The History of Iodine in Medicine
Part III: Thyroid fixation and medical iodophobia

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The thyroid gland-iodine connection was known just a few years following the discovery of iodine in seaweed in the 1811. Only 8 years after this discovery, iodine was used effectively in the treatment of simple goiter. However, the medical uses of iodine during the first century since the discovery of iodine were not restricted to diseases of the thyroid gland only but covered a wide range of clinical conditions (1).

In the early 1920's, Marine reported a positive effect of iodide supplementation at 9 mg/day in the prevention of simple goiter among adolescent girls (2,3). That amount of iodine was based on research performed in farm and laboratory animals regarding the effect of iodine on thyroid function and also overall performance. However, in Marine's studies on adolescent girls, the only parameter assessed was the presence of goiter. Following Marine's studies, iodine sufficiency became associated with the absence of goiter, not overall performance such as grades in classes, number of absences due to sickness, etc.

As a public measure to control goiter, iodization of table salt was implemented successfully in the USA between 1917 and 1924. That is, iodization of table salt was successful in decreasing markedly the incidence of simple goiter in the supplemented population. Keep in mind that the amount of bioavailable iodine (0.05 mg/day) needed to prevent cretinism, endemic goiter and hypothyroidism is 60 times less than the amount of iodide (9mg/day) used by Marine (2,3) in the original studies. Thyroidologists assumed that, with iodization of table salt, iodine deficiency became a thing of the past. That was the beginning of thyroid fixation.

Prior to the iodization program, the public was relying on iodine preparations from apothecaries for their iodine needs. The recommended daily amount of iodine was 0.1 ml to 0.3 ml Lugol containing 12.5 to 37.5 mg elemental iodine (4). This is exactly the amount of iodine needed for whole body sufficiency, based on a recently reported iodine/iodide loading test by the author (4). Some propaganda was used following iodization of salt to discourage the public from using the iodine preparations such a Lugol solution and to rely instead on iodized salt for their iodine needs. In 1926, physician C.L. Hartsock, from Cleveland, Ohio (5), wrote:

> "…iodized salt is now being very much more extensively used by the public than other forms of iodine, such as sodium iodide, iodostarine and compound solution of iodine (Lugol's solution), probably because of the propaganda to insure its use…".

Iodized salt was unfortunately used as substitute for the previously recommended forms of iodine/iodide. The bioavailable iodide from iodized salt is only 10% of the estimated 0.75 mg iodide in iodized salt consumed per day (6). That amount, 0.075 mg of bioavailable iodide, represents less than 1% of the amount of iodide used in Marine's study (2,3) that is, 9 mg; and also less than 1% of the recommended daily intake of iodine from Lugol solution. Implementation of iodization of

salt was associated with an increased incidence of autoimmune thyroiditis (4).

Instead of iodized salt, Hartsock (5) recommended the use of a tablet of iodine/iodide in known and fixed amounts as the best form of supplementation, just like the most popular form of supplementation used today for vitamins, minerals and trace elements.

> "Tablets containing definite amounts of iodine seemed to be the method of choice."

With the availability of thyroid hormones in the 1930's, iodine was completely ignored by thyroidologists in the treatment of iodine deficiency-induced goiter and hypothyroidism. A textbook entitled "Diagnosis and Treatment of Diseases of the Thyroid", edited by Amy Rowland and published in 1932, contained chapters from 24 thyroidologists of that time (7). Although the most common cause of hypothyroidism and simple goiter worldwide is iodine deficiency, the recommended treatment of hypothyroidism was summarized in 2 sentences:

> "The treatment of hypothyroidism of any type consists merely in the substitution of thyroid extract for the deficient secretion. Any form of prepared gland or the active principle, thyroxin, may be used."

Iodine neglect in the 1930's by thyroidologists progressed to medical iodophobia in the late 1940's and early 1950's. Following World War II, there was a systematic attempt to remove iodine from the food supply of Christian America. Iodophobic misinformation, well synchronized with the introduction of alternatives to iodine supplementation in medical practice, strongly suggest a well planned conspiracy by agents of foreign powers planted at strategic positions in academia and the regulatory agencies (8). U.S. physicians became the stooges of these agents of foreign powers. Iodophobic misinformation permeated all textbooks of medicine and the subspecialties. From books written by physicians for physicians and for the consumers, iodophobia, which has reached pandemic proportions, trickled down to books written by lay persons for consumers (4,9).

