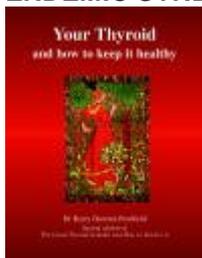


THYROID AND ADRENAL DYSFUNCTION; THE DIAGNOSIS AND TREATMENT OF AN ENDEMIC SYNDROME



Thyroid specialist Dr Barry Durrant-Peatfield explains why practitioners should check for thyroid and adrenal problems when presented with a familiar range of symptoms that often mask prolonged dysfunction in these two organs.

Introduction

The first quarrel many people may have – but most especially doctors – may well be in the title: the use of the word endemic. To read a number of learned articles would leave the impression that thyroid dysfunction was a matter of a few percent of the general population and adrenal dysfunction much less than this.

I have to say now, right at the outset, that this widely held view, held by orthodox medicine, is quite wrong, disgracefully so. It is, in the profession, politically correct to believe that thyroid dysfunction is not common at all and the symptoms are due to other things, notably depression. If you want to appear really learned, you will speak of maladaptation to one's environment due to a personality dysfunction.

Adrenal dysfunction fares worse and is believed to be about as rare as hens' teeth. In any event, the argument goes, Addison's disease (total adrenal failure), by definition, hardly occurs.

I cannot emphasise strongly enough that both thyroid and adrenal dysfunction are so commonly met with as indeed to be endemic and disgraceful diagnostic failure is the rule. I speak after more than 25 years of practical study and treatment of both conditions and I have suffered at the hands of an ignorant establishment for daring to diagnose and treat these problems. If you gain from this article nothing else, you must take away this fact: thyroid dysfunction, and its partner in crime, adrenal dysfunction are all around us, every day, in our clients/patients and even our colleagues. So common are they that whenever your opinion as a practitioner is asked on an apparently complex illness, you must ask yourself first: could there be an underlying thyroid and adrenal problem? (For the moment I shall not consider over-activity of either, but only sub-optimal activity.)

Down-regulation of the metabolism

Thyroid and adrenal dysfunction usually come together but in varying degrees of emphasis. They cause a down-regulation of all metabolic processes, hence these clients/patients are all hypometabolic. Metabolism, as I need not remind you, may be defined as the rate at which energy is produced and consumed by the tissues. Glucose, oxygen in; carbon dioxide, water, energy out. For everything to work properly this process has to be optimally regulated and it is the thyroid hormone that does this.

Thyroid hormones ensure that the cell membrane is actively and positively involved in the transport of the raw materials of energy production – not just oxygen and glucose but all the required enzymes and co-factors passing into the cell; and, equally, the passage out of the waste products once the energy has been released. The activity of the Krebs cycle within the mitochondrion is equally thyroid dependent.

It's helpful to think of the whole organism as an incredibly complex and intricate, electric machine that is built and wired up for 240 volts. If we try to run it at 190 volts it's not going to work properly. The lights flicker and grow dim. A down-regulated metabolism affects the organism in the same way. This means nothing works properly. Every tissue, every organ, every biological process, suffers. Some pieces of the equipment are affected more than others and differently, depending on the original design.

In humans probably the organ most sensitive to metabolic down-regulation is the brain. Not far behind are the highly metabolically active liver, then the kidneys, then digestive processes, then our temperature control and the intricate working of our immune system. You see, the point is you name it and it doesn't work properly. Our clients/patients may have lots of

different things wrong with them, so many that even they think they are hypochondriacs, never mind the doctor who came to that conclusion some time before.

It is essential to understand that a down-regulated metabolism has a total effect on the body's functioning. You cannot, you must not, focus on one or two symptoms; they must all be considered as a whole. How difficult this is in today's environment. There's never enough time, it's all got to be written down, quick decision, then out the door. Another symptom? Ah! Make another appointment and we will talk about it later. Here the advantage many of us have who practise away from the mainstream becomes apparent – we have time!

Hypothyroidism – recognising the symptoms

Let's take hypothyroidism first - probably the most common misdiagnosis of all common illnesses. A decent history soon gives us a few facts. Fatigue, weight gain, brain fog, depression, cold/heat intolerance, arthralgic aches and pains, fluid retention, bad skin, menstrual problems and infertility, hair loss, intractable constipation and lots of others. If you take these separately, how easy it is to narrow the focus and go for the wrong thing. Typically, a standard assessment might be:

Fatigue: Well, go to bed early, don't work so hard, you are depressed, anaemic - some Prozac and iron pills and you'll be OK.

