A Clinical Update on Diagnosing and Treating the Underactive Thyroid

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Preamble:

How this came about

When I was studying a manual-medicine profession at university, I couldn't see why we had to learn about biochemistry. However, being in practice for some years, and particularly, being in a situation where I see a lot of people who have tried everyone else first, has taught me that I need to understand far more about the subject than I do. It has become increasingly clear that a lot of the conditions I see in my clinic have a biochemical issue as at least one of the underlying causes. In particular, by talking to and learning from some incredibly knowledgeable people, I've come to realise that thyroid problems are widespread, and significant in musculoskeletal medicine (and most other kinds of medicine, for that matter).

These conditions are complex to diagnose, but generally straightforward to treat once they are understood. There is a lot of research available, and a number of books, web sites and webinars on the subject. Unfortunately many of these seem to be a combination of brilliant science and unsubstantiated folklore.

Incredibly, we haven't been able to find a single source that brings all of the relevant, current scientific information together in a clinically useful way. So this is an attempt to do something about that. Belinda is a patient, and a gifted amateur who has taught herself (and me) an enormous amount about health and nutrition. She has helped from both a research and an editing point of view.

How you can help

Let's be quite clear; this document is wrong in many places. Probably. It is just the best we could come up with at this point in time.

The science around the thyroid is moving faster than text books can keep up, which is one of the reasons we went back to original research papers to develop a better picture. Precisely because of that, we may not have found everything that is important, and at least some of the understanding around this topic is likely to change very quickly. Any corrections or updates you are able to supply will therefore be greatly appreciated.

Please pass this document on to any of your colleagues who might find it helpful; and please assist in two other ways.

- First, please keep a record of whom you have passed it to, so that corrections can be sent out and the information can be kept current. (You don't have to send me the details of who it has been passed to; I will send any corrections to the people I originally sent this to. Please do the same yourself.)
- Second, if you find any errors, omissions, or you have some updates for this information, please get back to me with them (info@smarttosteopath.com) so that I can check them out and issue an update.

Introduction

It may be interesting to think about the number of people you saw last week with at least one of these conditions:

- Tiredness (particularly finding it hard to get going in the morning, and only really waking up in the evening);
- Lack of "sparkles"; they feel that they are going through the motions, but it's a long time since they've "heard the music of life";
- Mood swings, with periods of high energy (and possibly anxiety) interspersed with periods of fatigue;
- Irritability which surprises them;
- Depression, mild or severe, sometimes with agitation and/or paranoia;
- Tenderness in their body;
- Hypertonic muscles;
- Difficulty losing weight;
- Easy bruising;
- Swelling at the anterior base of the neck;
- Headaches;
- Irregular menstrual cycle, in a woman below 45 years old;
- Diminished libido in either sex;
- Decreased fertility in either sex;
- Partial or complete impotence in men;
- Multiple miscarriages;
- Tender, non-cancerous lumps in the breasts;
- Frontal alopecia;
- Decreased night vision;
- Numbness or tingling in the extremities;
- Mild hearing loss;
- Thinning of the outer 1/3rd of the eyebrows;
- Cold hands and feet;
- Dry skin;
- Fluid retention;
- Constipation or sluggish digestion;
- An irritable bowel (often through a combination of reduced stomach acid making long-chain protein digestion difficult, damaged intestinal mucosa, decreased motility

Surprising facts about the thyroid.

- *It is linked to cholesterol, cardio-vascular health, and bone-mineral density. In fact, a poor thyroid function may put a person at greater risk of a heart attack than high cholesterol (which it may also be causing), high blood pressure, smoking or diabetes.*
- *It doesn't just cause mild depression; sometimes the depression can be severe, and can be associated with paranoia and psychosis.*
- *There appears to be a link with Alzheimers, although it isn't a simple cause-and-effect connection. But there is certainly also a dementia caused by thyroid deficiency that is not Alzheimers.*
- *Hashimoto's thyroiditis (associated with an underactive thyroid) may involve sudden releases of thyroid hormone from a damaged thyroid gland; particularly in the early stages. It can therefore present similarly with bipolar disorder, and even has a hyperthyroid (overactive thyroid) condition. It can get worse over time if it isn't treated properly.*

The good news? You can usually fix a broken thyroid mechanism, without needing a lifetime of medication.

and intestinal fluid retention (which can increase abdominal tenderness));
- Mild **proteinuria**;
- A change in the normal diurnal rhythm of **urine production**, so that urination is normal or a little slow during the day, but increased at night;
- Borderline or mild **anemia**;
- Forgetting things and the **brain seems foggy**;
- **Dementia** in the elderly;
- Just **feeling unwell**, but can't put their finger on why;
- Feeling **unable to cope**;
- **Calcification** of the blood vessels and/or connective tissues;
- High **cholesterol**;
- High **triglycerides**; or
- Low **bone-mineral density**.

If you often treat older people, at least one of these conditions probably applies to most of them. These conditions can be very common, and can also be frustratingly difficult to treat.

There are, of course, multiple potential causes for every one of them. Some would say that they are an inevitable part of aging, but there are enough counter examples to indicate that this isn't the case; they are just incredibly common, and become more common with aging. To compound the diagnostic challenge, an underactive thyroid doesn't usually present with all of these signs and symptoms, and may, in fact, occur with none of them.