A new syndrome, medical iodophobia, was recently reported (4). Medicoiodophobes suffer from: A) Split personality which results in iodophobia within the orthoiodosupplementation range previously used safely and successfully in medical practice and iodophylia for megadoses of iodide (up to 12gm/day). B) Double standards, which render those physicians intolerant to the minor side effects of the inorganic forms and extremely tolerant toward severe side effects of the radioactive and organic forms. C) Amnesia toward the inorganic nonradioactive forms when making therapeutic decisions. D) Confusion, attributing the severe side effects of organic iodine containing drugs to inorganic iodine/iodide. E) Altered state of consciousness, allowing doublethink, doublespeak and contradictory logic to become acceptable.

Although the factors involved in medical iodophobia are still unknown, decreased cognition seems involved. Since low iodine intake is associated with intellectual impairment, deficiency of this essential element cannot be ruled out, and if present, would create a self-perpetuating phenomenon. Needless to say that medical iodophobia is contagious and can be transmitted to patients and other physicians (iatrogenic iodophobia). Although there is yet no official report from

the Center for Disease Control regarding the prevalence of medical iodophobia in the U.S. medical community, it is likely that this syndrome has reached pandemic proportion.

Medical iodophobia will remain a syndrome until the causes are discovered and effective therapy implemented. The disastrous effect on the U.S. population of the zombification of the medical profession through iodine deprivation is already evident. Implementation of the orthoiodosupplementation program in the medical community is highly recommended. The increased cognition of health care professionals, resulting from orthoiodosupplementation, will eventually trickle down to patients in the form of a more enlightened approach to patient care.

Before World War II, non-radioactive forms of inorganic iodine were considered a panacea for all human ills (10), but today, they are avoided by physicians like leprosy. Who, what, killed iodine? The first nail in the iodine coffin was the publication by Wolff and Chaikoff from U.C. Berkley in 1948 (11), describing their finding in rats administered iodide in increasing amounts by intraperitoneal injection. When serum inorganic iodide levels reached 0.2 mg/L, that is 10^{-6}M, radioiodide uptake by the thyroid gland became undetectable. The correct interpretation would be: Iodide sufficiency of the thyroid gland was achieved when serum inorganic iodide levels reach 10^{-6}M, as we previously discussed (9). But Wolff and Chaikoff concluded that serum inorganic iodide levels at a concentration of 10^{-6}M blocks the synthesis of thyroid hormones, resulting in hypothyroidism and goiter. These authors did not measure thyroid hormones in the rats studied. Hypothyroidism and goiter were not observed in those rats. This fictitious phenomenon became known as the Wolff-Chaikoff Effect (12). Because these law-abiding rats refused to become hypothyroid and instead followed their normal physiological response to the iodide load, they were unjustly accused of escaping from the law of the Wolff-Chaikoff Effect. Labeling these innocent rats as fugitives was a great injustice against these rodents.

The second and final nail in the iodine coffin was hammered in by Wolff in 1969 (12). By 1969, Doctor Wolff had moved to the National Institute of Health from U.C. Berkley. Wolff arbitrarily defined 4 levels of "iodine excess". The first level of excess started with intake above 0.2 mg/day, and iodide intake of 2 mg or more was considered "excessive and potentially harmful". By the 1970's, physicians concluded that one must avoid inorganic non-radioactive iodine like leprosy, unless it was incorporated into the toxic organic iodine containing drugs. Then iodine could be tolerated because iodine could be blamed for the toxicity of these drugs.

Whereas the first wave of medical iodophobia was initiated in 1910 by the pen of one man, Swiss surgeon, Nobel laureate, Professor Theodore Kocher and lasted some 15 years (1910-1925) (1), the second wave of medical iodophobia initiated in 1948 by the pen of two men, Wolff and Chaikoff, is alive and well even after some 60 years of existence.

As unbelievable as it may sound, the Kocher Iodophobic Effect was initiated by a report from Kocher one year after he received the Nobel Prize, stating that he (Kocher) experienced symptoms of hyperthyroidism following ingestion of potassium iodide. One man, reporting his experience using iodine on himself initiated the first wave of medical iodophobia. After 15 years of intimidation, preventing the widespread effective use of iodine, due to the Kocher Iodophobic

Effect, physicians were able to escape from the Kocher inhibition because of poor synchronization of iodophobic publications and a small nucleus of enlightened members of the medical profession. The amazing success and long duration of the Wolff-Chaikoff Iodophobic Effect on the medical community is most likely due to the well synchronized timing of a series of iodophobic publications and also due to iodine deprived and zombified physicians who were unable to escape from the Wolff-Chaikoff Effect. The rats used in the Wolff-Chaikoff Experiment were successful in escaping from Wolff-Chaikoff Effect because they received significant amounts of iodine, improving their cognition.