Weight: you eat too much - try this diet.

Brain fog: well, none of us are getting any younger, are we – too much stress.

Depression. Ah! We can help you there, no trouble.

Cold/heat intolerance: your age - wear warmer things.

Arthralgia: bit of arthritis here I'm afraid - try these NSAIDs.

Fluid retention - lovely pills here, make you pee, no trouble.

Skin: bad diet, hormones.

Bad periods: one of the crosses you women all have to bear.

Hair loss: age, diet - better hairdresser.

Constipation: got something for that or try the chemist.

All the time the underlying problem is being missed. A simple examination can pick out the thyroid deficiency in minutes. And we'll come to the tests later.

Possible causes

Now, as to why the thyroid goes wrong there are various possibilities. It may be due to control failure – dysfunction in the hypothalamus or pituitary. It may be failure of conversion of T4 into T3, due to enzyme failure and poor adrenal function. There may be failure or resistance to binding of T3 at the receptor sites, damaging gene expression – the Gq/11 proteins may be activated to switch off this response. And, most of all, it may be primary failure in the gland itself. Primary failure in the gland may be:

1. Genetic, appearing at, or soon after, birth. A baby with thyroid failure is true cretinism.
2. It may be due to environmental deficiencies, like iodine, or the presence of toxins such as fluoride, PCBs, dioxins and endless others.
3. Trauma - general surgery, especially in women; for example, cholecystectomy, hysterectomy. Or damage to the gland from whiplash
4. Glandular fever
5. Autoimmune disease

6. Pregnancy and childbirth

A word about the over-active thyroid. Again, more common in women, but you don't see that many; probably two for every 100 under-active. Not a difficult diagnosis; the patient is hot, terribly nervy, has loose bowels, is losing weight and has a racing pulse. The only real differential diagnosis is an anxiety state.

Adrenal dysfunction and the General Adaption Syndrome

Now for adrenal dysfunction. The glands can go wrong in two ways: too much or too little. Too much and we have Cushing's disease. Only three causes really; too much cortisone for the asthma, rheumatoid arthritis or whatever; an adenoma on the gland itself; or one on the pituitary. We are all familiar with the moon face, thin skin, obesity, buffalo hump, so it shouldn't be difficult. A lot of people are checked out for Cushing's just because they are a funny shape but, unless caused by over-medication, it isn't very common at all.

Adrenal hypo-function is a different kettle of fish entirely. The diagnosis is missed more decisively even than hypothyroidism because most clinicians only seem to think of it in terms of Addison's disease. In fact, the adrenals can malfunction in degrees. A failure in pituitary control may play a role, often as a spin-off from a generally low metabolic state; but usually the failure is the result of long-term stress. Hans Selye recognised three major stages in adrenal hypo-function, which he called the General Adaption Syndrome (GAS).

Stage I is the stressed phase where, due to illness or other stress, the adrenals mobilise cortisol and DHEA (dehydroepiandrosterone) to help the body deal with the problem. This is an acute phase; the stress settles and the adrenals settle down.

Stage II, resistance, is where it all goes on and on and the adrenals enlarge and increase their hormone output on a longer-term basis. This can go on for weeks, months, or even years but eventually they can't take it any more and start to regress into exhaustion, stage III. The degree of exhaustion is reflected in their output and balance of their two chief hormones, cortisol and DHEA, which is where the Adrenal Stress Index (ASI) comes in.

The last phase of the GAS is what we will often see if we are looking for it. Important causes, of course, are major life-events, trauma, operations and the general and prolonged ghastliness of existence. But, for our purposes, the big cause is persistent and chronic illness. In the attempt to help the body deal with, and compensate for, the illness, adrenal exhaustion starts to take over. An example of a persistent and chronic illness untreated, or mistreated, is hypothyroidism.

