BEDSIDE MEDICINE FOR BEDSIDE DOCTORS

An Open Forum for brief discussions of the workaday problems of the bedside doctor. Suggestions of subjects for discussions invited.

HOW SHOULD IODIN BE USED IN THE TREATMENT OF DISEASES OF THE THYROID GLAND

H. H. Searls, M. D. (University of California Medical School, San Francisco).—*Etiology and Diagnosis.* Iodin plays a very important rôle in the physiology of the thyroid gland. Its chief source for human needs has been through the presence of soluble iodids in drinking water. Recently the popular and rational use of iodized salt has offered additional iodin for the requirements of the body. The endemic foci of goiter throughout the world, with few exceptions, are associated with drinking water of extreme purity. Rarely, however, a factor of intestinal infection of a specific nature (as described by McCarrison in his many reports), appears to be associated definitely with the development of goiter.

As our drinking water proceeds from precipitation toward consumption, it leaches from the soil soluble salts including iodids. Many factors influence the amount of such salts which, in this natural manner, may be added to our water supply. Glaciated regions notably are free from soluble salts, these having been washed out by the large amounts of water resulting from the melting of the glaciers. The regions in which endemic goiter is found in the United States and Canada closely correspond to the area covered by the great glaciers of the Pleistocene period. In areas where precipitation is heavy, there is obviously a greater dilution of soluble salts in the drinking water. Transportation of drinking water by wooden, metal or concrete flumes and pipelines prevents its natural accumulation of soluble salts. In many of our large cities today, water thus travels from precipitation to consumption without opportunity for the accumulation of iodin. The addition of iodin in the proximal reservoirs then becomes a rational measure for public health. At present, for example, a chemical study of San Francisco's new water supply is being conducted. If the iodin content is found to be deficient, it is planned to add an adequate amount.

Endemic goiter is found in northern California in a horseshoe-shaped region extending northward along the Coast Range from Monterey to the northern boundary of the state, then eastward through the Siskiyou mountains and southward over the northern half of the Sierra Nevada. Pure drinking water is found throughout these regions, and the addition of iodin to the drinking water, or the oral administration of iodin to school children, undoubtedly would reduce the incidence of endemic goiter.

Endemic types of goiter are (*a*) the colloid and (*b*) the adenomatous. Adolescent goiter approaches the colloid type.

The colloid goiter is a diffuse symmetrical enlargement of the thyroid gland without toxicity, often associated with hypothyroidism. The gland is firm and granular, and varies from moderate to massive size. Although occasionally there is an uncomfortable sense of constriction of the windpipe, the usual complaint is that of disfigurement. Rarely, the administration of iodin results in the return of the gland to normal size. Far better results are obtained by the use of thyroid substance.

The adenomatous goiter also is encountered commonly in endemic regions. In this case the enlargement of the thyroid is caused by the development and growth of encapsulated nodules (adenomata) within the glandular substance. Starting insidiously, they gradually exert a toxic influence on the organism, manifested by tachycardia, tremor, increased nervousness, and emotionalism. Iodin (contraindicated in the nontoxic adenoma because of its tendency to stimulate the development of toxicity), is of little value in the treatment of toxic adenoma.

In the diffuse, symmetrical, toxic goiter of the hyperplastic (exophthalmic) type, iodin is of great value in the preparation of the patient for surgery. Diagnosis is made, in this condition, from the story of a rapidly developing toxemia evidenced by extreme nervousness, increasing exophthalmos, tremor, feeling of warmth, tachycardia, loss of weight, increased appetite, and hyperhidrosis; and by the presence of a diffuse, smooth, firm, symmetrical goiter, often exhibiting thrill and bruit. Blood pressure readings disclose a high pulse pressure. The basal metabolic rate usually is elevated quite markedly.

Frequently the patient with exophthalmic goiter receives iodin over a long period of time. In such instances, there is improvement for a period of several weeks, and then the patient returns to a condition similar to that found before Lugol's solution was administered. Such "iodin fast" patients have lost their best opportunity for cure by surgery. Operative risk is increased, and operation should be performed only with great skill and celerity. The postoperative course of these patients usually is associated with severe crises of hyperthyroidism.

It is evident, therefore, that, in exophthalmic goiter, iodin should be reserved for preoperative administration, and for a short period following surgical treatment.

In the rational use of iodin for the treatment of goiter, it obviously is required that the attending physician be able to differentiate between the different types, as treatment varies with the pathologic picture. Of particular importance is the differential diagnosis between toxic adenoma and

exophthalmic goiter. The following points may aid in the recognition of the type encountered:

HYPERPLASIA	ADENOMA
1. Exophthalmos (about 70 per cent of patients).	1. No exophthalmos.
2. Thrill and bruit over thyroid.	2. No thrill or bruit.
3. Symmetric goiter of uniform consistency.	3. Asymmetric nodular goiter.
4. Parallel development of goiter and toxicity.	4. Goiter of years' duration before toxicity develops.
5. Rapid development of toxicity to advanced degree, with periods of remission.	5. Slow progressive development to a moderate degree, without remissions.
6. Etiology—? psychic trauma; ? oral infection.	6. Etiology—endemic.
7. Typical blood pressure, 140/60.	7. Typical blood pressure, 160/100.
8. B. M. R. ranges from 40 to 70 per cent plus.	8. B. M. R., from 25 to 50 per cent plus.
9. High percentage of lymphocytes.	9. Normal blood picture.
10. Rarely substernal or intrathoracic.	10. Most substernal and intrathoracic goiters are adenomatous.
11. Rarely any pressure signs or symptoms.	11. Commonly pressure is a main complaint.

