Townsend Letter
The Examiner of Alternative Medicine

From the Townsend Letter for Doctors & Patients
November 2005

Order this issue!

Alan R. Gaby, MD's Response to:
A Rebuttal of Dr. Gaby's Editorial on Iodine

Note: *Gaby's original editorial is also online, as well as a second rebuttal by Drs. Abraham and Brownstein.*

More on High-dose Iodine
by Alan R. Gaby, M.D.

In an editorial in the Aug/Sept issue of the *Townsend Letter*, I expressed concern about potential adverse effects that might occur with the routine use of megadose iodine therapy, which is currently being advocated by some practitioners. I cited research indicating that even modest increases in dietary iodine intake might increase the incidence of hypothyroidism, autoimmune thyroiditis, and possibly hyperthyroidism. In the following issue of the *Townsend Letter*, Drs. Abraham and Brownstein wrote a rebuttal to my editorial. They stated that, in Brownstein's experience with 4,000 patients at the Center for Holistic Medicine in West Bloomfield, Michigan, these side effects have not occurred; on the contrary, high-dose iodine has been effective in some cases as a treatment for these conditions. Although high-dose iodine therapy has a definite place in clinical medicine, I believe that some of their remarks warrant comment.

First, it does not seem appropriate to use the term "orthoiodosupplementation" to describe the treatment they are recommending. That term is borrowed from Linus Pauling's "orthomolecular medicine," which refers to the concept of creating the optimal molecular environment in the body ("orthomolecular" means "the right molecules"). Defining the optimal dosage range as an amount that is 40 to 320

times the usual dietary intake obfuscates any debate about whether such a high intake is desirable or safe. Therefore, until iodine doses of 6.25-50 mg per day are proven to be optimal, it would be more logical to refer to these doses as "high-dose iodine therapy."

Drs. Abraham and Brownstein stated that the thyroid disorders I mentioned that resulted from iodine supplementation occur mainly with "organic forms" of iodine, such as amiodarone and certain iodine-containing dyes used in radiology. However, all but one of the references I cited discussed the adverse effects of inorganic iodine. The other article concerned the use of an iodophore, which is a surfactant molecule that slowly releases inorganic iodine. As surfactants would not by themselves be expected to affect thyroid function, one might presume that the released inorganic iodine was responsible for the reported adverse effects.

I would also question the statement that our medical predecessors recommended daily iodine intake of 12.5 to 37.5 mg from Lugol's solution. While Dr. Lugol did use those doses, they were recommended primarily to treat infections (iodine is a broad-spectrum antimicrobial agent) and hyperthyroidism, not as routine nutritional support for the average person.

Regarding the safety data from the Michigan clinic, Dr. Brownstein learned about high-dose iodine only about two years ago, from a letter written by Dr. Abraham in the *Townsend Letter*. For a three-doctor practice to initiate high-dose iodine therapy on 4,000 patients over a two-year period seems like a daunting endeavor, and one wonders how meticulously these patients were monitored for adverse effects. How many patients discontinued the treatment because of side effects and never returned to the clinic to report their experiences? How many patients who did return were questioned in detail about potential side effects, including fatigue and other symptoms of hypothyroidism? How many patients showed a decline in their serum thyroxine level that was judged to be clinically insignificant because it remained in the normal range? Abraham has in fact, observed such decreases in thyroid hormone levels in patients receiving iodine therapy. One should not automatically assume that these changes are benign. Research has shown that each person has a unique "set point" for serum concentrations of T4, T3, and TSH. Any iodine-induced deviation from these set points may be result in suboptimal thyroid function for that person, even if all measurements remain within the normal range.[1] How many patients were tested serially to identify the appearance of thyroid antibodies during treatment with iodine? Before one could confidently conclude that high-dose iodine is safe for 99% of the population (as stated by

Abraham and Brownstein), it seems that a systematic toxicity study would be necessary.

It is also worth considering that the positive results observed in Michigan might not be reproducible in other geographical areas. Drs. Abraham and Brownstein hypothesized that the beneficial effect of iodine is due in part to removal of bromine from the body. In 1973-4, several thousand Michigan dairy farms were contaminated by polybrominated biphenyls (PBBs) from an industrial accident. This bromine-containing pollutant is known to persist in the body for long periods of time. Five years after the accident, 97% of adipose tissue samples taken from Michigan residents had detectable levels of PBBs,[2] and 96% of breast milk samples from women in densely populated areas of the state contained this chemical up to three years after the accident.[3] Because of its exceptionally long half-life, it is reasonable to assume that many Michigan residents still have a body burden of PBBs. As thyroid dysfunction is known to occur in people exposed to PBBs, it is possible that some of the beneficial effects attributed to iodine therapy were due to a reduction of the body burden of PBBs.[4]

High-dose iodine therapy is of great value in some circumstances. We should not forget, however, that this treatment was abandoned in the past, because it caused many deaths from heart failure, as well as a long list of other side effects. The doses used then were higher than those currently being advocated. However, it is premature to assert that more modest doses do not cause more modest side effects.

References

1. Andersen S, et al. Narrow individual variations in serum T4 and T3 in normal subjects: a clue to the understanding of subclinical thyroid disease. *J Clin Endocrinol Metab* 2002;87:1068-1072.
2. Wolff MS, et al. Human tissue burdens of halogenated aromatic chemicals in Michigan. *JAMA* 1982;247:2112-2116.
3. Brilliant LB, et al. Breast-milk monitoring to measure Michigan's contamination with polybrominated biphenyls. *Lancet* 1978;2:643-646.
4. Bahn AK, et al. Hypothyroidism in workers exposed to polybrominated biphenyls. *N Engl J Med* 1980;302:31-333.

March 20, 2006