

**Thyroid dysfunction in toxicosis / by William Seaman Bainbridge, A. M., Sc. D., M. D. New York.**

**Contributors**

Bainbridge, William Seaman, 1870-1947

**Publication/Creation**

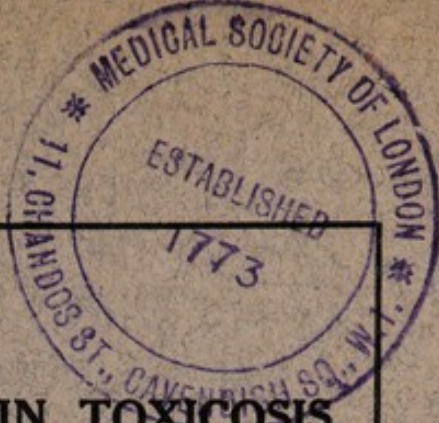
[Place of publication not identified] : [publisher not identified], [1926?]

**Persistent URL**

<https://wellcomecollection.org/works/csxebmnx>

**wellcome  
collection**

Wellcome Collection  
183 Euston Road  
London NW1 2BE UK  
T +44 (0)20 7611 8722  
E [library@wellcomecollection.org](mailto:library@wellcomecollection.org)  
<https://wellcomecollection.org>



**THYROID DYSFUNCTION IN TOXICOSIS**

*By*

**WILLIAM SEAMAN BAINBRIDGE, A. M., Sc. D., M. D.**

**New York**



**Reprinted from  
International Journal of Medicine and Surgery  
January, 1926**



## THYROID DYSFUNCTION IN TOXICOSIS.

By WILLIAM SEAMAN BAINBRIDGE, A. M., Sc. D.,  
M. D., New York.

As our title indicates, we shall try to confine this article, as far as possible, to one phase of dyscrinism—thyroid dysfunction as the result of systemic poisoning. As yet, the etiology of goiter is largely an unknown quantity—whether it is basically a disease of the thyroid gland or the result of auto-toxic or infective absorption is still an unsolved problem; but the writer wishes to go on record here, as he has in earlier communications, to the effect that he believes perversion of the thyroid function, with or without enlargement of the gland, to be due, in many instances, to focal infection or auto-toxic conditions.

Disturbance of the thyroid secretion is attributed to many causes—deficient nutrition, unsanitary surroundings, infectious diseases, heredity or psychic elements—but recent research has accumulated an impressive amount of evidence to prove that a large proportion of thyroid dysfunction is the result of systemic poisoning. The sources of such infection may be located anywhere in the body, but the tonsils, teeth, sinuses, the cervix uteri (or prostate and seminal vesicles) and intestinal tract are among the most important points of invasion, and I agree with such men as Hertoghe, McCarrison, Rowell and Chapple in their deductions that many pathological conditions of the thyroid may be traced directly to *intestinal* toxemia.

In an earlier paper<sup>1</sup> I called attention to the probability that auto-intoxication from any source would doubtless produce abnormal changes in the thyroid gland. The thyroid, as well as other glandular tissue, is dependent upon the character of its hemo-genous environment for its proper reaction in the body economy, and persistent retention of the putrefactive contents of the intestines must, of necessity, react morbidly on this environment. My hypothesis, therefore, is that a "goiter" that diminishes or disappears as the result of either therapeutic or surgical measures, which have the effect of draining the intestines (or a goiter which recedes as the result of eliminating any suspected source of infection in the body), is obviously due largely to that particular element of toxic absorption. The stability of the thyroid may be very seriously affected and the activity of the gland markedly impaired by a chronic static condition of the intestinal canal. It is true that the sufferer from chronic alimentary toxemia may acquire a passive immunity to the toxic reactions (perhaps due to having carried the causative toxic organism for such a prolonged period as finally to inhibit its activity) and that this immunity, while not absolute, is sufficient to save the patient, sometimes for years, from the serious sequelæ of his condition. It is also true that such a protective process may exist indefinitely until an added toxic element or a new site of infection may arise, and the combined toxemic forces prove too great a strain for the resistance of the host. The result may be evidenced

in lobulated breasts, enlarged or atrophic thyroids, or any number of other organic disturbances. Therefore, it is of paramount importance that before advising thyroid surgery, a preoperative survey of possible toxic centers in the patient be made and, wherever practicable, eliminated. While there are many goiter patients so thoroughly poisoned with thyroid toxins (from thyroid dysfunction of long standing) that such procedure will produce but little result and surgery of the thyroid will still be necessary, there are, doubtless, many other cases of "systemic goiter" which will respond to therapeutic or surgical measures directed towards the elimination of foci of infection or sites of toxic absorption.

