MANAGEMENT OF THYROID SWELLINGS

AN ESSAY

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of the Master Degree of
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BY

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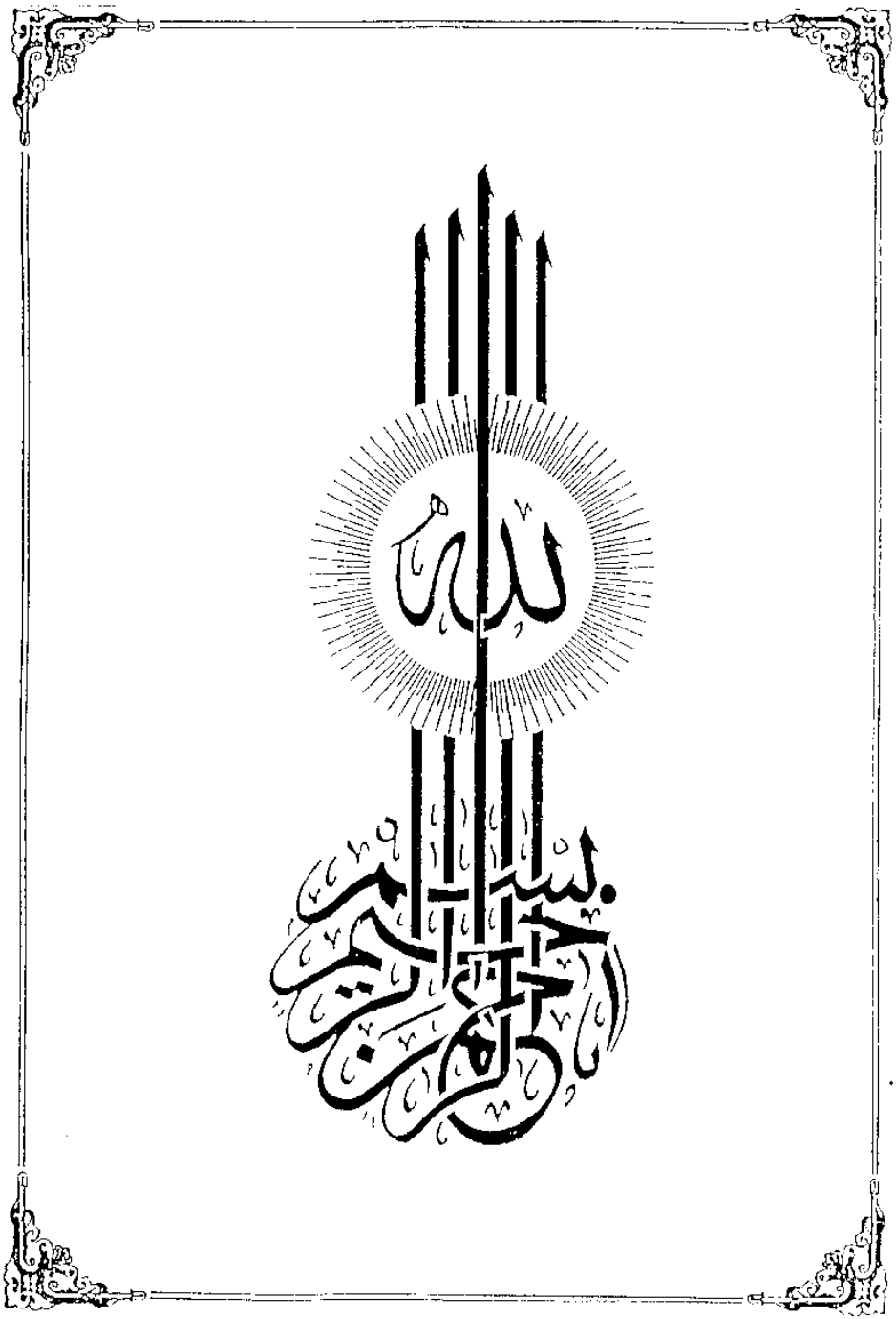
TO MY PARENTS

TO MY WIFE



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INTRODUCTION

Patients with generalised or localised enlargement of the thyroid gland are commonly referred to surgical clinics .

