Dynamic Chiropractic

HEALTH & WELLNESS / LIFESTYLE

T3 and Chronic Fatigue

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What follows is more "academic" than my usual column. But bear with me -- the basis of my brief and general description of muscle metabolism could have far-reaching clinical possibilities. I'd be interested in any comments readers may have.

Researchers believe that trigger points (TPs) form in areas of muscle where blood flow is impeded.[1] A spasm can start the process by compressing arteries that course through muscle.[2] When the arteries are sufficiently compressed, muscle fibers fed by those arteries become deprived of at least three chemicals vital to the fibers' energy metabolism -- glucose, B vitamins and the thyroid hormone T3. Mitochondria inside the muscle fibers require all three of these to generate ATP, the energy packets that fuel muscle fiber functions. When ATP production drops below the critical level, the contractile filaments inside the muscle fibers lose their ability to lengthen (or relax). These filaments stay locked to one another, racheted into an extremely shortened state. This is called an "energy-deficiency contracture."[3]

When the spasm releases, arterial flow increases. If the fresh arterial blood contains enough glucose, B vitamins and T3, they can restore ATP production to normal. Energy becomes available to release the contractile filaments and relax the involved muscle fibers. But if the blood is deficient in any of these three chemicals, ATP producion will remain inadequate to fuel relaxation of the filaments.

Mitochondria float alongside the contractile filaments in the muscle fiber's fluid. Inside the mitochondria, T3 enables the transfer of energy from glucose to ATP molecules. Then the ATP travels to the contractile filaments and energizes their release.

If the mitochondria don't get enough T3 (or if the T3 is faulty), ATP production is stifled. A person's muscle fibers will contract properly, but under-fueled by too little ATP, the fibers won't completely relax. This energy-deficiency contracture leads to the other local pathophysiological changes that constitute TPs.

T3 inadequacy is usually a systemic phenomenon. It can, therefore, cause energy-deficiency contractures in muscles throughout a patient's body. His contractures are tender to palpation and may wildly refer pain or paresthesia when compressed. Noxious signals from these contractures stimulate the patient's reticular activating system, intensely arousing him. From this point, a complex set of interactions ensue. His arousal causes insomnia and shallow, non-restorative sleep. The sleep disturbance, combined with his ATP deficiency, weakens him and causes sustained fatigue. His ATP deficiency, combined with his arousal, causes constipation, and then spastic colon or irritable bowel syndrome. And the lack of T3 can desensitize the brain's adrenergic receptors, dulling their response to the neurotransmitter norepinephrine. This leaves him depressed.

It's noteworthy that most of the above symptoms are the same as those that get patients the diagnosis of fibromyalgia, or chronic fatigue syndrome.

Many classic hypothyroid patients develop secondary energy-deficiency contractures and TPs.[4] But I've observed other patients who first injure a muscle that later develops active TPs. As time passes and these patients suffer neverending somatogenic stress in the form of myofascial pain, they develop insomnia, fatigue, gastrointestinal dysfunction and depression. Some of these patients are dramatically relieved of this syndrome when treated with a T3 supplement, although T4 seems to be of no benefit (which I'll explain below). Because of these observations, I've come to suspect that these patients' somatic stress has induced an intracellular T3 deficiency. This deficiency, in turn, has produced and sustained the complex clinical picture they suffer from.

Most of the hormones produced by the thyroid cells are T4. An intracellular enzyme in other cells removes an iodine from T4 to produce T3, the more metabolically active hormone. When T3 gets inside the mitochondria, it enables the transfer of energy from glucose to ATP.

In theory, T4 is converted to T3 as cells require it. Because of this, many MDs treat hypothyroidism with synthetic T4 (such as Synthroid) believing that this will be converted to T3 as needed by the cells. This has provided us with the means to see what happens when the patient takes too large a dose of T4, and what is significant here in the antidote.

When the patient takes too much T4, his cells produce too much T3. This accelerates his metabolism to the point that he develops the symptoms of hyperthyroidism. The antidote -- block the body's conversion of T4 to T3. Glucocorticoids, when injected, do exactly that.[5] They block the further conversion of T4 to T3, slowing the metabolic rate.

Noxious signals from TPs are transmitted into the CNS through types C and A delta fibers. Noxious input through these fibers constitutes severe physical stress -- so much so that the person is emotionalized and mobilized, presumably to remove the noxious stimulus.[6,7,8] When myofascial patients are continually aroused in this way, they excrete an excess amount of stress hormones. This is evidenced by their higher than normal urine levels of adrenaline and noradrenaline.[9] Noxious arousal of myofascial origin has also been shown to increase adrenal glucocorticoid secretion.[10] And this may be the biochemical link to profound fatigue in many patients.

The endogenous glucocorticoid output may be sufficient to block the conversion of the patient's own T4 to T3. If so, his body's own stress-induced block causes a chronic T3 inadequacy. This may impair his energy metabolism enough in various tissues to produce the complex of symptoms termed "fibromyalgia."

My hypothesis about this mechanism implies simple, practical treatment: myofascial therapy for physical stress reduction and referral for T3 supplementation. This has dramatically relieved not only some patients' chronic fatigue, but also their G.I. and sleep disturbances, muscle pain, and depression. Only clinical trials will tell which myofascial patients respond to this treatment. I'm now arranging such trials. Hopefully, for the benefit of many myofascial patients, my past observations will be supported by the results.

References

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