"Systemic goiter," a term which I first used in a paper published in 1914<sup>2</sup>, was intended *at that time* to designate a condition the symptoms of which I described as due to the introduction of thyroid toxins into the system, and which caused increased or perverted thyroid secretion. However, since recent research has established the likelihood that dysthyroidism in many instances is due to systemic poisoning from teeth, tonsils, sinuses, etc., the term "systemic dysthyroidism" will be used herein to indicate a condition of the thyroid apparently due to some form of autotoxic absorption or focal infection, while "toxic dysthyroidism" will refer to a morbid condition of the body characterized by the presence of symptoms due to the excessive absorption of *thyroid secretion*, and with or without enlargement of the gland. With *toxic goiter* there is

usually increased frequency of action and palpitation of the heart, protrusion of the eyeball, tremor, and a number of mental and nutritional disturbances. All of these conditions may be present, or one or more may be absent. Moreover, the enlargement of the thyroid gland is of secondary importance, since it is not the *size* but the *activity* of the gland which is the determining factor, inasmuch as even abnormally *small* glands may over-secrete and cause symptoms. While the symptom-complex just described is commonly known as Graves' disease, Basedow's disease, or exophthalmic goiter, the term "toxic goiter" seems a more accurate appellation, since many of these cases exhibit no signs of exophthalmos, and the attachment of the name of an individual to a disease is most unscientific.

The following cases from my private practice are all *systemic* enlargements of the thyroid, due to underlying factors of autointoxication or focal infection in the patient, and are cited to illustrate the close relationship between the autotoxic processes in the system and certain abnormal changes in the thyroid gland. All of the cases described had reached the point where marked absorption of *thyroid* toxins had produced conditions of toxic goiter.

*Case 1.*—S. G., 29 years of age, male, married, physician. This patient consulted me October 13, 1916, for enlargement of the neck, associated with increased irritability, shortness of breath, tremor and marked protrusion of the eyes. He gave a history of having had an acute attack of frontal and maxillary sinusitis two years earlier, and this was

followed by the enlargement of the neck. Treatment with iodine had had no apparent effect on the growth.

On examination I found a bilateral goiter, more marked on the left side; pulse 110 with considerable pounding in the neck, and distinct protrusion of the eyes. The foci of infection in the sinuses had evidently cleared, but the thyroid toxins had poisoned the system to such an extent that thyroidectomy was indicated and was performed.

Following the operation the patient at once improved. The exophthalmos was relieved 75 per cent.; the tremor and shortness of breath disappeared. September, 1925, I examined him and found the eyes normal. There were no symptoms of thyroid dysfunction. There was a gain of forty pounds in weight, and the patient was recently accepted by two insurance companies on policies aggregating \$25,000.

*Case 2.*—E. M., 28 years of age, female, married eight years; three children. The family history is irrelevant.

She consulted me October, 1925, for a "goiter" which had been present about three months and the removal of which had been advised by two physicians.

On examination I found a pulse rate of 160; prominence of the eyes; tremor of the hands and an increase in the reflexes. There was a moderate-sized goiter. There were some suspicious teeth and two enlarged tonsils with pus. The uterus was retroverted and retroflexed and both ovaries pro-



lapsed. The teeth were x-rayed and some points of pus absorption found. These conditions were corrected. The tonsils, which were embedded and badly diseased, were enucleated by the writer.

After a period of rest in bed, laparotomy was performed October 25, 1925, and the appendix, which was kinked and full of fecal matter, and some cysts of the left ovary were removed. Divulsion and curettage, anterior colporrhaphy, extensive perineorrhaphy, and a Johnson-Willis suspension of the uterus were performed.

