

1936- Sept

7

534

## Pronounced Weight Loss as a Precipitating Factor in Thyrotoxicosis

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### INTRODUCTION

THE etiology of thyrotoxicosis is unknown; but it is not infrequent that the clinical histories of patients who are ill with the disease contain major incidents to which the role of precipitating factor may be assigned. A grave psychic trauma, a severe or prolonged infection, an accident or a physiological strain such as puberty, pregnancy or the menopause have in the past been considered in this category.

It has been quite well established that the usual effect of starvation on the function of the thyroid gland in the human subject as measured by the basal metabolic rate is one of depression (Benedict<sup>1</sup>). On the other hand, hyperplasia of the thyroid can be induced in animals by starvation.

Recently we have been impressed in The Thyroid Clinic of The Massachusetts General Hospital by the occurrence of 21 cases of thyrotoxicosis which developed during or shortly after pronounced weight losses of various types. Since weight loss is one of the cardinal manifestations of thyrotoxicosis itself, we were careful to analyze these cases and to include only those cases in which the preliminary weight loss preceded the earliest possible symptom of thyrotoxicosis.

In eight cases (see Table) in the series the patients (Cases number 1 to number 8 inclusive) were under observation in the Out-Patient Department of the hospital during periods of voluntary reductions of weight, which were clearly independent of the weight loss incident to the thyrotoxicosis which later developed. Since reducing cures have increased in popularity in recent years, these cases are deemed worthy of special report together with 13 other cases whose histories, antecedent to the development of thyrotoxicosis, included pronounced weight loss due to independent diseases, e.g., of the gastrointestinal tract and other abnormal conditions of diet, food assimilation, and so forth (see Table I.).

### DISCUSSION

Although our attention was drawn to the possible role of pronounced weight loss as a precipitating factor in thyrotoxicosis by the cases in which thyroid medication was used as an aid in obesity regimes (Case numbers 4, 7 and 8), we were soon impressed with the fact that other types of reduction such as simple rigid dieting (Cases number 1, 2, 3, 5) and the prolonged use of saline purgation (Case number 6) were capable of pro-

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Weight Loss in Thyrotoxicosis—Hertz, Means

TABLE I.

CASE	NAME	AGE	SEX	Lab. No.	Wt. Loss	CAUSE	WHEN LOST	Developed Earliest Thyro- toxic Symptoms	BEHAVIOR OF WEIGHT WHILE THYROTOXIC
1	Stella H.	31	F	13167	29 lb.	Voluntary diet for 1 year +	1931 to 1933	Jan., 1934 palpitation	No weight loss during 1 year while thyrotoxic. Good appetite.
2	Fannie R.	54	F	15082	72 lb.	Prescribed diet in OPD for 5 years	1928 to 1934	May, 1935 diarrhea nervousness	Lost 40 additional pounds while thyrotoxic on high diet.
3	E. L. J.	39	F	14616	"30-40 lb."	Voluntary diet for obesity	July, 1934	Feb., 1935 eta. stopped nervousness	Continued weight loss with high diet.
4	Cardina M.	51	F	13790	53 lb.	Diet and thyroid gr. i - iii	March, 1934 to Nov., 1934	July, 1934 fatigability	Anorexia! Continued weight loss despite rest in bed.
5	Fannie Esc.	17	F	4462	15-20 lb.	Diet for ? Froeh- lich's syndrome	June, 1932 to Dec., 1932	Jan., 1933 Sweats +++ nervousness	Continued weight loss despite high food intake and good appetite.
6	Nellie Z.	39	F	13745	39 lb.	Diet and saline catharsis	Feb., 1934 to March, 1934	May, 1934 nervousness	Good appetite. Continued weight loss.
7	Nellie G.	50	F	12205	20 lb.	Thyroid treatment for "low rate"	Oct., 1933 to June, 1934	Jan., 1935 weakness	Continued weight loss. Appetite unchanged.
8	Alice deP.	27	F	12863	70 lb.	"Marmola" 4 mos. for obesity	1928 to 1929	1931 Goiter, staring expression	Gained 20 pounds during thyro- toxic period. Increased appetite!

ducing a similar effect. Indeed, weight loss due to such independent causes as diabetes, tuberculosis, peptic ulcer regimes and gastrointestinal disorders (colitis, Cases number 12, 13, 14, 16, 17, 18, 20 and 21) was quite capable of so doing. In Case number 19 there was complete observation while the patient remained on a highly restricted diet during a psychosis, and in Case number 15 the patient's thyrotoxicosis followed a period of relative starvation due to financial reverses.

In view of the above thesis it is not unlikely that certain of the other so-called common precipitating factors may exert this effect by causing decreased appetite, reduced food intake and consequent weight loss, which,

TABLE II.

CASE	NAME	AGE	SEX	LAB. NO.	CAUSE OF WT. LOSS
9	Annie I.	52	F	14016	Diabetes
10	Harold E.	33	M	H.289912	Ulcer treatment
11	Israel A.	53	M	14692	Diabetes
12	Kathrine C.	59	F	7045	Breast cancer
13	Joseph McL.	33	M	7389	Pulmonary tuberculosis (proved)
14	John R.	54	M	15301	Ulcer treatment
15	Isabella W.	43	F	14333	Poverty and starvation
16	Mrs. Farnum	58	F	15350	Restricted diet and G-I disorder
17	Mrs. Cantelon	48	F	15421	Colitis (with pus)
18	Wm. Ruggles	22	M	14521	Ulcerative colitis
19	Mrs. L.	53	F	15180	Psychosis and starvation
20	Anna Sh.	37	F	14426	Breast cancer
21	Eliz. Del.	59	F	8307	Ulcer treatment

in the past, has been taken to be a symptom of thyrotoxicosis without sufficient analysis. Anorexia in an emotional crisis, or during an infection, the vomiting of early pregnancy, the privation during poor economic conditions can conceivably be included in this category and deserve further consideration from this point of view.

The possible implications of this phenomenon, if it be a true one, are of considerable interest. Granted that through some fault in homeostasis a particular individual can develop thyroid overactivity in response to the stimulus of pronounced weight loss, what should be our interpretation of the nature of that stimulus? What is the mechanism of this attempt upon the part of the thyroid to correct the lowered metabolic rate of starvation? Or, is there a specific food or vitamin deficiency in the background of

what appears at first thought to be a vastly heterogenous etiology of thyrotoxicosis?

The possibility that such a phenomenon as we have described might be on the basis of iodine deficiency suggests itself at once. But, its occurrence on reduction diets rich in vegetables grown in a nongoitrous section (Boston) speaks strongly in favor of some other explanation.

It would seem that a closer experimental attack upon this problem which the clinic has suggested in this series of 21 cases is definitely indicated. A careful follow-up of the status of thyroid function at various stages of the treatment of large groups of obesity patients would be of great value.

REFERENCES

1. Benedict, F. G., Miles, W. K., Roth, P., and Smith, H. M.: Carnegie Institute of Washington, 1919, Pub. 218.

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