If a patient presents with any of these symptoms, though, it is standard practice to include thyroid dysfunction in the differential diagnoses, and to use blood tests for thyroid stimulating hormone (TSH) and thyroxine (T4) to rule it in or out.

If these tests come back saying "consistent with euthyroid" the practitioner probably assumes that the thyroid isn't the problem. Unfortunately, it still could be. These patients may be told to:

- diet more,
- exercise more,
- take a statin for cholesterol for the rest of their lives[*],
- take medication for osteoporosis,
- use a laxative,
- use a diuretic,
- see a psychologist,
- have a holiday,
- be kinder to themselves,
- take an anti-depressant[**], or
- just get used to feeling the way they feel, and accept that they need to expect this as they get older.

If you are a manual practitioner, these people will probably come to you complaining that they have already seen their doctor, who hasn't been able to help them. You probably expect to be treating these people for a long time, because what you do will help them to feel temporarily a bit better. Their whole system will be tender, and so what would be mild

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[*] One of the many potential side effects of statins is increased depression. One wonders how much of the depression seen in nursing homes is a result of undiagnosed thyroid deficiency, compounded by statin side effects.

[**] Unfortunately some antidepressants can, themselves, cause symptoms very similar to thyroid dysfunction. This is, at least in part, because they can have an impact on the thyroid metabolism.

musculo-skeletal discomfort that other people would ignore, for these people is often pain that they feel they need to get treated.

But if the underlying problem is their thyroid (and there is a decent chance than it is) then what you do will only be temporary. They will be back again soon (if they can't find anyone they like more) with the same or a different problem.

Thyroid Physiology

Thyroid hormone is the only hormone*** with receptors in every cell in the body. Problems with the thyroid mechanism can quite literally impact every system. As well as increasing the metabolic rate in every cell, it has a wide range of lesser-known functions. For example:

- It facilitates the formation of myelin in the brain, and enables growth hormone to work.
- It affects nerve growth factor, insulin-like growth factor, inhibin and sex-hormone binding globulin.
- It increases the activity of sodium-potassium pumps.
- It not only increases the production of ATP within a given mitochondrion, but it also increases the number of mitochondria.
- It stimulates the absorption of glucose in the intestines, stimulates hepatic glycogenolysis, stimulates insulin break down, and facilitates the glycogenolytic actions of adrenaline.
- It has a direct action in breaking down fats, and an indirect action by facilitating the fat-breakdown activities of glucocorticoids, glucangon, growth hormone and adrenaline. It also increases the oxidation of free fatty acids.
- It decreases cholesterol, not just by increasing the metabolic rate, but also by stimulating bile acid formation in the liver, which results in excretion of cholesterol derivatives.

An indication of the importance of the thyroid can be gathered by the fact that it receives a larger blood flow that the kidneys[1].

It is estimated that between 3% and 5% of people in the developed world have a thyroid deficiency[2] In the developing world, largely through low levels of iodine, the numbers are thought to be considerably higher; a billion people may be affected. These numbers are based on people with problems with the thyroid hormones that are normally assessed in blood tests.

However, the thyroid mechanism is a complex biochemical cascade, and the normal thyroid blood tests only measure the top of the cascade. The problem is likely to be more widespread than is commonly recognised.

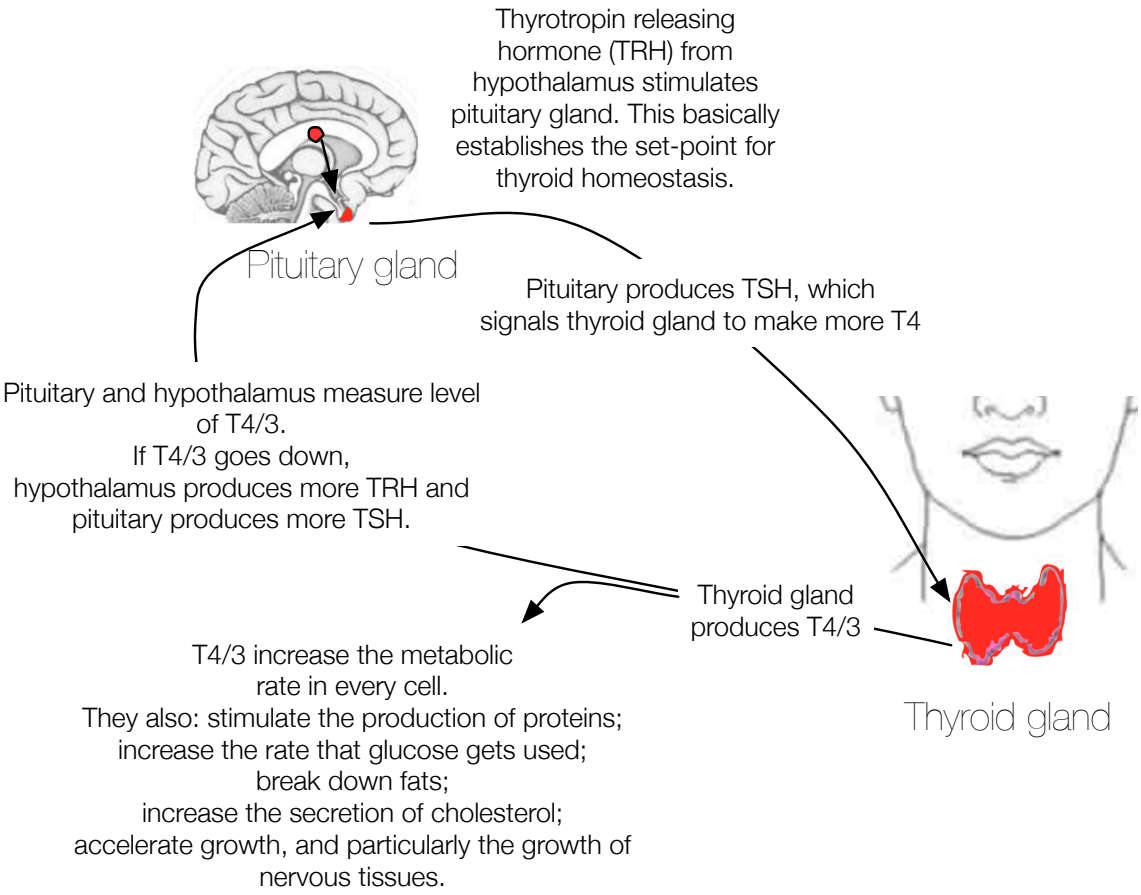
What you probably learned when you were studying