Following the operation she improved rapidly; the tremor became less noticeable, the eyes returned to normal, the pulse fell to 78, and the goiter practically disappeared. At present, December 5, 1925, the patient is in excellent condition.

In this instance it was far more important to relieve the several toxic causes of the thyroid dysfunction than to operate on the goiter, since simple removal of the goiter, without the elimination of these underlying toxic factors, would, doubtless, mean danger of recurrence of the thyroid condition at an early date.

The third case is one in which the autointoxication was so evidently that of *intestinal* toxemia, that operative procedure on the intestinal tract was absolutely indicated. The static condition of the intestines had been present for years and the thyroid gland had become so atrophic that correction of the underlying toxic factor only partially relieved the hypothyroidism and a certain amount of thyroid therapy must be continued.

*Case 3.*—N. M., female, married, 38 years of age. In February, 1910, when the patient first consulted me, she was very fat and flabby, suffered from constipation, and had much pain and distress in the abdomen.

Examination showed chronic appendix and many adhesions and kinks. The neck was very fat and the thyroid gland decidedly atrophic.

Operation was performed March, 1910. An omental mass from two hernial sacs, each the size of a hen's egg (in the abdominal wall), a chronically inflamed appendix and many adhesions were removed. The abdominal incision was through 8 cm. of adipose tissue.

After the removal of the bands and adhesions and the straightening of some important intestinal angulations and kinks, the patient improved very much. The tremor and shortness of breath disappeared with the other organic disturbances. She was then placed on thyroid, which she continued to take at intervals from 1910 to 1919. In that year she discontinued the thyroid.

January, 1921, this patient again consulted me for a lump in the right breast. She stated that she had hit the breast about a year earlier and the lump had slowly developed.

Examination showed that the patient was again suffering from autointoxication and hypothyroidism. She was given laxatives, intestinal antiseptics, thyroid, and a properly fitted brassiere was prescribed. Six weeks later the lump in the breast was softer and smaller and eventually it disap-

peared entirely. There is no longer any pain or distress in the abdomen; the mammary gland is perfectly normal, and the hypothyroidism has improved 75 per cent.

*Case 4* is that of a clear condition of autointoxication from the intestinal canal.

A. S., female, single, 24 years of age. This patient consulted me May 10, 1916. She had a large goiter; there was pulsation in the neck, and the eyes were prominent. The patient complained of pain and soreness over the appendicular region.

X-ray showed intestinal bands and adhesions, and operation was performed June 10, 1916.

The head of the colon, with very short mesentery, and the terminal ileum were found to be attached to the posterior wall with a massive attachment, binding the terminal ileum down in the cavity of the true pelvis. The appendix was subacutely inflamed and filled with material. The "last kink" was accentuated. These conditions were corrected, and the patient's recovery was uneventful.

Four months after the laparotomy there were no longer any abdominal symptoms, and the goiter had completely disappeared. Recently, November, 1925, the patient reported that she was in excellent condition and that there has been no recurrence of the goiter.

*Case 5.*—R. A., female, married, 36 years of age. For eight years prior to operation this patient was subject to attacks of bloating, severe abdominal pain, nausea and vomiting, with marked constipation. The attacks were usually from a few days

to a month apart and lasted eight or ten hours. Her neck was much enlarged, there was considerable pulsation, and the eyes were very prominent.

X-ray showed a marked ileo-pelvic band and the cecum rotated at the terminal ileum.

Laparotomy was performed March, 1916. The ascending colon was twisted over to one side, just below the hepatic flexure, and across this point was a mass of adherent omentum. Where the cecum rotated inward when the patient was erect, there was a point of almost complete obstruction. These conditions were corrected surgically.

Six months later the patient reported that the abdominal pain and nausea, the indigestion and constipation had disappeared. On examination it was found that the goiter had so diminished in size that operation for this condition was no longer considered necessary. To date, there has been no recurrence of the goiter.

In this case there remains no question but that the intestinal toxemia and resultant enlargement of the gland were due directly to defective drainage of the alimentary tract.