The proper terminology for the Wolff-Chaikoff effect is "The Wolff-Chaikoff Iodophobic Domino Effect". I will give one example, just one example, of the iodophobic domino effect of the Wolff-Chaikoff 1948 publication (11), resulting in the removal of iodine from a very important staple food in the USA, that is our daily bread which contained the full RDA of 0.15 mg per slice for a period of about 20 years between 1960 to 1980 (4,9).

Wherefore do ye spend money for that which is not bread? (Isaiah 55:2)

In the early 1960's, potassium iodate was added to bread as a dough conditioner. Iodate was added with the purpose of oxidizing sulfhydryl groups of flour proteins and thereby improving the rheological properties of the dough. By oxidizing sulfhydryl groups, iodate is reduced already during mixing of the dough and it reaches the consumer as iodide (13). This was an oversight by the agents of foreign powers planted at strategic positions in academia and the regulatory agencies. Obviously, they are not infallible. As mentioned previously, one slice of bread contained the full RDA of 150 ug (14,15). This amount of the dezombifier iodine in a major staple food of Christian America could not be tolerated for long. Something had to be done and fast. The Wolff-Chaikoff Domino Effect was used to deiodize bread, concomitant with an increased concentration of the goitrogenic, carcinogenic and zombifying bromate in our food and water supplies (8). The following describes the sequence of events in this Domino Effect.

Because of isotope dilution effect, the percent of radioiodide uptake by the thyroid gland decreased from 20-30% to 10-20%, following iodization of bread. In 1965, London et al (16), from the National Institute of Health, evaluated the amount of iodine present in 32 bakery products from 12 different commercial bakeries. They reported that a typical diet contributed to approximately one mg of iodine per day and 726 ug came from bakery products. Concern was expressed over the inhibition of thyroid hormone synthesis in thyrotoxic patients at those levels of iodine. The last sentence of their publication read: "One milligram of iodine will suppress the uptake of radioactive iodine by the normal thyroid gland, probably by simple dilution of the dose, and may considerably reduce organic binding of iodine in the thyroid glands of thyrotoxic persons (8)." Reference 7 of their manuscript is a study published in 1949 by Stanley (17) one year after the Wolff-Chaikoff Effect was reported in rats (11). The first paragraph of Stanley's manuscript stated the objective: "The interest of thyroidologists was recently aroused by the demonstration by Wolff and Chaikoff (2) that, with levels of serum iodide higher than 20 to 30 micrograms per cent, organic binding of iodine in the rat thyroid was inhibited. Extension of these observations to man was undertaken…".

The interest of thyroidologists could not have been aroused so quickly by the publication of Wolff and Chaikoff in The Journal of Biological Chemistry (11), a journal involved in publishing research in the basic sciences, not clinical medicine. The thyroidologist with aroused interest was Stanley himself who obviously had insider's information in order to publish his manuscript within a year following the Wolff-Chaikoff publication, considering the fact that it takes several months for the review process in peer review journals, and that it would have required several months for him to design and perform his experiments after reading the Wolff-Chaikoff paper. During the year Stanley published his "extension of the Wolff-Chaikoff Effect to man", he co-authored a paper with Astwood on the use of goitrogens in the management of patients with Graves' disease as an alternative to inorganic iodine/iodide.

It is a strange coincidence that the investigators who authored the iodophobic publications, regarding the so-called inhibition of organic binding of radioactive iodide in the thyroid gland by the administration of inorganic nonradioactive iodide, were also involved in testing goitrogens in laboratory animals and in normal human subjects; and implementing the use of these goitrogens as an alternative to inorganic iodine/iodide in patients with Graves' disease (4).

Some 4 years after London's publication (16), Pittman et al (14) reported in 1969 on the negative impact of iodization of bread, that is, as far as Pittman et al (14) appraised it. Remember this is the same year Wolff published his iodophobic review (12). Again, timing and synchronization of iodophobic misinformation is critical for the destruction of Christian America. Events that seem unrelated but well synchronized for maximum effect is a key ingredient for the successful outcome of deception.