When you were learning your profession, you were probably taught that thyroid hormone - thyroxine, or T4 - drives the metabolic rate. There is a feedback mechanism with the anterior pituitary, so that when T4 goes down, TSH goes up. You would have learned that if the thyroid has an auto-immune inflammation, it might make T4 levels go up (Graves disease) or down (Hashimoto's thyroiditis).

T4 can also go down for "idiopathic" reasons. If TSH goes up, it means T4 is down. If T4 is down, the patient should be given more T4.

*** Unless Vitamin D is classed as a hormone, which it probably should be.

What you were probably taught about the thyroid



That is all reasonably complicated, but you probably had to learn even more than that.

If you are a doctor, you had to learn about thyroid hormone dosage recommendations. You had to learn what drugs, in what dosages, to give for an overactive thyroid; and you had to learn that standard anti-inflammatory medication isn't the treatment of choice for autoimmune thryoiditis (although it is the treatment of choice for viral sub-acute thyroiditis).

You also probably learned that autoimmunity is genetic, and that the best you could really do for it was to treat the specific inflammation with pharmacology.

You had to learn about thyrotoxicosis, thyroid cancers, benign thyroid nodules and tumours, pituitary tumours, sub-acute thyroiditis, thyroid surgery, and thyroid radio-ablation. Some of the things associated with the thyroid (the treatments as well as the disease) can be life-threatening. And if you did a lot of your training in a hospital, it would have been the more immediately-life-threatening conditions that you saw.

What you were taught (mostly) isn't wrong; it's just a potentially dangerous oversimplification. This results in two problems. The first is the under-diagnosis and under-treatment of a lot of thyroid conditions. The second problem is that, if T4 is low and TSH is high, the treatment is usually ongoing administration of thyroxine sodium (Oroxine® in Australia). The assumption tends to be that this is "primary hypothyroidism", and that the patient will therefore need to be on medication indefinitely. With more thorough diagnosis this is often not the case.

A more thorough explanation

Thyroxin is essentially the amino acid tyrosine with four molecules of iodine attached to two carbon rings attached to it (which is what the "4" stands for).

Technically, thyroxine probably isn't a hormone at all; it's a "pro hormone"[3]. This means that it is stable, but it doesn't do much (although there is some recent information that it may have an active role to play in a specific process[4]). The fact that it is stable makes it good for measuring in a blood test; but not good for much else - until it is converted.

Healthy people keep large reserves of it (weeks to months of supply) in their system. It is formed within large protein molecules called thyroglobulins, which exist entirely within the thyroid, and are not to be confused with thyroxine-binding globulins. Thyroxine-binding globulins are mainly produced in the liver.

In order to become biologically useful, it needs to be freed from its binding proteins and lose a molecule of iodine (become "deiodinated") to form 3,5,3′ triiodothyronine (T3).

Free T3 gets used very quickly, which means that firstly, giving supplemental T3 on its own gives people unpleasant energy fluctuations, and secondly that T3 blood tests aren't considered as helpful as those for TSH and T4, which is why these two latter tests are used more routinely[5].

About 80% of the deiodination of T4 to T3 happens outside of the thyroid gland; largely in the liver, but also in the kidneys, the central nervous system, brown adipose (fat) tissue, and the skin[6]. It can even happen within the final target cell.

T4 and T3 are fat-soluble. As such, it used to be believed that they entered cells though simple diffusion across the phospho-lipid membrane. They are, in fact, theoretically capable of doing this, but the actual process uses specific receptors, and these operate differently in different types of cells.

There are also specific carriers that transport thyroid hormones across the blood-brain barrier. There are some rare, severe mutations that prevent these transporters from working well, but, at this stage (and despite what several authorities maintain) we have not been able to identify any factors that limit this activity in the majority of the population[7]. In other words, thyroid hormones seem to move well and easily across cell membranes.

Once T3 has entered target cells it needs to couple with receptors in order to achieve anything.

These processes are all complex, and our understanding of the biochemistry involved is still emerging. What is clearly understood, though, is that there is a lot that can go wrong with most of them.

The causes of the things that can go wrong can be summed up under five headings: nutritional deficiencies, toxic interference, stress responses, autoimmunity and liver dysfunction.