Thyroid enlargement is seldom responsible for symptoms and the reason for referral is usually the presence of a swelling. When local symptoms are present they influence both diagnosis and management, but apparent symptoms especially mild dysphagia, may be spurious and mistakenly attributed to the swelling. In most patients with generalised thyroid enlargement the diagnosis of the underlying pathological process may be made on clinical examination whereas this is seldom possible in isolated swellings.

In isolated swellings diagnostic effort is focussed on the possibility of neoplasia which though infrequent has none the less traditionally dictated management. Fear of neoplasia is such less often a consideration in the evaluation of generalised thyroid enlargement. In generalised enlargement the reliability of diagnosis and the infrequency of neoplasia mean that operation is seldom indicated except for pressure symptoms or cosmesis whereas in isolated swelligns though few are malignant,

operation has commonly been the ultimate diagnostic process. Many of these operations for what proves to be benign degenerative process are at least in theory unnecessary and if a reliable diagnosis could be made, operation would be avoided in a substantial number of patients. Accurate diagnosis is therefore an important aim to achieve a proper management.

The present literature focusses on the recent methods and trends in establishing such accurate diagnosis and appropriate lines of treatment for each individual case. In an exercise of this nature the author made use of materials largely provided in the form of "Reference work", and I have duely registered my indebtedness and gratitude to the respective authors as evident in the reference column. The original materials are authentic and any errors or shortcomings in their compilation remain the responsibility of the present author.

PHYSIOLOGY OF THE THYROID GLAND

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I. Synthesis and Secretion of Thyroid Hormones:

1) Requirement of iodine for formation of thyroid hormones:

To form normal quantities of thyroid hormones, approximately 1 mg of ingested iodine is required per week (Guyton, 1981). The normal daily requirement is about 100-125 ug (Wade, 1984). Iodides ingested orally are absorbed rapidly from the intestine, it is extracted from the plasma both by the thyroid and the kidneys. Within the first 3 days about 2/3 of the ingested iodides are normally lost in the urine and almost all remaining 1/3 is selectively removed from the blood by the thyroid gland. (Kaplan, 1984).

2) Formation and secretion of thyroglobulin by the thyroid cells:

The thyroid cells are typical protein secreting cells. The endoplasmic reticulum and Golgi complex synthesize and secrete into the follicles a large Glycoprotein molecule called thyroglobulin with a molecular weight of 660,000. Each molecule of thyroglobulin contains 140 tyrosine amino acids. These amino acids combine with iodine to form the thyroid hormones. The thyroid

cells not only provide thyroglobulin, but also provide the iodine, the enzymes and other substances necessary for thyroid hormone synthesis. Thyroid stimulating hormone (T.S.H.) increases the synthesis and secretion of thyroglobulin by the cells of the gland.

3. Iodide Trapping:

The first step in the formation of thyroid hormone formation is active transport of iodide from the plasma into the thyroidal glandular cells. The activity of the concentration mechanism is stimulated by T.S.H. and depletion of iodine stores within the gland. It is blocked by perchlorate and thiocyanate (ionic inhibitors). In presence of high iodide intake, iodides will enter the cells by diffusion and so these transport inhibitors will lose their effect as antithyroid drugs.

4. Oxidation of Trapped Iodides into Iodine:

Oxidation of the trapped inorganic iodides (I^-) to organic iodine (I_2) is promoted by the action of peroxidase enzyme. This step is stimulated by T.S.H. and blocked by thiourea derivatives e.g. propylthiouracil and carbimazole,

5. Iodinaiton of Tyrosine:

Tyrosine radicals of thyroglobulin molecules are iodinated to form monoiodotyrosine (Mit) and di-iodotyrosine(Dit).

6. Coupling of Iodotyrosines:

Thyroxine (T_4) is formed by coupling of two (Dit) radicals, while tri-iodothyronin (T_3) is formed by coupling of one (Mit) and one (Dit) radical, both (T_4) and (T_3) remain as a part of the thyroglobulin molecule.

Iodination of tyrozine and coupling of iodotyrosines are stimulated by T.S.H., and blocked by; (a) thiourea derivatives; (b) some aminoheterocyclic compounds e.g. PABA, sulphonamides, sulphonylurea, PAS; and (c) high doses of iodides may interfere with iodination of tyrosine resulting in goitre formaiton. (Astwood et al., 1975).

