

THE TOXIC GOITERS OF THE ADOLESCENT AGE*

J. EARL ELSE, M.S., M.D., F.A.C.S.

Assistant Professor and Chairman
Executive Committee, Department of Surgery,
University of Oregon Medical School
AND

BARTON PEDEN, B.S., M.D.

PORTLAND, ORE.

Goiter is more prevalent during the adolescent age than at any other age during life. In a partial survey made in the public schools in Portland it would seem that approximately 60 per cent of the girls and 30 per cent of the boys have a goiter during this period. In the Goiter Clinic of the University of Oregon Medical School, we use the following classifications of adolescent goiter:

- I. Simple Goiter
 - A. Colloid
 - B. Hyperplastic
 - 1. Diffuse
 - 2. Localized
- II. Toxic Goiter
 - A. Toxic hyperplastic adolescent
 - 1. Diffuse
 - 2. Localized
 - B. Toxic hyperplastic (exophthalmic)
 - C. Toxic adenomas
- III. Tumors
 - A. Adenoma
 - B. Sarcoma

We have seen none of the other varieties of goiter in the adolescent age.

An understanding of the various types of adolescent goiters necessitates a brief review of the anatomy and physiology.

Anatomy. The thyroid gland is made up of acini, supported by a thin interstitial structure containing connective tissue, blood vessels, lymph channels, interstitial cells of Wolfler, and nerves. The acini differ from the acini of other glands in two essentials: first, they have no ducts through which to discharge their secretion; and second, they are normally distended with a homogenous material, colloid.

Physiology. The cells lining the acini secrete two substances: first, the active principle, termed

by Kendall thyroxin, a substance probably needed by every cell in the body and the secretion of which may be measured in terms of heat output or oxygen consumption; second, the colloid, an inert, homogenous substance, the purpose of which is to hold the thyroxin in suspension until needed.

Due to the rapid changes incident to the adolescent period, there is an increase in the demand thrown upon the thyroid gland as well as upon the other important organs of the body. Owing to an iodine deficiency in the food and water intake in many sections of the world, and probably also to a variability, first, in the absorption of iodine from the intestinal tract; and, second, in its utilization by the thyroid, the thyroid, which previous to the adolescent period was able to meet the demand placed upon it, now becomes deficient.

This deficiency is ordinarily manifest in one of two ways: (1) by an oversecretion of colloid in the attempt to secrete a sufficient amount of thyroxin, which results in a distension of the acini, increasing the size of the gland, and is known as colloid goiter; and (2) by an increase in the size and number of the epithelial cells in order that there may be a greater secretion of thyroxin. This condition we call the hyperplastic goiter of the adolescent age. It does not differ greatly in the gross or microscopic appearance from the hyperplastic goiter of the adult, except that frequently there are localized areas of hyperplasia, simulating an adenoma but differing from it in that it has no capsule and microscopically it resembles the hyperplastic type of goiter and not the adenomatous type, in which there is an increase in the number of acini. The former condition we designate as diffuse hyperplastic adolescent goiter and the latter localized hyperplastic adolescent goiter.

While in the majority of the cases of adolescent hyperplastic goiter there is no evidence of hyperthyroidism, in many there is such evidence. Pathologically these cases do not differ from the non-toxic hyperplastic adolescent goiter, but they must be grouped separately because of the clinical manifestations.

Colloid goiter is the most frequent type of goiter occurring in the adolescent age. Other than the

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presence of goiter there are no symptoms. It is easily preventable by the administration of iodine and easily curable by the judicious use of desiccated thyroid. It has been our experience that iodine is of great prophylactic value, but of almost no curative value. This statement is based upon a series of cases studied over a period of from six months to two years, in which the patients were seen at intervals of two weeks, the neck measured, the pulse counted, and the general physical condition observed.

The remainder of this paper is based upon a study made by Dr. Peden for his senior surgical thesis. One hundred seven consecutive cases of adolescent goiter were studied. Of this group fifteen were classified as toxic hyperplastic adolescent goiters; one as toxic adenoma and two as toxic hyperplastic goiter (exophthalmic).

