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TEMPERATURE DYSREGULATION IN STRESS

Chronic stress sufferers often have a significant problem with cold hands and feet by the time they consult me. Eventually all people suffering from unremitting stress develop temperature dysregulation and low body temperature—and with that lose the efficiency of their enzymes, much like a defect in the furnace reduces the efficiency of a home heating system. There are five core points in this discussion:

First,

adrenaline and related stress hormones are potent vasoconstrictors. When present in excess, those hormones cause spasm of the muscle in arterial walls, narrow the lumens of vessels, impede the flow of blood in tissues and cause the tissue temperature to fall. Every week, I spend about three hours in my autoregulation laboratory teaching my patients how to flush their cold hands and feet with warm blood by allowing their arteries to open up by shutting out the thinking mind. I do not recall a single laboratory session in which one or more patients did not see their skin temperature shoot up from below 80 degrees to 90 degrees or over. Clearly, this is an autonomic function. (The term autoregulation actually derives from my early work in this area, when I used to call it autonomic regulation. My patients shortened it to autoregulation, then to autoreg.) Equally clearly, such a rise in skin temperature cannot be attributed to thyroid manipulation because no thyroid hormone is used in such work. Furthermore, temperature regulation through thyroid hormone therapy takes several days or weeks, whereas I observe changes in the skin temperature in minutes.

Second,

accelerated oxidative injury in chronic stress damages the membranes of red blood cells and makes them sticky, resulting in cellular sludging. I commonly observe large clumps of red blood cells when I examine a drop of blood from patients suffering from chronic stress with a high-resolution microscope with phase-contrast and dark-field optics. Normally, tiny blood capillaries in tissues open wide enough to let only one or two red blood cells flow through them side by side. Large clumps of blood cells cannot flow freely through capillaries, and literally choke off tiny capillaries.

Third,

Oxidative injury also causes blood platelets to form clumps that impede blood circulation and increase blood coagulability. The same holds true for the white blood cells, though to a lesser degree.

Fourth,

Accelerated oxidative injury damages the receptor sites of the autonomic nervous system, and so causes autonomic dysregulations. This adds to the vasoconstriction caused by excess adrenaline and further lowers the tissue temperature.

Fifth,

Accelerated oxidative injury in chronic stress eventually leads to hypothyroidism. I am certain that the mechanism of such injury is accelerated oxidative injury to the enzymes which are necessary for production of metabolically active thyroid hormones from their inactive precursor. Furthermore, I confidently predict that this will be proven with future research.

Underactivity of the thyroid gland slows down the entire metabolism and results in a fall in the body temperature. Even a small drop in body temperature, such as three-quarters to one degree, can significantly impair enzyme efficiency, sometimes causing as much as a 50% drop. Lowering the body temperature by one-half to one degree has the same effect on human metabolism as lowering the thermal efficiency of a home heating system—the fuel burns inefficiently. The firm proof for this phenomenon is the common observation that people living highly stressful lives feel much better and more energetic on days when their temperature is normal and do poorly on days when their temperature is low. This question, within the context of human total body metabolism, has not been well investigated. I am confident that when it is, it will reveal a large drop in metabolic enzyme efficiency—a major physiologic handicap for those with low body temperature.

Many environmental pollutant molecules such as dioxins show close structural similarity to thyroid hormones and fool thyroid receptors on cell membranes. Thyroid dysfunction so caused further slows down metabolism. This mechanism, though not directly related to the stress chemistry, adds to the degree of thyroid gland injury.

TEMPERATURE UP-REGULATION WITH T3 AND T4 THERAPIES

This subject is generally considered very complex. It need not be. Below, I describe how thyroid dysfunction occurs in chronic stress states. Seven mistakes are commonly made in this area:

First,

the impact of persistently low body temperature on energy and detoxification enzymes is not recognized, either by the patient or the physician. The fundamental difference between living beings and nonliving things, as I wrote earlier in this volume, is that living beings are enzymes beings, and all enzymes are temperature-dependent.

Second,

the enzymes that convert inactive and weakly active thyroid hormone precursors to active hormones are damaged by oxidative injury. I am certain of this, though this has not yet been proven with actual studies. This oxidative injury leads to diminished production of active T3

hormone and persistently low body temperature. The proof of this is in the common observation that body temperature cannot be normalized in many patients even with large doses of natural thyroid extract or synthetic T4 preparations, such as synthroid. Yet, the temperature rises within days when optimal doses of T3 are used. Furthermore, oxidative injury damages the thyroid hormone receptors. I address this subject later in this section.