Pittman et al (14) compared the mean value of the 24 hr radioiodide uptake by the thyroid gland in a group of 63 euthyroid subjects prior to iodization of bread with another group of 53 euthyroid subjects following the use of potassium iodate in bread. These investigators also measured 24 hr urine iodide levels and serum inorganic iodide in some subjects of both groups. The 24 hr radioiodide uptake by the thyroid gland for both groups were ($\times\pm$ SD): pre-iodization of bread: 28.6 $\pm$ 6.5%; and post-iodization of bread 15.4 $\pm$ 6.8%. We have previously reported the correlation between 24 hr radioiodide uptake by the thyroid gland with the average daily intake of iodine, based on a review of the published literature (9). The data simplified is displayed in Fig. 1. Thyroid gland sufficiency for iodide is achieved with a daily intake of 6 mg. For whole body sufficiency, daily intake between 12.5 to 50 mg is required (4).

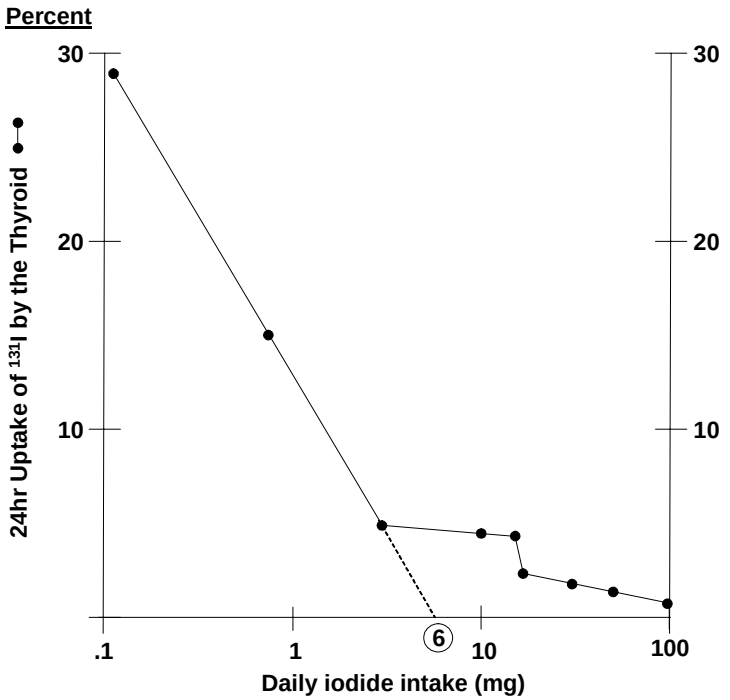

Fig. 1 Percent 24hr uptake of radioiodide following intake of increasing amount of iodide. Thyroid sufficiency is achieved with a daily iodine intake of 6mg, that is where the slope crosses the x axis at zero radioiodide uptake.
(Modified from Abraham et al, Original Internist 9:30-41, 2002).

Pittman et al (14) estimated a mean daily intake of 680 ug, that is 0.68 mg iodine in the group studied post-iodization of bread. These investigators were alarmed by such unexpectedly "excessive" intake of iodine, resulting in these subjects being "heavily loaded with iodine". This is a quote from the discussion section of their publication:

> "Evaluation of several aspects of iodine kinetics in 30 of our euthyroid subjects revealed them to be heavily loaded with iodine. We had anticipated that local subjects probably ingested liberal quantities of iodine, but we had not expected to find such high values as 680 ug per day for the urinary iodine excretion or 1.9 ug per 100 ml for the plasma inorganic iodide concentration (PII). These values are far in excess of most in the literature and approach those found in groups ingesting diets unusually rich in iodine."

Pittman et al were referring to mainland Japanese who consume a daily average of 13.8 mg (13,800 micrograms) of iodine from seaweed when they mentioned "groups ingesting diet unusually rich with iodine". Based on statistics generated some 20 years ago, mainland Japanese represent one of the healthiest nations on earth (4,9). At the time of Pittman's publication, iodophobic misinformation from the Wolff-Chaikoff's Domino Effect was so widespread that bread makers were already looking for an alternative to iodates as dough conditioners. They first considered azodicarbonamide, but it was too toxic, so they settled for bromate, a goitrogen with carcinogenic and zombifying potentials (4). Pittman et al (14) were elated with this move by bakers to replace iodates with azodicarbonamide. They stated:

> "Bread makers are using a new organic agent, azodicarbonamide, to an increasing extent to replace the halogens. If this trend continues, dietary iodine from this source may fall to low levels."