Autoimmunity is, itself, increasingly recognised as being caused by a combination of genetic susceptibility, toxic interference, and an inappropriate systemic response to pathogens at some point in time.

When there is insufficient production of T4, this will normally show up in blood tests with decreased T4 and an elevated TSH. That does not mean that administration of thyroxine is automatically the best treatment.

A much bigger problem, however, is that TSH and T4 can be completely normal, and yet

problems with conversion to T3, unbinding from proteins, and receptor coupling can mean that the patient has to live with an undiagnosed, untreated, thyroid malfunction. This can drastically affect their short-term quality of life, and eventually, their longevity.

It would be great if the solution was as simple as giving people a standard range of supplements, putting them onto a standard detoxification/anti-inflammatory diet and helping them to lower their stress levels. That is exactly what is sometimes done by naturopaths, and "integrative" or "functional" doctors. It sometimes works. Unfortunately there are three problems with this one-size-fits-all approach.

- The first is that some of the **important nutritional elements (iodine, selenium and copper) can cause problems for the thyroid metabolism when they are in oversupply, as well as when they are deficient.** Giving everyone the same supplements without thorough diagnosis may be making the problem worse.
- The second is that the toxicity can be quiet specific, and can need a targeted, rather than a general, remedy. For example, a standard "detox diet" lasting for a few weeks will be unlikely to resolve a high level of mercury contamination, which is one of the more common problems I find in thyroid dysfunction.
- The third problem is that the stress that has most impact on the thyroid is often long-term, out of control stress (physical or emotional) in the *past*, rather than a present problem. **People in acute stress normally have too much adrenalin and too much happening to notice their hypothyroid symptoms**; they might come to see you for help to be calmer, but they probably won't notice until later than their metabolism is slowing. These patients can get very frustrated - and stressed - in fact, when they are told that their problem is stress and that they need to reduce it. They have typically been through a great deal of stress. They know what that feels like; they have dealt with it to their own satisfaction, and now the health problems have started, just as their stress levels are falling.

The solutions are, in fact, mostly straightforward. It is usually the diagnosis which is challenging, and which, all too often, is too superficial.

Nutritional considerations

While the evidence is poor (we could only find one study, which related to chickens[8]) it is at least theoretically possible that if there is a problem with protein digestion, the patient might be having trouble getting enough of the amino acid tyrosine. Some "thyroid supplements" contain tyrosine. This probably doesn't do any harm, but it probably doesn't help much, either.

On the other hand, there is exceptionally good evidence going back for many, many years that iodine deficiency can impact on the production of thyroxine. Thyroxine is 64% iodine by weight[9].

Iodine was one of the first trace elements ever discovered; and yet today there are approximately one billion people in the world at risk of iodine deficiency[10]. This is not just a third-world problem; it is causing particular concern in relation to pregnant women[11], because it can have a severe effect on the development of the central nervous system of the foetus[12]. For many years, we've been able to buy iodised salt. But people eat less salt than they used to, and they often eat newer types of salt that aren't iodised. People on strict vegetarian diets seem to be at particular risk from iodine deficiency[13]. "Thus, iodine-deficiency disorders, including endemic goiter and cretinism, are the most common thyroid-related human illnesses, indeed the most common endocrine disorders worldwide"[14].

There is also mounting evidence that iodine deficiency is one of the most important factors

in the development of thyroid cancer[15].

Iodine is a "frenemy" of thyroid production: both too much and too little can cause problems. If patients have too much of it, the Wolff-Chaikoff effect results in a decrease in T4 levels when a large amount of iodine is consumed. This lasts for about 10 days[16]. This is such a significant effect that, until better anti-thyroid drugs were developed, iodine was used as the primary means of control of an overactive thyroid[17].

The body's initial response to iodine deficiency is to convert a higher proportion of the T4 to T3, because that needs 25% less iodine[18]. Free T3 is used very quickly, and so when this happens people are more prone to energy fluctuations, and potentially to symptoms that more closely resemble cyclothymic mood disorder ("bilpolar lite").

Cruciferous vegetables, which are healthy in many ways, can block the uptake of iodine into the thyroid gland. People who are iodine deficient should therefore reduce their intake of cruciferous vegetables until their iodine levels stabilize. (Cooking reduces the amount of goitrogenic compounds; those which inhibit thyroid production; in cruciferous vegetables by about 30%.)

Iodine deficiency is also associated with an increased risk of thyroid cancer, which is common, and becoming more so[19].

Proteins that contain selenium (selenoproteins) are required for the production of T4 in the thyroid gland[20]. Hydrogen peroxide; bleach, effectively; is produced as a temporary bi-product of the production of thyroid hormone and selenium is necessary to convert this to water. When selenium is deficient, the excess hydrogen peroxide can damage the gland, and cause thyroiditis. Selenium deficiency has been linked to auto-immune thyroiditis; Graves disease[21] (hyperthyroidism) as well as Hashimoto's thyroiditis[22].

The conversion of T4 to T3 requires enzymes called "deiodinases"; and these contain selenium. So a selenium deficiency potentially creates a lack of T3 with normal T4 and TSH.

