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Treatment of Graves' disease

The thyroid gland acts as our "thermostat." It makes hormones that set our metabolic rate – the process of making and using energy. People with Graves' disease who have high thyroid levels and a high metabolism need treatment.

In Graves' disease, the immune system interferes with the proper regulation of thyroid hormone output. An abnormal immune globulin inappropriately drives the gland to produce thyroid hormone. Because the normal control of the thyroid gland is thus bypassed, high thyroid hormone (TH) levels – called hyperthyroidism or thyrotoxicosis – can result.

Oddly enough, patients with early Graves' and moderately high TH may have no symptoms or even feel low-thyroid: their body adapts to block the effects of excessive hormone. This "defense" fails if TH gets too high, resulting in the typical "hyper" symptoms of hyperthyroidism. High levels of TH damage the body – all cells can be hurt by hyperthyroidism.

The course of Graves' disease is unpredictable: Thirty-40% of people simply get over it; a spontaneous remission in Doc-talk. If the disease continues, other antibodies (TPO-Ab and Tg-Ab) ultimately destroy the gland. Before it "burns out" to a low-thyroid state, though, it can truly race. Really high levels cause "thyroid storm," which may be deadly.

The foremost goal of treating Graves' disease is to protect the patient from the damaging effects of high thyroid hormone. Secondly, normal hormone levels and balance should be restored in a safe, efficient manner. Of course, we want to protect our patients from other risks and complications of the disease itself and against the possibility of a later relapse.

The first goal is achieved with a prescription: Beta-blockers are given to patients with a runaway rapid metabolism and fast pulse. These drugs don't lower thyroid hormone levels; they protect the heart, blood vessels and other cells from the excessive stimulation caused by high TH. The beta-blocker can be stopped once normal TH levels are achieved.

Anti-thyroid drugs help achieve the second goal. Propylthiouracil (PTU) and methimazole block the enzyme that assembles thyroid hormones; thus, less TH is made. In prescription strength, lithium can also block thyroid hormone production, though less satisfactorily and with greater risk of side-effects (including *worse* hyperthyroidism).

At this point, European and American treatment strategies differ. Europeans continue the anti-thyroid drug for 18-24 months; they then stop it, as some 30-40% of patients with Graves' will have gone into remission. If so, the relieved patient is monitored with regular thyroid tests, as relapse can occur. If the disease returns, the gland will be destroyed.

Americans have arrived at a different solution. Most doctors in the U.S. don't give the gland a second chance, as 60-70% of Graves' cases promptly recur after the drugs are withdrawn – and because the disease is indeed prone to later relapses. Once the diagnosis is certain and anti-thyroid drugs have taken good effect, the gland will be destroyed.

An overactive thyroid avidly takes up iodine. A "deadly" radioactive-iodine isotope (131*) is given to kill the thyroid. The dose must be enough – but not too much. Iodine is also taken up by the breasts, tear- and salivary glands, so side-effects can include dry eyes, dry mouth and increased incidence of breast cancer. Women may also have reduced fertility.

Surgical removal of the diseased gland is effective but leaves a scar. Complications are uncommon, including hoarseness and unplanned loss of the parathyroid glands. On very rare occasion, a person's voice can be irreparably harmed.

Some patients don't want to be permanently hypothyroid and take TH replacement the rest of their lives. Though Graves' disease has no "cure," less destructive methods may be used for treatment. Anti-thyroid drugs can be continued as long as no side-effects arise. An aggressive method called "block and replace" can be effective but is "high-maintenance." Low-dose Naltrexone seems useful for some immune-related diseases but clinical experience with the thyroid is limited.

Each patient with confirmed Graves' disease must decide on the treatment that suits her or him best as an individual. We must understand the disease and its treatment options – with their pros and cons – to give informed consent. This paper is respectfully offered to help our patients arrive at a wise, personal decision.