To recapitulate on the Iodophobic Domino Effect of the Wolff-Chaikoff forgery:

1948: Wolff-Chaikoff (W-C) forgery (11)
1949: Stanley supposedly extended the fictitious W-C effect observed in rats to humans (17)
1965: London et al (16) from the National Institute of Health quoted Stanley's forgery to alarm their readers about their findings of "large quantities" of iodine in bread
1969: Pittman et al (14) confirmed London's findings of "excessive iodine" in bread
1969: Wolff's iodophobic review (12)
Late 1970s to Early 1980s: Bakers replace iodate with bromate as a dough conditioner. Bromate is a goitrogen, carcinogen, and a zombifying agent (4).
From 1980 to 2000: Increased prevalence of obesity, diabetes, hypertension, cancer of the breast and thyroid glands in the U.S. Populations (4).

We have previously presented evidence that iodophobic misinformation in medical textbooks may have contributed to the high prevalence of breast cancer in the U.S. female population (4,9). Unfortunately, the latest Ninth Edition of Werner & Ingbar's The Thyroid, published in 2005 (18) contains the same iodophobic misinformation promulgated in the Eighth Edition published in 2000 (19). In the Eighth and Ninth Editions, Roti and Vagenakis wrote the section on "Effect of Excess Iodide" (20,21). Quotes from the Eighth Edition:

> "Strong evidence indicates that excess iodide can induce thyroid dysfunction, and these iodine-induced abnormalities in thyroid function are the subject of this subchapter."

> "Occasionally drinking water may be a source of excess iodine intake, such as in some Chinese countries where the drinking water has an iodine concentration of 300 to 462 μg/L. The population residing in those areas has a urinary iodine excretion rate as high as 900 μg/L."

These authors used micrograms instead of milligrams to make the numbers appear "excessive". They considered iodide concentrations between 300 to 462 μg/L (0.3- 0.46 mg/L) in drinking water as excessive. Yet, studies performed in the U.S. for 5 years in a prison inmate population consuming drinking water containing 1 to 2 mg/L (1,000 to 2,000 ug/L) of iodine (22) reported no complication.

> "Because of the increasing difficulty experienced by many communities in achieving satisfactory disinfection of public water supplies with acceptable concentrations of chlorine, a feasibility study on the use of iodine for this purpose was undertaken". "The effectiveness, ease of administration and palatability were prime reasons for considering iodine as a disinfectant of community water supplies". "effective

> bacteriological control of the water was maintained by all concentrations of iodine used in this study". "At an iodine concentration of 1 mg/liter (1 ppm), the water met all standards for safety and palatability (1962 USPHS Drinking Water Standards)". "During the five years in which this study was conducted no instances of urticaria or iodism were observed. … for serum thyroxine were unaffected by iodination of the water supply. … None of the prison inmates developed clinical evidence of hyperthyroidism or hypothyroidism throughout this study."

Several other studies confirmed the safety of inorganic non-radioactive iodine in daily amount greater than the amount Roti and Vagenakis considered toxic. For example, Clement (23) in Tasmania, reported that a daily intake of 1.4 mg of potassium iodide (10 times the RDA) by infants and children for 16 years resulted in reduction in the prevalence of goiter, but in some regions, that amount of iodine was not sufficient enough to have a significant effect on the rates of goiter.

In the 2005 Edition (21), Roti and Vagenakis repeated the same iodophobic misinformation promulgated in 2000 Edition and added a new one:

> "A group of American volunteers working in west Africa had a median urinary iodide excretion of 5.048 ug/L, due to a faulty iodination system, and some developed goiter and subclinical hypothyroidism (26)."