The consensus of opinion in the medical profession today is that most *substantial* goiters require operation. Surgical procedure is certainly the only method to be considered in the cases of *malignant, colloid, adenomatous* or *degenerated thyroid* glands. There have been very few instances reported where medical treatment has had any effect on colloid or adenomatous goiters.

*Simple goiters* require surgery only when pres-

sure symptoms are severe or for cosmetic reasons. In many of these elimination of the underlying *toxic* factor and careful therapy will restore the the gland to normal. Attempts to treat such goiters with iodine have frequently been successful. Recently iodized table salt has been advocated as a preventive of hyperthyroidism. Whether the favorable results obtained from iodine medication in such cases are due to the relief of the iodine deficiency in the thyroid, or to the anti-syphilitic effect of the drug on an underlying but unrecognized condition of "luetism"—"a much attenuated form of syphilis, modified by passing through a number of generations, and non-reactive to present laboratory tests"—remains a problem for future research. However, the indiscriminate administration of iodine, without careful supervision of the patient, is not a form of medication to be lightly employed. The favorable attitude towards iodine therapy for goiter is by no means unanimous.

Toxic goiter is usually considered a surgical disease and the *early, radical* removal of pronounced goiters of this type is advocated by many modern operators. Recent scientific investigation, however, favors the theory of an infective or toxic factor as the underlying cause of many of these systemic conditions, and the cases herein reported would seem to lend confirmatory emphasis to this viewpoint.

Since many goiters are, apparently, due to *toxic* or *infective processes*, the elimination of these sources of absorption in rational sequence, teeth,

tonsils, sinuses, cervix uteri (or prostate and seminal vesicles), alimentary tract, etc., in many cases, should precede thyroid surgery. To restore the normal functioning power of the gland—relief of the dysthyroidism, rather than mere removal of the tumor—is the primary object of treatment. As we have stated, there are cases of thyroid dysfunction with no evidence of goiter, and it is in such conditions as these that the basal metabolism test may prove a valuable aid in diagnosis. This test gives an index to the degree of *functional activity* of the thyroid gland and is a helpful method of governing the dosage of thyroid in hypothyroidism. The Massachusetts General Hospital, in metabolic tests on 1,000 patients with goiter, found that patients with *hyperthyroidism* invariably showed *increased* metabolism and those with definite clinical pictures of *hypothyroidism* showed *decreased* metabolism. A general conclusion is that the metabolism test is useful but, like the Wassermann and other laboratory tests, it must be interpreted with due regard for all clinical findings and its limitations and possibilities of error.

In conclusion, may I again stress the following points: There are some cases of thyroid dysfunction, particularly in the adolescent, which are amenable to medical treatment. In many of these, topical applications of cold to the neck, the use of sedatives, prolonged physical and emotional rest and thyroid therapy are measures which go far towards restoring the normal functioning power of the thyroid gland.

For *malignant, colloid, adenomatous* and degenerated thyroids, the one solution, as a rule, is operation.

In systemic goiter the removal, wherever possible, of all toxic sources should precede any contemplated surgical procedure on the thyroid gland. While thyroid surgery is certainly indicated in those instances of systemic goiter which evidence excessive and prolonged absorption of thyroid toxins, by the resultant organic disturbance in the patient, other goiters of this type may be completely eradicated by the elimination of the autointoxication.

Finally, the prevention of goiter depends, essentially, upon the building of normal tissue, a process possible *only* to a system free from any type of focal infection or toxemia—and free *particularly* from *intestinal poisoning*. Morbid anatomical changes in the thyroid and other glandular tissue are caused and fostered, in a large proportion of cases, by *inefficient drainage* of the intestinal canal.

#### REFERENCES.

- Bainbridge, W. S.: "The Thyroid Gland and the Toxemias." Illinois Medical Journal, January, 1922.  
Bainbridge, W. S.: "The Present Status of the Surgery of Systemic Goiter." Journal Michigan State Medical Society, April, 1914.  
Hertoghe, Eugene, and Bainbridge, W. S.: "Lues and Luetism in Abdominal Conditions." Franco-British Medical Review, January, 1925.

34 Gramercy Park.