Because selenium is also involved in the initial production of T4, one might expect that T4 would fall when selenium is deficient. But in fact this doesn't happen with a mild deficiency, because there is a hierarchy by which selenium is supplied to different tissues. It is decreased first in muscle, the liver and the kidney, whilst supplies in the brain and the endocrine tissues are preserved for as long as possible[23]. This makes sense in terms of the thyroid, because a selenium deficiency in that gland can result in damage to the gland.) A mild selenium deficiency is therefore likely to result in a hidden thyroid problem with normal TSH and T4 but low T3; a more severe one might show up as auto-immune thyroiditis.

On the other hand, selenium is also a "frenemy" of the thyroid; too much can also cause problems. An excess of selenium appears to block the release of T3 from thyroglobulin. The total T3 may be normal, but the free T3 (which is the small proportion that can enter cells and impact on metabolic rate) may be deficient[24].

Zinc and copper deficiencies also seem to cause low T3 levels, based on studies of low-thyroid populations and the impact of different forms of supplementation, but the biochemistry of relationship is not well understood yet[25].

Copper excess can also cause problems, because an excess of copper can lead to an excess of estrogen, and an excess of estrogen makes it harder for T3 to release from thyroglobulins.

Note that the contraceptive pill can cause a lowering of zinc and selenium levels, and so people on the pill, in particular, should have their levels of these trace elements monitored[26].

Iron, and particularly ferritin, is sometimes cited as a necessary co-factor in the thyroid metabolism. This may be the case, but our reading of the literature suggests that strong correlation between ferritin levels and thyroid function actually works the other way around; T3 appears to be important to ferritin physiology[27] Epidemiological studies, at least, suggest that this may be a cause of anemia which does not respond to iron supplementation[28].

Toxicity

A wide range of substances can interfere with thyroid metabolism.

And if people have a small amount of some toxins in their system; particularly some pesticides, mercury, lead or cadmium (they don't need much; a little bit is extremely common), then it can get in the way of the necessary trace-element co-factors. In other words, for example, a very low level of mercury, which could normally be safely ignored, may be enough to interfere with the normal functioning of selenium in its role as a cofactor in the conversion of T4 to T3, if selenium levels are marginal.

Some toxins can damage thyroid receptors, or there can be competitive binding from halogens, pesticides, or other pollutants[29]. Chlorine can bind to thyroid receptor sites. As far as can be determined, for healthy people, the chlorination of water does not cause a problem, but there are people who are susceptible to chlorine. These people probably feel bad when they smell a chlorinated swimming pool. People like that would be well advised to filter their water to avoid consuming more chlorine, because it may be affecting their thyroid metabolism. They would probably also benefit from a shower filter if their water smells of chlorine[30].

It is said that fluoride can displace iodine, and this is certainly biochemically possible, but the only evidence we have been able to find indicates that it doesn't actually occur and that it isn't a problem[31].

Some pharmaceuticals can cause problems: particularly beta blockers (for high blood pressure and heart disease), dilantin (for epilepsy) amiodarine (for heart arrhythmias), glucocorticoids (for inflammation), SSRIs (for depression and anxiety), opiates (for pain), lithium (for bipolar depression) theophylin (for chronic obstructive pulmonary disease), and chemotherapy.

The phyto-estrogens in soy have been shown to give a three-fold risk of worsening the effectiveness of the thyroid[32]. (Naturally fermented soy, found in tofu and traditional soy source, is probably less of a problem than modern, industrially produced soy protein.) It is interesting to note that many "natural, healthy" food products have a high amount of soy in them.

Unfortunately the earth is basically a toxic waste dump, with hundreds of new chemicals being developed every month, and the no reliable pathology tests for most of them. We will consider how this can be dealt with later.

Stress

There are at least four different relationships between the stress response system and the thyroid mechanism. One of these is the way that T3 potentiates some of the actions of adrenaline. The other three relationships all involve a slowing down of the thyroid function by the stress response.

It appears as though we have a built in system to slow our metabolism down after long periods of stress. This is probably designed to enable our systems to recover; until the next time a major stressor is encountered. In animals in the Northern hemisphere, it is these mechanisms that bring about hibernation. Animals have been running around all summer getting stressed; then they want to sleep all winter. Which is a good description for the way that people who are experiencing this feel. (This can be really frustrating for people who are used to running at a fast pace.)

Cortisol has many functions; these can basically be summed up by saying that it protects the body from some of the damaging effects of an extended period of high adrenaline production.

One of the ways it does this is by suppressing the production of thyroid stimulating hormone (TSH). It also disrupts the conversion of T4 to T3, by creating “reverse T3” (rT3) instead of T3. Reverse T3 clogs up the thyroid receptors, but it doesn't actually seem to do anything.

The stress that people have been under doesn’t have to be emotional stress. It can be physical illness, an operation, fasting or even excessive exercise.

When cortisol becomes chronically low (which it can after years of stress) then T3 transportation can also be affected.

Autoimmunity

Autoimmunity generally is on the increase. As a topic it is beyond the scope of this document. As mentioned earlier, it is increasingly seen as resulting from a combination of genetic susceptibility, toxic load, and an inappropriate response to pathogens at some point in time[^]. It appears to be closely linked to digestive health, because a healthy digestive system is better at preventing toxins from entering the system, and because much of the immune signalling seems to be initiated in the gut.

When considering thyroid auto-immunity, the most important thing to understand is that it does not usually happen in isolation. There is a strong link with other autoimmune conditions, and the best results seem to be obtained by dealing with nutritional imbalances (particularly iodine and selenium deficiencies) and toxic load, and addressing the autoimmunity holistically, rather than seeing the thyroiditis in isolation.