So now you see why thyroid and adrenal hypo-function have to be considered together. Here's a precept for you. Anyone with undiagnosed or unsatisfactorily treated hypothyroidism will sooner or later inevitably slip into adrenal exhaustion. May take months or years, but it will happen. Symptoms of adrenal exhaustion exaggerate pre-existing thyroid symptoms. There will be ongoing and endless fatigue, even less tolerance to cold, depression, dizziness (postural hypotension), body hair loss, pigmentation (especially in skin folds), poor response to treatments generally and an obviously weak immune system. Most particularly, if there is an adrenal malfunction – I still prefer to call this low adrenal reserve – the patient responds poorly to thyroid hormone, or rapidly gets ill and toxic on even quite small doses of thyroxine.

Chronic fatigue and attendant problems

And now I want to broaden my sweep even further because we have to consider chronic fatigue. Something else to remember: all clients/patients suffering from chronic fatigue, CFS, ME, and fibromyalgia have a number of features in common. Most importantly, they are metabolically down-regulated. Whatever else is wrong, their thyroid and adrenal function is damaged. It's not as simple as that, of course, because the illness brings in its train other problems too.

There is likely to be viral load. This may follow from the original event that started it all off, a nasty flu or glandular fever. If there has been a deficiency in EFAs at any time, there may have been a lower than normal eicosapentaenoic acid (EPA) level since some of these big, nasty viruses can damage the 6-delta-desaturase enzyme that makes EPA (which is viricidal and makes interferon) from linolenic acid.

There may be systemic candida. Because it's systemic and lurking in the gut, it may not have been thought of but will be producing toxins and allowing all the problems of dysbiosis and a leaky gut. This must be looked for and treated.

There may be sex hormone imbalance, especially around the menopause; oestrogen dominance, which interferes with thyroid hormone, transport, production and receptor uptake. Intervention may be considered here if there is marked deficiency or imbalance. Food allergies can be an associated problem, especially if dysbiosis has been marked. There is often deficiency of essential minerals and vitamins which have to be sought for, especially if malabsorption is a feature, as is likely. Prof Basant Puri's work at Imperial College, London, has focused on EPA and virgin EPO as a method of clearing the viral load, which has had some success; and Dr Sarah Myhill, a private GP in north Wales, uses D-ribose, L-carnitine, magnesium, co-enzyme Q10 and high dosage B12 as another approach.

Treatment strategies for thyroid and adrenal dysfunction

I am going to pull together later the diagnostic approach to the detection of thyroid and adrenal dysfunction but before I do, it's time to consider our treatment strategies.

Thyroid

There are three levels of approach. The first is nutritional. To manufacture thyroid hormone there needs to be tyrosine, selenium and iodine, together with vitamin and mineral cofactors. We are most especially thinking of the B complex – B6 is crucially important – and magnesium, zinc, manganese and chromium. A number of companies make excellent thyroid support products – an example is Thyrocomplex from Nutri Ltd.

The next level is the use of natural glandular concentrates. In this country they are successfully produced as nutritional supplements. Nutri make Nutri Thyroid, which contains 130 mg of glandular concentrate and enzymes and enough thyroid hormones to improve thyroid levels greatly. A dose of one to four tablets daily is recommended.

The third level is thyroid hormone replacement. Many of you are aware of the prescription-only medicine, natural desiccated thyroid; Armour is the most well-known. This is available on-line in 30 mg, 60 mg, 120 mg and 240 mg tablets and may be used with confidence where the glandular concentrate has not provided significant improvement. A usual starting dose would be either 30 or 60 mg.

Then there is the use of synthetic thyroxine or levothyroxine. Because the generics appear to vary in potency (an accusation thrown quite unfairly and wrongly at Armour thyroid), clients/patients may not do awfully well on them, first because the dose is managed purely on the outcome of blood tests - instead of asking the patient and actually listening to them as an alternative to blood tests, which is actually not just frowned upon, it is now a hanging offence. (Thus far has evidence-based medicine brought us!) And, secondly, there is often an adrenal problem which, if not dealt with, will cause T4 toxicosis and/or an adrenal crisis.

Adrenal

Adrenal dysfunction may be detected clinically without difficulty – the Raglan test, Romberg test and pupillary reflex are most helpful and the Adrenal Stress Index will confirm the diagnosis. Treatment of low adrenal reserve has, like the thyroid, three levels and, if hypothyroidism is present, must be put in place before thyroid supplementation is begun. Nutritionally, the adrenals need vitamin C, 4 grams or more daily; they need pantothenic acid (B5) and benefit from the use of liquorice (wood or tincture), Siberian ginseng and coenzyme Q10.