7. Storage of Thyroid Hormones:

The normonally active iodotyrosines (T_4) and (T_3) and also (Mit) and (Dit) are held by peptide linkage with thyroglobulin which form the major component of the intrafollicular colloid. The storage of the hormones is of such a magnitude that the normal gland contains enough preformed thyroid hormones to maintain the euthyroid state for 2 months without synthesis, also administration of antithyroid agents for as long as 2-3 weeks results in only minimal decrease in T_4 and T_3. (Kaplan, 1984).

8. Release of (T_4) and (T_3) from thyroglobulin:

Release of thyroid hormones into the circulation involves hydrolysis of thyroglobulin by proteolytic enzymes. This is stimulated by T.S.H. and blocked by excess iodides (Astwood et al., 1975).

9. Intrathyroidal recycling of iodine:

One half or more of iodinated tyrosines never become thyroid hormones but instead remian (Mit) and (Dit). During release of (T_4) and (T_3), (Mit) and (Dit) are also freed from thyroglobulin, but they are not secreted into blood, instead their iodine is cleaved from them by dehalogenase deiodinase enzyme, the released iodine will be reutilized for synthesis of thyroid hormones. (Guyton, 1981).

10. Circulation of thyroid hormones:

On entering the blood (T_4) and (T_3), to a varying degree, are bound to plasma proteins (thyroxine binding globulin (T.B.G.), thyroxine binding prealbumin (T.B.P.A.) and albumin). (T_4) binds more strongly to plasma proteins than (T_3). A small amount of the thyroid hormones remains free in the serum in equilibrium with the protein-bound hormone and is biologicallly active; unbound T_3 is normally about 10 times greater than T_4. (Harrison, 1981). In the plasma the ratio of T_4:T_3 is 10:1 to 20:1 (Kaplan, 1984).

Peripheral conversion of T_4 to T_3 takes place, suggesting that T_3 is a specific stimulus for certain

thyroid hormone effects, and it is the more important physiological hormone. Because T_3 binds less firmly to plasma protein, it enters the peripheral tissues more rapidly. T_3 is quick acting (within few hours) while T_4 acts more slowly (within few days) (Wade, 1984). T_3 is about 4 times as potent in stimulating metabolism and causing other intracellular effects as T_4, also the duration of action of T_4 is about 4 times as long as duration of action of T_3 (Guyton, 1981).

II. Regulation of thyroid Hormones Secretion:

Synthesis and liberation of thyroid hormones from the thyroid gland is stimulated by T.S.H. from the anterior pituitary. T.S.H. stimulates all the processes leading to synthesis and secretion of thyroid hormones. The most important early effect after administration of T.S.H. is proteolysis of thyroglobulin which causes release of T_3 and T_4 within 30 minutes, the other effects require hours or even days and weeks to develop. The effect of T.S.H. is mediated by cyclic AMP. (Guyton 1981). The secretioin of T.S.H. is influenced by the hypothalamus which produces thyrotropin releasing hormone (T.R.H.) which is transmitted in the hypothalamic-hypophyseal portal blood to the anterior

PATHOLOGY OF THYROID SWELLINGS

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Enlargement of thyroid gland is termed goitre. The term goitre is derived from the Latin "gutter" meaning throat. (Kaplan, 1984)

Thyroid swellings, Goiters, can be classified into:

I. **Non toxic goitres (simple goitres), which include:-**

a. diffuse hyperplastic goitre.

b. diffuse colloid goitre.

c. multinodular goitre.

d. solitary nodule.

II. **Toxic goitres:**

a. diffuse toxic goitre.

b. nodular toxic goitre.

c. toxic nodule.

III. **Thyroiditis:**

a. Hashimoto's thyroiditis.

b. Subacute thyroiditis.

c. Riedel's throiditis.

d. Other rare forms of thyroiditis are:-

acute suppurative thyroiditis, tuberculosis, syphilis and actinomycosis.

IV. **Neoplastic goitres:**

1. Benign thyroid neoplasms (Adenomas)

2. Malignant thyroid neoplasms which may be primary or secondary.