As the material for this study comes largely through patients sent to the clinic by nurses or brought by anxious parents, this group of cases must not be considered as a representative group of patients with adolescent goiter. Toxic goiter does not exist in such percentage as is found in this group. These are the patients who have come for treatment, many of them being sent by the school nurses, because they have symptoms of hyperthyroidism, while the patients without such symptoms are, for the most part, going without treatment.

The simple diffuse hyperplastic adolescent goiter shows no symptoms or signs other than the enlargement of the thyroid gland. The gland is moderately enlarged, is fairly firm in consistency, being firmer than the colloid goiter except when the latter develops rapidly. The upper poles are usually fairly sharp.

The simple localized hyperplastic adolescent goiter differs from the diffuse hyperplastic type in that, while there is usually some increase in firmness of the gland, there are definite nodules. These nodules simulate adenomas, but differ from them in not being as definite, and disappearing under proper treatment. That this condition exists as a definite pathologic entity we are certain because of an error in diagnosis a few years ago, resulting in the re-

moval of such a nodule. Since that time we have studied the patients of this age with nodular necks carefully.

While we have seen a few adenomas, the majority of them have been of the localized hyperplastic type of goiter, as proven by the complete disappearance with desiccated thyroid. Adenomas will decrease in size under desiccated thyroid, if there is a considerable amount of colloid within the adenoma. The tumor does not, however, completely disappear. In fact, in many cases, while there is a decrease in the size of the adenoma, the tumor is even more perceptible because of a corresponding decrease in the size of the thyroid gland, due to a reduction of its colloid content under desiccated thyroid.

The toxic hyperplastic goiter of both the diffuse and localized varieties does not differ from the simple hyperplastic adolescent goiters pathologically. Clinically, however, the former group presents definite manifestations, while the latter group has no symptoms derived from hyperthyroid function. The toxic symptoms of the former are transitory. At times the patients are perfectly normal except for the thyroid enlargement. At other times they complain of tachycardia, palpitation, nervousness, tiring easily, and often there is some difficulty in concentration upon their school work. During these times the appetite may be variable. The patients sometimes lose in weight. The pulse is rapid, tremor present, and the blood pressure usually below the average.

The basal metabolic rate is increased during the period of hyperthyroidism. In the fifteen patients belonging to this group the highest rate was plus 54.7. There was one rate of plus 44.8, another of plus 43, another of plus 40, two of plus 34, one of plus 30, four between plus 20-30, and the rest plus 20 or below. Usually the rate remains up for a comparatively short time, if the patient's activity can be controlled. The giving of desiccated thyroid while the rate is up increases the rate, but after the basal metabolic rate has returned to normal the giving of desiccated thyroid carefully controlled usually does not increase the rate, nor cause a return of symptoms, but results in the diminishing of the size of the gland.

There is no apparent difference between the diffuse hyperplasia and the localized hyperplasia, clinically or in the relationship to treatment. The following case history serves as an illustration of this type of toxic goiter.

Patient, female, age 14. Chief complaint: Goiter, nervousness, sweating, palpitation, nausea, loss of appetite, sense of pressure in the neck.

Previous illness: Measles, whooping cough, scarlet fever, influenza.

Family history: Negative.

Present illness: First noticed goiter about four months before seen.

Physical examination: Fairly well nourished girl, pulse 112 and regular. Eyes react to light and accommodation, with no evidence of exophthalmus. Tonsils have been removed. Neck: Cervical glands palpable both sides. Thyroid: Diffuse enlargement of both lobes and the isthmus, with a nodule about 2.5 cm. in diameter in the right lobe. Lungs: Negative. Heart: Negative. Abdomen: Negative. No tremor. Skin: Moist. The basal metabolic rate at the time of the examination was plus 34.

No treatment was given at this time. The patient was advised to reduce activities to the minimum and permitted to continue attending school. Basal metabolic rate two months later plus 12. Four months later plus 8; one week after former, at time of menstruation, plus 15; nine and a half months after first seen plus 5. Eleven months after first observation, patient returned to clinic for observation, at which time the thyroid gland was found to be normal.

Diagnosis: Adolescent hyperplastic goiter with localized nodule, transitorily toxic when first seen.

