CLINICALLY INACTIVE THYROID *U.S.P.**

A Preliminary Report

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IN the last nine months we have observed a lack of clinical and protein-bound-iodine response in hypothyroid patients to a nonbrand name of thyroid *(U.S.P.)*. Several of these patients were euthyroid while on a brand of this preparation obtained from a different supplier.

TABLE 1. *Data in 9 Cases of Hypothyroidism.*

PATIENT	PROTEIN-BOUND IODINE WITH PATIENT EUTHYROID	DOSE OF *U.S.P.* THYROID		PROTEIN-BOUND IODINE ON DEFICIENT *U.S.P.* THYROID	DOSE OF DEFICIENT *U.S.P.* THYROID		PROTEIN-BOUND IODINE ON OTHER *U.S.P.* THYROID	DOSE OF OTHER *U.S.P.* THYROID	
	microgm./100 ml.	*gm.*	*gr.*	*microgm./100 ml.*	*gm.*	*gr.*	*microgm./100 ml.*	*gm.*	*gr.*
E.B.	8.2	0.24	4	3.8	0.24	4	6.8	0.24	4
R.G.	6.6	0.24	4	2.5	0.24	4	5.2	0.2	3
F.W.				3.1	0.2	3	5.6	0.2	3
H.R.	6.7	0.12	2	4.2	0.24	4	7.9	0.24	4
M.J.				5.6	0.4	6	7.3	0.4	6
G.D.	5.8	0.12	2	3.0	0.2	3	8.1	0.2	3
V.G.				4.2	0.2	3	8.0	(0.2 mg. of levothyroxine)	
L.T.	6.6	0.12	2	2.3	0.3	5	5.5	0.3	5
J.H.S.	—	—	—	3.0	0.24	4	4.4	0.24	4

TABLE 2. *Lack of Clinical and Protein-Bound-Iodine Responses to Deficient Thyroid.*

PATIENT	PROTEIN-BOUND IODINE WITH PATIENT EUTHYROID	DOSE OF *U.S.P.* THYROID		PROTEIN-BOUND IODINE ON DEFICIENT *U.S.P.* THYROID	DOSE OF DEFICIENT *U.S.P.* THYROID	
	microgm./100 ml.	*gm.*	*gr.*	*microgm./100 ml.*	*gm.*	*gr.*
R.J.	—	—	—	1.9	0.12	2
B.C.	5.9	0.12	2	4.7	0.2	3
R.Y.	8.7	0.3	5	2.2	0.3	5
L.P.	7.7	0.2	3	3.1	0.3	5
M.H.	5.1	0.15	2½	3.5	0.15	2½
F.T.	8.3	0.24	4	5.5	0.2	3
J.P.	5.3	0.2	3	4.9	0.24	4
R.G.	—	—	—	1.1	0.24	4
K.D.	—	—	—	4.1	0.24	4
W.H.	—	—	—	3.1	0.2	3
J.T.	5.3	0.2	3	3.0	0.2	3
M.K.	5.3	0.12	2	3.3	0.2	3

Table 1 presents 9 cases of hypothyroidism showing the lack of response of protein-bound iodine with the deficient thyroid and their improvement when changed to another type of thyroid *(U.S.P.)*¶ or levothyroxine.‖ Clinically, while on the deficient preparation they complained of tiredness, intolerance to cold, constipation, dryness of the skin and weight gain. When medication was changed the symptoms disappeared.

Table 2 shows 12 cases with symptoms of hypothyroidism and no response clinically and by determination of protein-bound iodine with the deficient thyroid preparation.

Table 3 presents the data in a patient with primary myxedema in whom reversal of well-being occurred when the deficient thyroid preparation was given. When the protein-bound iodine was 4.0 microgm. per 100 ml. in December, 1960, symptoms of intolerance to cold, weight gain and tiredness appeared. In June, 1961, while she was receiving this preparation, 0.2 gm. (3 gr.), the hypothyroid symptoms persisted, and congestive heart failure similar to the initial episode in 1953, when she was myxedematous, was noticed. The protein-bound iodine on June 21 was only 2.8 microgm. per 100 ml.

DISCUSSION

The deficient thyroid preparation was assayed chemically and shown to have the amount of iodine required by the *United States Pharmacopoeia*. More extensive studies, including chromatography, are being conducted in other centers.