Extremely valuable is the adrenal glandular concentrate; that made by Nutri Ltd is widely available (I recommend them because I have found their products to be very efficacious). One product contains 80 mg of the concentrate alone and another, 221 mg, together with a number of vitamins and minerals.

If this proves unsatisfactory, which is uncommon and usually because the adrenals have been really badly damaged over a period of time, the use of the adrenal hormone, cortisol (hydrocortisone), 2.5 mg up to 25 mg, may be considered by the practitioner, together with DHEA 25 mg or 7-keto DHEA 50 mg.

These measures will remedy the underlying damage, but intervention may be necessary to balance low progesterone or high oestrogen – the use of natural transdermal creams is the best method.

Systemic candida must be treated where present, using a fungicide (fluconazole can be helpful), together with grapefruit seed extract, caprylic acid, horopito, etc, along with an effective pre- and probiotic.

Food allergies have to be dealt with on their merits, usually by simple avoidance, while malabsorption may require the use of Betaine HCl and/or pancreatic enzymes. Nutri Ltd make a combination called NutriGEST.

A viral load may respond to VegEPA, the EPA – EPO formulation I mentioned earlier. Treatment of thyroid and adrenal insufficiency along these lines can be extremely rewarding and successful and a similarly broad approach in dealing with CFS has been proving very helpful also.

Pathological aids to diagnosis

Now we have a working knowledge of diagnosis and treatment, it is right to discuss the use of available pathological aids to diagnosis. Long recognised by many practitioners as an invaluable indication of thyroid deficiency is the waking temperature, as described by the American pioneer in this field, Dr Broda Barnes (author of *The Unsuspected Illness* and founder of the Barnes Foundation in Trumble, Conn).

Thyroid first of all. Standard NHS testing tends to be restricted to TSH (thyroid-stimulating hormone) usually, with Free T4 thrown in if you're lucky. The full range, which includes T4, T3, TSH and TPO and TgAb antibodies will not usually be done, even under extreme demand, and so clients/patients will often seek the help of private laboratories.

The full thyroid screen, if possible, should always be done, but you must remember that the TSH is often unreliable if the pituitary is suffering from a hypo-metabolic state and there are other reasons that can affect its reliability. If it's high, well and good; if it's at normal levels, it proves nothing. T4, if low, again good, if it's normal, it may mean that it is not being properly used, is building up and causing a false result. The same argument applies to the T3, but the antibody test may be relied upon.

Thyroid tests should always be considered against the clinical findings and, remember, clinical observation is very much more reliable than tests. As you may have observed, I have a deep-rooted cynicism about the politically correct obsession with evidence-based medicine. Surely it should be observation first, tests second?

Where doubt continues to exist, this may be resolved by the 24-hour urine test. As far as I know only Individual Wellbeing and Diagnostic Lab, New Malden, Surrey, do this test in the UK; European Laboratories of Nutrition do it in The Netherlands. It relies on the findings of Hertoghe & Baisier, who showed the greatly increased reliability of the 24-hour urine which, after all, measures the thyroid production over a whole day, as opposed to the blood test, which is a snapshot of a single moment showing levels that can vary widely with time of day and other variables. The test demonstrates the amount of T4 and T3 actually used and passed through the tissues. It is much more useful than other tests and will show even minor degrees of low thyroid function. In an ideal world both the full-serum, thyroid screen and the 24-hour urine should be done, but there are logistic and financial difficulties that have to restrain one's enthusiasm. While interpretation of the 24-hour urine is perfectly straightforward, the blood thyroid screen has to be carefully assessed.

Beware the pitfalls

The first stumbling block is the TSH; a rise of TSH indicates poor thyroid response but the level considered to be indicative of this can be a matter of different interpretation by laboratories and doctors. The recent guidelines issued by the British Thyroid Association suggesting that treatment should not be offered below 10 units is quite unbelievable. The American Association of Clinical Endocrinologists has recently considered a level of 3.2 to be the cut-off point and in my view anything over this, combined of course with a clinical appraisal pointing to the diagnosis, should demand intervention. Labs seem to vary between

about four and six; but I say again, anything over two should arouse suspicion and anything over 2.5 should result in a trial of treatment at the very least.