Results: Cured, providing patient continues to take preventive dose of iodine.

Adenoma. In this series of cases, only one toxic adenoma was found. There have, however, been more adenomas seen in the clinic during the adolescent age, and have for the most part been nontoxic. The one in this series serves as an illustration of the toxic adenoma.

Patient: Female, age 14. Chief complaint: Goiter, nervousness, tachycardia, palpitation.

Family history: Mother was operated on in this clinic for toxic adenoma.

Present illness: The goiter was first noticed at the age of 11. There had been a history of symptoms suggestive of hyperthyroidism since the age of 10. At the present time she is nervous, sweats easily, faints frequently, tires easily. Appetite is good, is nauseated and vomits occasionally; has tachycardia, and palpitation frequently.

Physical examination: Somewhat slender, fair height, undernourished. The thyroid diffusely enlarged, with a definite palpable nodule in the lower right lobe. Tremor present. No abnormal eye signs. Basal metabolic rate January 5, 1923, plus 37; February 27, 1923, plus 62; March 2, 1923, plus 10; May 26, 1923, plus 42.

Diagnosis: Toxic adenoma.

Treatment: Patient was operated upon August 1, 1923, when a definite encapsulated nodule was found and removed, following which she made an uneventful recovery, and since which time has been practically normal.

Exophthalmic goiter: In two patients a diagnosis of exophthalmic goiter was made. While exophthalmic goiter in the adolescent age is uncommon, we have seen it in private practice as early as eight years of age.

In this group there were no tumors other than the adenoma. In private practice, however, a sarcoma has been seen during the adolescent period.

Treatment: The treatment of toxic goiter of the adolescent age like the nontoxic is primarily prophylactic. In none of the patients seen, presenting the evidence of toxic goiter, was there a history of taking iodine in prophylactic doses before the development of goiter. Some of them had taken iodine after the development of goiter, and the symptoms we believe are in some directly traceable to the effect of the iodine. We believe that, had the patients been given iodine from early childhood, the probabilities are there would have been no goiter to have become toxic.

Treatment of nontoxic goiter. Nontoxic goiters are treated in our clinic with desiccated thyroid. We begin with one grain three times daily. The patients are required to report at intervals of two weeks for observation. Patients not yielding to one grain three times a day and not showing any evidence of hyperthyroidism are given more. The majority of the patients are cured within three months. When the gland returns to normal, the patients are put on one grain of desiccated thyroid daily for one month, and then given either a grain of sodium iodide once a week or an iodostarine tablet once a week, or advised to use the iodized salt, and instructed to report back to the clinic every six months.

While it is too early to draw final conclusions, to the present we have seen very few recurrences in the cases thus treated and pronounced cured. There are a few cases diagnosed as simple hyperplastic adolescent goiters that have become toxic when given desiccated thyroid, but they have all

cleared up when it was discontinued. The toxic hyperplastic adolescent goiters are treated according to the intensity of the symptoms. The more severe cases are put at rest in bed. The milder cases have their activities limited and for the most part permitted to attend school. In this way the majority of them become nontoxic. They are then put on small doses of desiccated thyroid, which we find does not cause a return of symptoms if carefully controlled. The thyroid gland decreases in size, and upon its returning to normal the patients are put on iodine as a preventive measure.

The toxic hyperplastic exophthalmic goiter should be operated upon in the adolescent the same as in the adult. The same care should be used in preparing the patient and the same type of operation should be followed. With proper preoperative care

and with preliminary ligations where indicated, these patients respond well to operative procedure.

While the majority of the adenomas seen in the adolescent age are not toxic, we believe that all adenomas become toxic in the course of time, and therefore advise their removal.

Conclusions. Toxic goiter exists in the adolescent age in three forms: (1) Toxic hyperplastic adolescent goiter, which exists both as the diffuse and localized varieties, (2) adenoma, (3) toxic hyperplastic exophthalmic goiter. The former is the most common and if properly treated does not require operation. If improperly treated, permanent hyperplastic goiter may develop.

Adenoma and toxic hyperplastic exophthalmic goiters are not common but do occur and require surgical procedure.