects with subclinical hypothyroidism. The standard TSH stimulation test cannot therefore be relied on to identify subjects with mild hypothyroidism, nor can it be used to identify subjects with subclinical hypothyroidism. For many years the TSH stimulation test has been regarded as the absolute criterion for establishing the presence of impaired thyroid reserve.[19] However, critical evaluation by comparison with more sensitive tests of hypothalamic-pituitary function clearly showed that the standard TSH stimulation test is not an entirely satisfactory test of thyroid reserve. Probably thyroid ^{131}I uptake can be significantly increased by the very large and unphysiological dose of TSH used even in the face of mild thyroid failure. Clearly a more sensitive test of thyroid reserve is required.

Estimation of the serum TSH concentration proved to be the most sensitive index of thyroid failure. Now that the range of serum TSH concentrations in normal subjects has been defined[10] it may be assumed that any increase in serum TSH concentration reflects a downward deviation from the optimum level of circulating thyroid hormones and to some extent gives an indication of the magnitude of this deviation. These data show a general inverse correlation between circulating thyroid hormone concentrations and serum TSH and thus support earlier findings.[20] The hypothalamus and pituitary may therefore be regarded as the most sensitive peripheral tissues in terms of a reduced circulating thyroid hormone concentration. Although a prolonged and exaggerated rise of serum TSH concentration is observed after the administration of TRH in hypothyroid subjects this investigation rarely does more than confirm the already raised basal serum TSH. A TRH test may occasionally be of value in detecting minimal thyroid failure in subjects who have a basal serum TSH concentration close to the upper limits of normal.

Tests of peripheral tissue function were carried out in members of all groups. Abnormalities were found with increasing frequency as thyroid failure became more severe, but the scatter in each group was so great that the diagnostic value of these investigations was found to be extremely limited. Abnormalities were found in most subjects with overt hypothyroidism in whom diagnostic difficulty was not encountered, and the sensitivity of these tests appeared to be no greater than those of indirect estimations of thyroid hormone concentration. The possible significance of raised lipid concentrations has already been discussed.

The sera of all patients were examined for circulating thyroid antibodies, which were found in over 85% of subjects with hypothyroidism. It was noted that there was a much closer correlation between the presence of cytoplasmic antibodies and thyroid failure than between thyroglobulin antibodies and thyroid failure. These findings are consistent with current views on the relation between thyroid autoimmunity and thyroid failure.[21] The prevalence of thyroglobulin antibodies in the subjects with thyroid failure was lower than that reported by other workers.[22 23] The titre of thyroglobulin antibodies was also considerably lower than has been generally experienced, and probably this was the result of a reduced sensitivity of the antigenic material used in the assay as well as of more precise methods of serum dilution.

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Hospital Topics

Cubital Tunnel External Compression Syndrome

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Summary

External compression of the cubital tunnel comprises the acute and subacute forms of ulnar nerve compression at the elbow. Subacute compression is often seen in hospital practice and sometimes results in partial crippling of the hand. Prognosis for complete recovery is poor. Avoidance of a position of the elbow which predisposes to external compression of the ulnar nerve within the cubital tunnel is advised when a patient is on the operating table, in bed or in an armchair. Prolonged severe elbow flexion in these circumstances should also be avoided. The patient suffering from the syndrome should be instructed to avoid further pressure so that worsening of the palsy is minimized. A compressed nerve is likely to be more sensitive than a normal nerve to ischaemia produced by subsequent pressure. Surgical treatment is sometimes indicated at least to halt progression of the palsy.

Introduction

Ulnar nerve palsy from compression at the elbow may conveniently be classified into three types (fig. 1): (1) *acute*, resulting from a single episode of force; (2) *subacute*, resulting from external pressure applied over a limited period; and (3) *chronic*. The chronic type includes pressure from lesions within the cubital tunnel (osteoarthritis, rheumatoid disease, ganglion, and other soft-tissue tumours) as well as diminution of the capacity of the tunnel from lateral shift of the ulnar and approximation of the roof to the floor. A common cause of the latter is injury to the capitular epiphysis leading to a deficient lateral trochlear lip[1] and sometimes to resultant "tardy ulnar palsy" in adult life.

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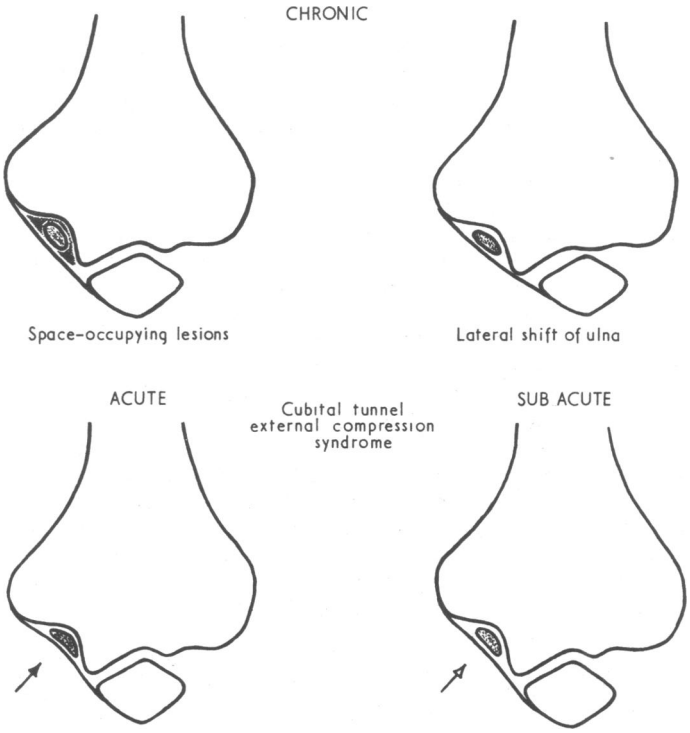

FIG. 1—Diagram illustrating causative factors in *chronic*, *acute*, and *subacute* ulnar palsy, the acute and subacute constituting the *cubital tunnel external compression syndrome*. (Arrows indicate direction of pressure.)

Cases of the acute and subacute types due to external compression of the cubital tunnel have been only sporadically reported[2-6] but it should be appreciated that the subacute form in particular is common in hospital practice. The degree of palsy can usefully be graded by following McGowan's[7] description, which is mainly concerned with the condition of the interossei. Grade 1 consists of paraesthesiae and minor hypoaesthesia. Grade 2 consists of weakness and wasting of the interossei and medial two lumbricals with incomplete hypoaesthesia. Grade 3 consists of paralysis of the interossei and medical two lumbricals together with, in most cases,