The trouble with the TSH is that it may not be a proper response to thyroid uptake since there are four types of thyroid receptor and a metabolically challenged pituitary cannot respond properly anyway. So a high level is valuable, a low level may mean nothing.

Measurement of T4 and T3 has always been very much subject to error. The obvious difficulty is that it is only, as I said earlier, a snapshot of a level that may vary a lot during the day, but it goes deeper than that. Failure of uptake by exhausted and missing receptors will allow levels to be normal or even high in the bloodstream, simply since thyroid hormone isn't being used, and conversion deficits can further distort the picture. The presence of thyroid antibodies (TPO and TgAb) should not usually cause much difficulty: either they are well raised or they are not, allowing an immediate diagnosis of Hashimoto's or Graves' disease to be made. It should be borne in mind that the levels fall away with the passage of time.

Reverse T3 is sometimes of help; high levels may indicate poor T3 receptor uptake, with the system trying to rid itself of surplus T3. General illness, malnutrition and trauma will also raise the T3.

Tests for adrenal function

We come now to the tests for adrenal function. Once again, one simply must place the clinical picture to the fore. A serum cortisol is greatly relied on by orthodox medicine. It is almost useless. Similarly, the Synacthen (long or short), in which ACTH is injected to test adrenal function seems to be of significance only where the adrenal insufficiency is plainly Addisonian.

More helpful by far is the salivary Adrenal Stress Index, which measures cortisol and DHEA output in 24 hours. Here the true picture of adrenal stress may be recognised and the three stages of the General Adaptation Syndrome clearly shown.

High levels of cortisol and DHEA show adrenals under stress. Sometimes the cortisol pathway starts to fade as exhaustion sets in, with DHEA still reasonably present. Less commonly, there may be a really high DHEA – a response to ACTH stimulation – but with the cortisol pathways responding poorly. Erratic levels in both are evidence of strain and uneven response.

The 21-hydroxylase and 17-hydroxylase enzyme deficiencies may be apparent here; really weak cortisol with high androgenic output enough to cause virilisation. Where cortisol levels are obviously weak and DHEA response is weak and flat, the diagnosis of adrenal insufficiency (low adrenal reserve) slowly heading toward Addison's disease may be made.

Sex hormones

We have to consider the sex hormones in more depth. Many of us are aware that in women hypothyroidism may start with, and parallel, the menopause. One scenario is the progressive loss of progesterone often some years before the menopause itself. Apart from the obvious increased risk of osteoporosis, the imbalance will lead to oestrogen dominance – with weight gain, bloating, mastalgia and heavy, painful periods. This will increase thyroid-binding globulin, thus taking some thyroid out of use; it will adversely affect thyroid manufacture and receptor uptake. All the symptoms are put down to the menopause, of course, when actually the increasing deficiency of thyroid is the main problem.

Later, both hormones run down and under the principle of permissive action, where hormone production requires the other players in the endocrine orchestra to play their parts well and in tune, thyroid production and processing may be affected adversely. So being able to detect oestrogen dominance is pretty helpful – one can correct the balance with natural, transdermal progesterone. If levels of both are low, careful use of natural progesterone and oestrogen can be very valuable.

So, we should consider most carefully the menopause profile, or at the very least, a spot check of a day's output of progesterone and oestrogen. In menstruating women, thought may be given to assessment of both throughout a month. In general, however, getting the thyroid

and adrenal status right will often provide welcome correction in much of the menstrual difficulties and it may perhaps be considered at a later date.

The lads must not be left out of the equation. The male menopause most certainly happens – we can call it the andropause – but is usually an altogether more gradual and insidious affair. Ideally, assessment of testosterone levels should be carried out if the slightest doubt exists.

Conclusion

To sum it all up, I am here making a plea for the recognition of the very common syndrome of thyroid and adrenal deficiency, using observation and medicine practised as an art, as the primary diagnostic method, with the lab playing a secondary role. Further, be aware that the syndrome has global effects, with imbalance of other hormones, the likely presence of systemic candida and dysbiosis, malabsorption and food allergy all playing a probable role.

Dr Durrant-Peatfield is the author of *The Great Thyroid Scandal and How to Survive it*, which is about to be re-published in April in a fully updated, 2nd edition as *Your Thyroid and How to Keep it Healthy* by Hammersmith Press (www.hammersmithpress.co.uk), from which the above information is taken.

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