There is a strong correlation between Hashimoto’s thyroiditis and coeliac disease; or simply gluten intolerance. There are mixed results in studies that try to improve auto-immune thyroiditis with a gluten-free diet; the most likely reason is that it is good for some people, and not for others. But it is certainly worth trying gluten elimination as a treatment for thyroiditis[33].

Identifying other food intolerances may also be significant, as can reducing underlying bacterial load.

Note that Hashimoto’s thyroiditis does not always drive T4 down. Sometimes the damage to the thyroid gland results in intermittent, uncontrolled releases of T4, causing short-term overstimulation of the metabolism. It can therefore, and particularly in the early stages, present in some people with similar symptoms to a mild bipolar depression (or cyclothymic mood disorder).

[^] The jury is still out in relation to the question of vitamin D and autoimmunity. Vitamin D appears to have an important role in regulating the immune system and, as people working longer hours away from direct sunlight, this may be having an adverse impact on vitamin D levels. But there is also mounting evidence that falling levels of vitamin D are a result of, rather than a cause of, ill health.

Liver dysfunction

A liver with sub-optimal function can potentially impact on the thyroid in three different ways: firstly, by failing to produce sufficient thyroxine-binding globulin (TBG), secondly by limiting the liver's capacity to convert T4 to T3, and thirdly by inefficiently removing toxins which may be interfering with the thyroid metabolism.

Low TBG levels do not reduce free T4 or T3; there are other proteins (albumin, transthyretin) that can also transport thyroid hormones. The main problem with low TBG is when total T4 and T3 are used as indicators, rather than free T4 and T3. Low TBG reduces the total hormone in circulation, just the not the available free proportions of it. When hormones are administered on this basis, they have been known to cause problems[34].

Severe liver disease will definitely impact on free T3, but not all that severely; even patients with cirrhosis of the liver appear to be able to maintain low to normal levels of free T3[35].

The main problem is likely to be the inefficient removal of toxins by the liver; we will deal with this in the next section.

An Approach to Diagnosis and Treatment

Diagnosis

The best place to start is probably by taking the temperature[^^]. (An oral mercury thermometer, under the tongue for 2 minutes, tends to be the most accurate in my experience). A single measure can be useful, but for the most helpful information, you can ask the patient to measure their temperature first thing in the morning, before they have eaten, drunk, showered or exercised. Because core temperature varies with a menstrual cycle, some authors recommend doing this for a month. But this isn't normally necessary to tell if there is a problem.

If the resting temperature is consistently between 36.5 and 37 degrees celsius, the person probably doesn't have a thyroid problem. If it isn't, they probably do[36]. A fluctuating temperature may suggest either the early stages of Hashimoto's thyroiditis, or an iodine deficiency.

Three simple lifestyle changes that may be appropriate for some people at this point are: eliminating soy from the diet, reducing the consumption of cruciferous vegetables (and eliminating raw ones) and switching to iodised salt. If these aren't appropriate, the person already does this, or you want to move faster, it is time for a thorough diagnosis.

If you routinely do blood tests it makes sense to continue to do these. In general, though, I find that iodine tests and hair-tissue mineral analysis (HTMA) are the most useful places to start[^^^] [37].

Blood tests are not a good way to assess the levels of most minerals. Heavy metals are displaced from the blood quite quickly; any heavy metal in the blood suggests a very recent exposure. Many trace elements (for example, calcium and phosphorus) are maintained within very tight parameters in the blood stream, and the tissue levels may be quite different to those in the blood. The application of a tourniquet is enough to alter magnesium levels. But blood tests for thyroid hormones are still useful (and, because they are fat soluble, thyroid hormones do not show up in saliva).

As well as TSH and free T4, it can be helpful, depending on the clinical picture, to look at the ratio between total T3 and free T3 (bearing in mind that this number might fluctuate), thyroid auto-antibodies, reverse T3, cortisol, CRP, and estrogen.

[^^] There is controversy about using the core temperature to diagnose thyroid conditions, but most of the controversy relates to 2 issues. The first is that temperature should not be used as a definitive test for thyroid medication. I couldn't agree more. The second is that there are other potential causes of a low core temperature, such as (possibly) adrenal fatigue and low protein levels. But these also often correlate with low thyroid function. As an easy and inexpensive test, I haven't read anything that contradicts the use of this as a first-line test, and I certainly haven't found anything that works better in a clinical setting.

[^^^] Hair-tissue mineral analysis is controversial, because of some studies that showed that different laboratories gave different values. In 2001 the Journal of the American Medical Association published a study recommending that the test not be used for diagnostic purposes, because of the variation between different laboratories. This variation was later shown to be largely a function of variations in washing procedures, and of poor procedures in specific laboratories. The best laboratories have now resolved this problem, and there is a lot of literature that supports the procedure (see the end note). Human hair is accepted as an effective tissue for biological monitoring of toxic heavy metals by the US Environmental Protection Agency, and is used for this purpose throughout the world.

If T4 and TSH are normal (or if TSH is a bit low and T4 is normal), reverse T3 is high, and if cortisol is above or below the reference range (but not sufficiently so to suspect Addison's or Cushing's diseases), and particularly if the patient tells you about a long period of severe stress, the problem is probably simply that the body is putting them into recovery mode.