SUMMARY AND CONCLUSIONS

A thyroid preparation supplied as a nonbrand name was shown to be ineffective clinically and by protein-

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¶In the form of thyroid *(U.S.P.)*, Armour Pharmaceutical Company, Kankakee, Illinois.

‖In the form of Synthroid Sodium, Flint, Eaton and Company, Morton Grove, Illinois.

bound-iodine determinations in 22 cases of hypothyroidism. When another type of thyroid or levothyroxine was given a good clinical and protein-bound-iodine response was obtained. The deficient preparation contained the amount of iodine required by the *United States Pharmacopoeia*. This preliminary report indicates the need for new *U.S.P.* standards for thyroid preparations.

TABLE 3. *Data in I.E., a Sixty-six-Year-Old Woman with Primary Myxedema.*

DATE	DOSE OF *U.S.P.* THYROID		PROTEIN-BOUND IODINE	CHOLESTEROL	SYMPTOMS
	gm.	*yr.*	*microgm./100 ml.*	*mg./100 ml.*	
1/16/53			0.5	555	Myxedematous congestive heart failure
1/23/57	0.12	2	3.2		None (patient euthyroid)
12/10/58	0.2	3	5.0	250	None (patient euthyroid)
7/22/59*	0.2	3	7.0	166	None (patient euthyroid)
12/7/60	0.15	2½	4.0	266	Patient tired & intolerant to cold
1/4/61	0.2	3			Patient tired & intolerant to cold
6/21/61	0.2	3	2.8		Congestive heart failure
7/24/61	0.24	4	3.8		Patient hospitalized with congestive heart failure & hypothyroidism

*Deficient preparation started at this time.

HAZARDS TO HEALTH

Air Pollution — Engineering Aspects

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AIR pollution from man-made sources is not new — it has been present for a long time, as witness the words that Shakespeare put into the mouth of Hamlet: "This most excellent canopy, the air — why it appears no other thing to me than a foul and pestilent congregation of vapours." Since World War II the problem has become more intense as a result of the vast population gains in urban areas, with the attending growth in number of motor vehicles and the usage of fuel for these, for power plants, for industries and increased domestic and commercial activities. Pollution has become not only greater in quantity but infinitely more complex in kind. In some areas the limit of the ability of the atmosphere to dilute and disperse contamination has been reached.

The adverse effects have been well publicized and are identified as smoke in Chicago, smog in Los Angeles and smaze (smoke combined with haze) in New York City. This publicity has built up public interest in and a demand for clean air. In Massachusetts the General Court recently authorized the formation of the Metropolitan Air Pollution Control District and gave authority to the Department of Public Health for the adoption of a series of special regulations to prevent pollution or undue contamination of the atmosphere within the metropolitan area of Boston. The Congress has delegated authority to the Surgeon General of the United States Public Health Service to assist the states in the determination of the epidemiologic significance of air pollution and the causes and methods for control of the sources to obtain an environment satisfactory to man.

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The Public Health Service has defined air pollution as "The presence in the atmosphere of substances put there by the acts of man in concentrations sufficient to interfere directly or indirectly with his comfort, safety or health, or with the full use and enjoyment of his property."

CAUSES

The greatest source is from combustion — fossil fuels such as coal, oil and gas, and refuse burning. I estimate that during the winter 1500 tons of aerosols are discharged per day in the metropolitan area of Boston from domestic, municipal, industrial and transportation sources. These would include 500 tons of organics, principally hydrocarbons, 150 tons of nitrogen dioxide, 250 tons of sulfur dioxide and 200 tons of assorted acids. The automobile is a big offender, with an estimated discharge of 300 tons of hydrocarbons per day. Open dumps, backyard burning and other sources of refuse contribute 10 tons of solids and 20 tons of organics to the atmosphere daily. Home heating by oil would contribute another 60 tons of sulfur dioxide and 60 tons of organics and solids per day. The industrial sources of air pollution in Massachusetts are many, including power plants, industrial heating and power, metallurgical, chemical, paper and food processing, tanneries and rubber and textile plants.

HEALTH EFFECTS

Man breathes 1500 quarts of air per day, or about 35 pounds. He eats about 1 pound of food and drinks 2 pounds of water, and with this has a very high level of food and water sanitation — a distinct achievement