Third,

in the prevailing dogma of endocrinology, thyroid function is frequently assumed to be normal if the commonly performed T4, T3 uptake, quantitative T3 and TSH tests show negative results. Rather large drops in the blood hormone levels are ignored simply because the test value falls within the "normal" range. The frequency with which otherwise knowledgeable physicians make this mistake amazes me. For example, the normal range for T4 in most laboratories is 4.5 to 12 mcg/ml. However, a fatigue sufferer with a normal hormone level of 12 mcg/ml might drop it by 50% down to 6 mcg/ml. According to the prevailing dogma, this would be considered a negative result, while in reality it represents a significant degree of hypothyroidism (underactive thyroid gland).

Fourth,

hypothyroidism is assumed to exist when the body temperature is below normal, and no attempts are made to investigate dysfunction of the autonomic nervous system. Predictably, when low body temperature is due to autonomic dysfunction, thyroid replacement fails to restore the body temperature to the normal range.

Fifth,

the dose of thyroid hormone is continually increased even when such therapy neither affords clinical benefits nor raises the body temperature. I have seen cases where the dose of T4 or T3 was pushed to a very high value with resulting rapid heart rate, palpitations, and in some cases, cardiac arrhythmia.

Sixth,

insufficient attention is paid to the impaired function of the adrenal gland and other hormonal dysfunctions that frequently coexist with underactive thyroid status. The oxidative injury that slows down enzymes involved with thyroid hormone synthesis can also be fully expected to slow down the enzymes that are essential for production of adrenal hormone. This indeed does happen. The blood levels of DHEA, an adrenal hormone, are almost always low, often markedly, in patients with persistent chronic fatigue.

Seventh,

little, if any, attempts are made in clinical endocrinology to consider the nutritional basis of hormonal dysregulations. This is remarkable because even a cursory look at the biochemistry charts outlining enzyme pathways shows how multiple nutrients are essential for each enzymatic step in hormone synthesis.

I carefully study temperature dysregulation in all patients with chronic illness using appropriate blood tests and asking the patient to take oral as well as axillary (underarm) temperature readings immediately after waking up and before getting out of bed. The oral and axillary temperature readings are again taken three and six hours after the morning reading. In addition, I ask the patient to record his/her pulse in the morning. These steps are repeated on three consecutive days.

The oral temperature should range from 98.2 to 98.6 degrees. Axillary temperature should range from 97.5 to 98 degrees.

In my clinical practice, if the body temperature is more than a half degree lower on average, I address all the issues listed above. For thyroid hypofunction, I usually prescribe a small dose of one grain of natural thyroid extract (Armour brand). Four weeks later, I do a clinical evaluation and repeat body temperature readings — oral, axillary or both, depending on the pattern of temperatures observed during the initial readings. If the small dose of thyroid extract fails to raise the body temperature to the desired level, I increase the dose by small increments such as one-half to one grain at a time. It is essential to monitor the pulse rate during all types of thyroid therapy. I clearly instruct the patient not to increase the thyroid dose if the pulse rate begins to rise by more than 10 beats per minute above the initial reading or when the pulse rate climbs above 90 per minute.

T3 TEMPERATURE UP-REGULATION

At times, thyroid extract therapy fails to give any clinical benefits and low body temperature persists. After careful re-evaluation, if I still think the thyroid gland is not functioning properly, I move on to the use of small doses (7.5 to 30 mcg daily in two divided doses) of slow-acting T3 preparations, and increase the dose in small increments of 7.5 mcg (as suggested by my friend, Dennis Wilson, M.D.), carefully watching the pulse rate and body temperature and looking for clinical signs of improvement.

In my office, I use temperature and pulse sheets for recording daily temperatures, pulse rates and symptom scores. I designed these worksheets specifically to assist and guide the patient in following my instructions. I cannot overemphasize the absolute need for careful monitoring of both body temperature and pulse rate so that an overcorrection of the thyroid gland does not lead to hyperthyroidism and excessive stress on the heart.

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LAST UPDATE ON: 12/23/2004

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