I use a urine iodine spot test from Healthscope Pathology, although an iodine test with a 24-hour urine collection may be more accurate; I'm just not aware of where I can get this done in Australia. The hair-tissue mineral analysis I prefer to use is from Interclinical Laboratories, because they report on ratios in a particularly helpful way.

For reasons I haven't been able to ascertain (but probably because of impacts on the Kreb's cycle and the sodium-potassium pump), in a patient with low thyroid function, the HTMA will show a high ratio of calcium to phosphorus, and a high ratio of calcium to potassium. Some suggest that there is an inverse relationship between thyroid function and parathyroid function quite apart from the anatomical proximity of the glands, although if there is, this has not been well studied.

The hair test will also show the tissue levels of the necessary trace elements selenium, zinc and copper (but not halogens: iodine, chlorine, fluorine and bromine, unfortunately), and will also show toxic minerals such as mercury, lead and cadmium that may be interfering with the thyroid. Nutritional elements such as copper or cobalt, that can also have an adverse impact in toxic quantities, will also be shown.

One of the most useful parts of the HTMA is the ratio of nutritional elements to toxic elements. As mentioned early, a low ratio of zinc to mercury or selenium to mercury seems to invariably correlate with a thyroid problem, even if the zinc or selenium levels appear normal and the mercury level appears small.

Finally, the HTMA can give an indication of how effectively the liver eliminates toxins - or at least, it reveals the liver's glutathionation pathway, which removes metals, and also pesticides and petroleum distillates. People with a large number of elevated toxic minerals, even when the level of elevation is quite small, probably have a problem getting rid of things that shouldn't be in their system, including compounds that the HTMA can't detect.

There are a number of clinical indications that some sort of toxicity may be part of the picture. A multi-system presentation, rashes and other autoimmune conditions, and, particularly, irritation of the optic nerve (don't like bright lights; get a headache from seeing strange patterns) are all suggestive of toxicity, and especially in combination,.

Apart from heavy metals, there is no way to measure most of the environmental toxins that people are exposed to. What you can do is measure the liver's ability to eliminate toxins, using its various detoxification pathways. I use the Healthscope Pathology Functional Liver Detoxification Profile (FLDP) for this. It is a true test of liver function, in terms of detoxification, as opposed to most the standard blood tests for the liver, which are actually markers of inflammation. The FLDP involves salivary and urine tests to see what the liver does with paracetamol, aspirin and caffeine, and identifies which chemical detoxification pathways are doing what.

Treatment

The easiest conditions to treat are simple deficiencies of iodine, selenium, zinc or copper; patients can buy what they need from a chemist or a health-food store, and they feel better really quickly.

The second easiest are reactions to long-term stress. The best you can probably do for these people is to educate them: they need good food, gentle exercise, and above all, lots of rest and recuperation, with all the sleep they feel like. The problem will generally resolve itself within a few months, and the person will be able to gradually get back to the lifestyle they enjoy. If they try to push themselves through this time, they may be setting themselves up for chronic fatigue or something similar (which seems to happen when hard-driving people push themselves beyond the point of exhaustion, and their immune system loses its capacity to deal with a range of different pathogens). Slow release T3 may be appropriate for some of these people if they absolutely need to have more energy, but I'm not a fan. Their body seems to be trying hard to make them slow down and recuperate; I'm inclined to think it is best to let it have its way. Giving T3 to someone who is in stress recovery seems to me to be equivalent to giving a stimulant to a patient with a serious illness.

Toxicity (and that is closely linked to autoimmunity) is more complex, and it usually requires a multi-pronged approach. The FLDP comes with recommendations for foods and supplements to support the relevant liver-detoxification pathway(s). Please note that it is usually best to support the glycination pathway before attempting other detoxification, to minimize the adverse affects of detoxification.

The HTMA comes with recommendations of vitamins and minerals that will act as antagonists to whatever toxic mineral is identified; displacing them, essentially. There are a number of foods and vegetables (particularly organic leafy-green vegetables) that are thought to be generally helpful for detoxification, and some specific supplements which may be useful (such as modified citrus pectin). It is occasionally necessary to use chelating pharmaceuticals, but these tend to have side effects that should be avoided if possible.

Improving intestinal function is normally helpful as well; eliminating gluten is beneficial for most people, and some people also benefit from eliminating dairy, eggs, nuts, sugar, and other individual-specific foods. There are also a number of substances that are helpful for reducing intestinal permeability and improving gut function.

Manual treatment of the lymphatic system also seems to be beneficial in detoxification.

The most important part of detoxification, however, is to eliminate anything that is causing an ongoing contamination. Mercury is particularly significant in this regard. I used to think that mercury-amalgam fillings were not a problem, because I saw patients with fillings and no discernable mercury in the hair. I now think that mercury fillings are safe as long as they don't break down, but that when they do there is no way to eliminate mercury from a person's system until the filling is removed.

One patient, with high and intransigent mercury, reported spitting liquid mercury into the bowl when one of her mercury fillings was removed; the rest were fine. Once that filling was removed her mercury levels started dropping quickly, and her health improved. (If patients are having their fillings removed, advise them to ask the dentist for a rubber dam so that they cannot swallow any amalgam).

Detoxification is generally a good way to treat autoimmunity. Removing any specific allergens, and removing things that are generally difficult to deal with like gluten and casein (the protein in dairy products) are also often helpful. Improving gut flora by eliminating sugar and adding oats can often make a significant difference.