A review of their reference 26 revealed that this manuscript was poorly documented and should not have qualified for publication in the Journal of Clinical Endocrinology & Metabolism unless some heavy weight coauthor threw his weight around to get it through. In the manuscript entitled "Effects of Chronic Iodine Excess in a Cohort of Long-Term American Workers in West Africa" by Pearce, et al (24), 102 Peace Corp volunteers were evaluated during and 30 weeks after they cease to ingest water from filters containing **organic-iodine iodophores**. During the period the subjects were using the iodine containing filters, the urinary concentrations of iodide had a mean value of 5 mg/L. Serum iodide levels had a mean value of 0.29 mg/L. Based on renal clearance of iodide, that is 43.5 L/day (6), the average daily intake of iodine in these subjects calculated from the mean serum iodide level is: 0.29 mg/L × 43.5 L/day = 12.6 mg/day. This is the average daily intake of 60 million mainland Japanese (4,9), one of the healthiest populations on planet earth. The following quotes from Pearce's publication are evidence of a faulty experimental design. It is very surprising that such a mediocre manuscript made it through just because it is iodophobic:

> "Corps volunteers were authorized to receive a follow-up evaluation by an endocrinologist after returning from Niger. Some follow-up evaluations were incomplete, as some of the subjects chose not to visit an endocrinologist upon returning, and different endocrinologist obtained different follow-up laboratory studies. … Ultrasound evaluation was not performed. … No volunteers had overt symptoms of thyroid dysfunction as evaluated clinically."

In the discussion section of their publication, Pearce et al did not fail to mention the fictitious Wolff-Chaikoff Effect as if it was a proven fact. More than 50 years after the Wolff-Chaikoff

forgery, it is still quoted in iodophobic publications.

> "Acute excess iodine ingestion has long been known to result in a transient decrease in iodine organification, termed the acute Wolff-Chaikoff effect."

Attempts to reproduce the Wolff-Chaikoff experiments in rats by other investigators were unsuccessful. In vitro studies revealed that concentrations of iodide as high as 10^{-2}M were required to interfere with the mechanisms involved in cellular uptake and organification of iodide (25). These amounts are 4 orders of magnitude greater than 10^{-6}M serum iodide proposed by Wolff and Chaikoff to cause inhibition of organification of iodide by the thyroid gland. Yet, thyroidologists refer to these in vitro studies to confirm the Wolff-Chaikoff Effect. They must think we are really stupid. Daily intake of 50 gm (50,000,000 micrograms) iodide would be required to achieve these peripheral levels of 10^{-2}M in the adult human subject (4), a heroic amount by any standard.

In the Eighth Edition of "The Thyroid", Meier and Burger (26) called iodine a contaminant that interferes with the destructive effect of goitrogens. Obviously, thyroidologists hate the thyroid gland.

> "There is a marked competition between iodide and the thionamides for the active site of TPO. … In situations of severe <u>iodine contamination</u>, these are the two major mechanisms leading to the loss of efficiency of these drugs. It is also likely that <u>iodine contamination</u> reduces the capacity of the thyroid to concentrate the thionamides."

However, in the Ninth Edition, they were kinder and gentler to iodine – they stopped calling iodine a contaminant (27). They just wrote that it is "excess" iodine that is the problem. The amount of daily intake of iodine that protects the thyroid gland from the harmful effects of iodine inhibitors is called by these thyroidologists "severe iodine excess". They have gone berserk!

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Keep in mind that these drugs block the uptake of iodide not only by the thyroid gland but also by every target organ of the human body. Why would anyone in his/her right mind want to concentrate iodine-blocking agents in the thyroid gland and the rest of the body because of iodine-deficiency induced hyperthyroidism? These patients need more iodine, not iodine blocking agents.

Thyroidologists have become so destructive that some of them recommend radioiodine ablation of the thyroid to allow the reintroduction of the toxic organic iodine containing drug amiodarone in patients with a prior history of amiodarone-induced thyrotoxicosis. To quote Hormida et al (28):

> "…However, hypothyroidism should be viewed as a goal, rather than a complication of treatment in these patients".

Farwell et al (29) recommend "near total thyroidectomy" in cases of "resistant amiodarone-induced thyrotoxicosis:

> "…we suggest that near-total thyroidectomy warrants considerations as definitive treatment for resistant amiodarone-induced thyrotoxicosis."

How come cardiologists never considered inorganic non-radioactive iodine as first line of therapy in cardiac arrhythmias instead of the toxic sustained release iodine drug, amiodarone?? A careful review of published data on amiodarone suggests that this organic iodine containing drug is a sustained release form of iodine. The iodine released is the active agent with the drug itself being the cause of its toxicity (30). Inorganic non radioactive iodine is the treatment of choice in those clinical conditions currently treated with amiodarone.