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CLARENCE G. TOLAND, M. D. (1930 Wilshire Boulevard, Los Angeles).—In 1200 A. D., Roger of Palermo advocated burnt sponge in the treatment of goiter. Coindet, the Swiss physician, established iodin as the effective agent, in 1829. Following that time, for nearly one hundred years, iodin was alternately praised and condemned. Trousseau, in a brilliant therapeutic mistake, discovered its value in toxic goiter. Kocher, with his imposing influence, condemned it, and its use was abandoned.

Plummer, again in 1922, by an incontrovertible array of clinical tests forced the recognition of its value on a partly believing, partly skeptical medical world. Eight years ago a distinguished doctor, in *The Journal of the American Medical Association,* said iodin was "a double-edged sword, with its sharpest edge menacingly turned toward the patient." In the last few years, however, we have come to learn that it is a shield protecting the patient from the fury of Graves's disease.

We do not understand how iodin works, but we do know what it does. Why iodin, on the one hand, should prevent the accumulation and storage of colloid in a patient with endemic goiter and, on the other hand, promote such storage of colloid and formation or normal thyroxin in the patient with Graves's disease, seems contradictory. A consideration of the normal iodin metabolism is essential to an understanding of how iodin operates.

An infinitesimal daily dose of iodin, .16 milligram or about 1/400 of a grain, is all that is required to keep the body in iodin equilibrium. In Graves's disease the usual iodin dosage has been about 100 to 400 milligrams per day in the form of Lugol's solution. We are thus giving 600 to 2600 times the physiologic requirement of the body. Thompson, in Boston, has found that one minim of Lugol's solution, or 6 milligrams daily, will effect a maximum reduction of basal metabolism in Graves's disease. This is an interesting fact, but we feel that it is neither advisable nor safe, as an excess of iodin is not dangerous. We always give about 15 to 20 minims, which we feel is a safer procedure.

The maximum improvement of patients with Graves's disease, previously untreated with iodin, occurs in about eight to fourteen days. The rate of improvement depends chiefly upon the intensity of the intoxication, and also the manner in which the disease was progressing spontaneously before the iodin was started. Occasionally a patient may not respond, or may become worse. These are the cases illustrating to us the fact that the story of iodin effect has not yet been written.

We use iodin in both patients with Graves's disease and those with toxic nodular goiters, and have encountered very few cases of so-called iodin hyperthyroidism. It should be a rule not to employ iodin in patients with nontoxic adenomas, as there is the risk of inducing iodin hyperthyroidism.

We decry the prolonged use of iodin for toxic goiter, save for a very rare type of mild hyperthyroidism in the young adult, where it may be used in small doses with careful observation.

The moderately and severely toxic patients, receiving prolonged iodin treatment, will eventually require surgery, will be poorer surgical risks, and will account for a decidedly higher mortality.

The indications for the use of iodin other than for the preoperative preparation of the patient with toxic goiter are few. About one in two hundred patients following thyroidectomy will develop some postoperative hyperplasia, with varying degrees of return of toxic symptoms. A certain percentage of these may be kept under control by the intermittent administration of iodin.

In hyperthyroid crises following subtotal thyroidectomy, we routinely give sodium iodid by vein, and feel that it is of value in certain cases. The blood iodin is low, following subtotal thyroidectomy, and it may operate by supplying that deficiency; but we do not believe that it has any specificity in the treatment of crisis. It is difficult to evaluate the efficacy of any procedure here, as many therapeutic measures are usually resorted to in such a crisis.

The use of iodin for adolescent goiter has an established place, and is definitely effective.

In all these types where iodin is used, we must remember that we are not treating the cause of goiter by giving iodin, and that by any type of indirect therapy we always shall beget some failures. But until the etiology has been definitely established, we must be grateful for such a powerful ally as iodin in the treatment of goiter.

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A. B. COOKE, M. D. (504 Medico-Dental Building, Los Angeles).—It sometimes happens that discussion of the abuse of a remedy or measure becomes fully as important as discussion of its use. I am persuaded that this is true in the present instance. Probably nowhere in the whole range of clinical medicine can be found a more conspicuous example of the association of good and evil possibilities in a single medicament, than in the case of iodin in the treatment of diseases of the thyroid gland.

Others have presented the benefits of iodin. The present writer accepts the much less inviting

part of emphasizing its possibilities of harm, and sounding an earnest note of caution.

Somewhere in his numerous contributions to the study of the goiter problem, Crile poetically says that the "thyroid gland is the musical instrument upon which nature plays the tune of life," or words to that effect. In similar fanciful vein it may be said that iodin determines the key and pitch of the tune, whether it shall be one of enchanting harmony or one of discordant jazz.