Detoxification is also probably the best place to start treating other hormonal imbalances that may be affecting the thyroid, such as an excess of estrogen, because many of the toxins that people are routinely exposed to are known hormone disruptors. Examples are

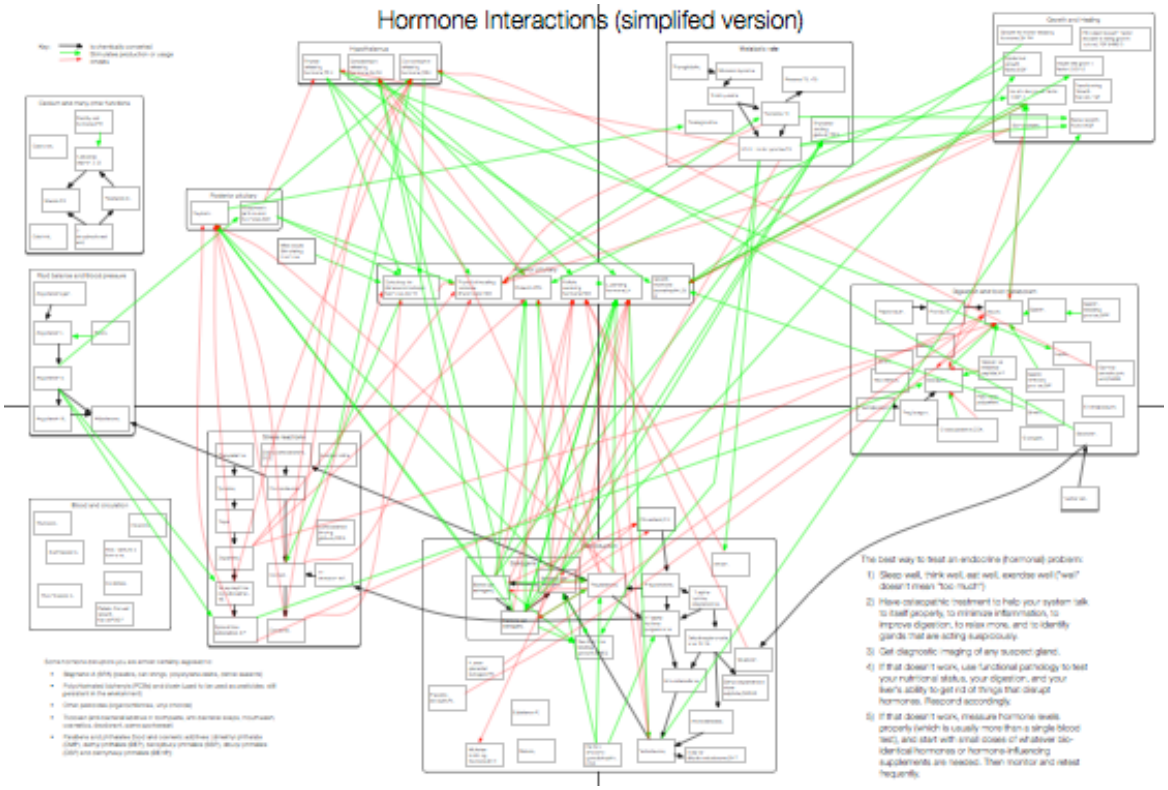
bisphenol A (BPA) (plastics, can linings, polystyrene resins, dental sealants); polychlorinated biphenyls (PCBs) and dioxin (used to be used as pesticides; still persistent in the environment); other pesticides (organochlorines, vinyl chloride); triclosan (anti-bacterial additive in toothpaste, anti-bacterial soaps, mouthwash, cosmetics, deodorant, some sportswear), parabens and phthalates (food and cosmetic additives: (dimethyl phthalate (DMP), diethyl phthalate (DEP), benzylbutyl phthalate (BBP), dibutyl phthalate (DBP) and diethylhexyl phthalate (DEHP)). These are far to common in the environment for most people to avoid in their daily lives, and so the best approach would appear to be doing everything possible to ensure that the patient's system can eliminate them efficiently.

Once significant and obvious toxicity has been decreased, most cases of estrogen/progesterone imbalance seem to respond quickly to osteopathic treatment (focusing on the pituitary, the thoraco-lumbar junction (which supplies most of the nerves to the pelvis), the pelvic mechanics and the pelvic lymphatics.

If you decide to give a thyroid supplement, something that combines T4 and T3 is probably the best. For some people this can be the quickest way to get them feeling better, but it will tend to create dependence (because supplemental thyroid hormone will reduce the production of TRH and TSH), and it may not resolve the underlying problem.

Hormones work together in an incredibly complex web; you take one, you impact others. They are all very powerful, and they all have multiple effects; they can change emotional responses, thought patterns, physical appearance, growth of particular parts of the body, and many other things.

The following diagram is a shrunk-down version of a large document, which shows a simplified version of the interrelationships between hormones. The green arrows show that a hormone stimulates another, the red arrow indicates inhibition, and the black arrows show precursors. When a person takes a hormone as a supplement, their natural homeostatic mechanisms can't operate correctly. I would therefore recommend treating any form of hormone replacement as a last resort, rather than a first line of treatment.



(The diagram is available from me as a PDF which is actually readable if anyone would like it.)

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