In their 2001 publication, Martino et al (31) reported a list of side effects and complications of amiodarone: corneal microdeposits = 100% of the cases; anorexia, nausea = 80%; skin photosensitivity and discoloration = 55-75%; neurological symptoms = 48%; abnormal liver tests = 25%; thyroid dysfunction = 14-18%; lung dysfunction = 10-13%. The pulmonary toxicity is the most serious complication of amiodarone therapy, with a fatal outcome in 9% of the patients experiencing this side effect of amiodarone (32).

It is hard to believe that such a drug is widely used by U.S. physicians in medical conditions where inorganic non-radioactive iodine has never been tested. Connolly (33) in his 1999 review of amiodarone efficacy and safety reported:

> "On the basis of the number of prescriptions filled in retail pharmacies, amiodarone was the most often prescribed antiarrhythmic agent, account for 24.1% of the total antiarrhythmic prescriptions in 1998."

He further commented that amiodarone accounted for 33 to 74% of prescriptions in Europe, North and South America, compared to 0.3% in Japan, which is 100 times less than the other countries mentioned. It is of interest that mainland Japanese consume at least 100 times the RDA for iodine (9,34). That is at least 100 times more iodine than countries with 100 times more prescriptions for amiodarone. Regarding the evidence based analysis of amiodarone efficacy and safety, Connolly stated:

> "The general view that amiodarone is the most useful drug for VT and VF, notwithstanding the rather modest evidence from randomized trials, led to its being adopted as the standard medical therapy in several recent randomized secondary prevention trials evaluating the ICD. … A meta-analysis of these trials based on individual patient data yielded a relative risk reduction in all-case mortality of 13% to 15%, which was of borderline statistical significance (P=0.03 or 0.06 depending on analytical method used)."

When endocrinologists from India reported the presence of biologically active sodium/iodide symporter (NIS) in breast tissue from women with intraductal carcinoma (35), they totally ignored the obvious implications for the therapeutic use of inorganic non-radioactive iodine in patients with breast cancer. They showed their preference for the systemic use of radioiodide. This form of therapy would expose every organ of the body to the carcinogenic and cytotoxic radioiodide. They have gone berserk!

> "The unequivocal demonstration of NIS expression, its functionality and retention of iodine by organification further provides supportive evidence for use of radioiodine as an additional treatment modality of human breast carcinoma." (35)

After 60 years in the Dark Ages, following the second wave of medical iodophobia, inaugurated by the Wolff-Chaikoff Iodophobic Effect (11,12), iodine is emerging recently as an important nutrient for protection against breast cancer and the degenerative diseases of the Western World (4,6,8,9,36-45). For the first time, a simple loading test became available to assess whole body sufficiency for iodine (4). For the first time, a simple test became available to asses the efficiency of cellular iodide uptake system using the saliva/serum stable iodide ratio (44). For the first time, the detoxifying effect of iodine at 50 mg/day on the toxic halides fluoride and bromide was reported (40). For the first time, evidence for an enterohepatic circulation of inorganic iodine was presented (41).

For the first time, a mechanism used by the human body to prevent iodine overload was reported (4,38): In cases of whole body deficiency, the ingested iodine/iodide is retained by the body in proportion to the degree of deficiency. At sufficiency, the amount of iodine absorbed is quantitatively excreted in the urine as iodide, therefore protecting the body against iodine overload. In the adult, 1500 mg of iodine was retained at sufficiency (41), an amount 50 times higher than the amount of total body iodine reported in medical textbooks. We have confirmed (38) the observation of our medical predecessors (46) that iodine detoxifies the body from the heavy metals, lead and mercury.

For the first time, evidence that the administration of Vitamin C improves a defective cellular transport system for iodine was reported (39). So far, every case of iodine transport inefficiency we had studied, has responded to a complete nutritional program, including several grams of Vitamin C. Iodine alone in daily amounts of 50 mg or more is also effective in cases of iodide symport inefficiency.

The iodine/iodide loading test to assess whole body sufficiency for iodine becomes more accurate by implementing a complete nutritional program for one month prior to the loading test. In cases of iodine transport inefficiency, the high urinary excretion of iodide would give the false impression of iodine sufficiency (38,39). By correcting this inefficiency of the iodine transport system through nutritional intervention (38,39), prior to performing the loading test, this test becomes more accurate. The loading test is not reliable in patients on antithyroid drugs which inhibit oxidation and organification of symported iodide in the target cells. This results in a high urinary excretion of iodide, giving the false impression of whole body sufficiency.

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