That iodin has a place, and a most important place, in the therapy of goiter, is a firmly established fact. But we shut our eyes to an equally important fact when we fail to realize that its indiscriminate use constitutes a distinct and widespread menace. In reality it is a two-edged sword, as potent for harm as for good. To employ the agent merely because there is a goiter, is often to woefully misuse a valuable remedy, with serious and not infrequently disastrous consequences to a confiding patient.

And yet this is exactly what occurs so constantly that it may almost be regarded as established routine. I see few cases of any type in which iodin, in some form, has not already been resorted to by the patient; either upon misinformation acquired from the public press or some well-meaning friend, or, sad to say, often from the profession itself.

Surely the members of the medical profession are not willing to acquiesce in the imputation of carelessness or lack of ability to discriminate. The indications for the use of iodin in goiter therapy are definite and easily understood. It can be considered curative only in certain cases of the simple or nontoxic variety. In toxic goiter it can never be considered curative. True, toxic hyperplastic goiter usually responds magically to the proper exhibition of the remedy, but the response is always temporary. If the iodin is persisted in, the patient will very probably become immune to it, with subsequent return of the toxicity.

In toxic adenoma iodin is to be employed with great caution, if at all. There can be no question that it is capable of much harm in these cases.

Therefore in toxic goiter—since iodin is not curative in any type of case, and since its beneficial action at best is only temporary—it follows that the sole purpose in administering it should be to control symptoms and to prepare the patient for safe surgery.

In colloid goiter there is already a superabundance of iodin stored in the gland. To administer more would seem to be adding insult to injury. When for any reason nonsurgical treatment is adopted in this type of case, far more favorable results may be expected from thyroid extract than from any form of iodin. In the absence of toxicity the disfigurement is the only indication for intervention, and here surgery certainly offers more than any kind of medicinal treatment.

One other abuse calls for protest. Many erroneous ideas are prevalent in regard to "iodized salt." Lay publications and billboards are utilized by sundry commercial interests to promote the consumption of this so-called table necessity. A moment's reflection should make it clear that the claims made are both false and pernicious. Iodized salt provides an easy means of increasing the iodin intake; but rightly conceived, the only indication for its use is the prevention of endemic goiter in districts where there is a deficiency of iodin in the diet. And it is agreed by those in position to know that the efficacy of the agent is limited to the prepuberty years.

When iodized salt replaces ordinary salt as a condiment, adults as well as children partake of it. The possibility of harmful results is apparent. There can be no doubt that in latent cases of nontoxic adenoma active symptoms are often excited. In toxic adenoma it may stimulate to increased toxicity, and in hyperplastic cases the dosage is wholly insufficient to control the symptoms.

All this would seem to be obvious in the mere statement. Yet a half-page advertisement appearing in current issues of *The Journal of the American Medical Association* contains the remarkable statements, quoted from the report of the Committee on Foods: " . . . Iodized salt is for all table and cooking uses of salt," . . . "Used daily as the only salt on the table and in cooking," . . . !!

The profession of California is to be congratulated that CALIFORNIA AND WESTERN MEDICINE has not succumbed to this insidious form of questionable commercialism.

Value of Calcium in Preoperative Preparation.—Evans presents a preliminary report on the routine use of calcium in preoperative preparation. Each adult patient is instructed to eat a sufficient amount of plain food (meat once a day, not fried), to drink at least one glass of milk with each meal, six glasses of orange juice daily, two and three ounces of stick candy daily, to take deep breathing exercises at least three times each day, to see that the bowels move every twenty-four hours, and to sleep at least nine hours each night. On the morning of the third day before operation, each adult patient receives a sufficient amount of calcium so that there will be a slight hypercalcemia at the time of operation: three grams of calcium gluconate by mouth immediately after each meal. On the night before operation, 10 cubic centimeters of a sterile 10 per cent solution of calcium gluconate is given intramuscularly and a sufficient amount of alkali so that the urine will be slightly alkaline on the morning of operation. A plain soapsuds enema is given on the evening before operation, and at bedtime 0.1 gram of phenobarbital is given by mouth. Three-quarters of an hour before the operation each patient receives from 0.008 to 0.016 gram of morphin sulphate intramuscularly. After the operation, a retention enema of from 500 to 800 cubic centimeters of tap water is given. As soon as the patient begins to react from the anesthesia, 10 cubic centimeters of a 10 per cent sterile solution of calcium gluconate is given intramuscularly and the patient is placed in a slight Fowler position. No abdominal binders or tight abdominal dressings are used. Fluids, if desired, are given immediately after operation. The patient is turned on the side every three or four hours, and during the first four or six days is made to take a series of deep breaths three or four times a day. During the first three or five days, especially in upper abdominal operations, a sufficient amount of morphin sulphate is given intramuscularly to prevent any sharp pain, restlessness or fear. A small rectal tube is used if there is any distention. Enemas or cathartics are given on the third or fourth day after operation. Patients are catheterized if they do not void within twelve hours after the operation, and every eight hours until they void voluntarily. Dressings are not changed except to remove drains and sutures.—*N. J. Medical Society Journal.*