Doctor Says No Patient Needs to Use T3, and T3 Can Be Dangerous

Reply by Dr. John C. Lowe

December 6, 2003

In the email below, a physician wrote to a colleague of Dr. Lowe, arguing that no patient needs to use T3 and that T3 can be dangerous. Below the physician's email, Dr. Lowe replies to his arguments.

Subj: T3 can be dangerous
Date: Dec. 2, 2003
From: Dr. XXXXX
To: Dr. XXXXX

Dear Doctor,

I have enjoyed your emails and have read them with interest. Having done quite a bit of Thyroid [sic] research, spent three years in Dr.'s [sic] Sidney Ingbar and Louis Braverman labs, the previous and most recent authors of the medical text *The Thyroid*, I feel I can speak with a little bit of knowledge . . .

It is extremely unusual for patients not to be able to convert T4 to T3. The common exception is when patients are deathly ill and they convert T4 to rT3 (reverse T3) which is inactive; this is referred to as euthyroid sick [syndrome]. Several ICU [intensive care unit] studies have not shown any better survival with the addition of T3 to these patients and is not recommended.

Outpatient use of T3 with or without T4 can be dangerous. You may recall the great rematch of Mohammed Ali with Joe Frasier. Mr. Ali needed to loose [sic] weight to get in shape for the fight and took Thyrolar, a T4/T3 combination medicine. He lost plenty of weight and the fight early in it's course. I believe his physician also lost his job. T3 is nothing to play around with. Moreover, I am concerned with your recommendations [of T3] to the less knowledgeable.

Dear Doctor:

A colleague of mine, to whom you wrote the email above, asked me to reply to your comments about T3. I've taken the liberty to do so because the replies to your arguments can be extremely important to the health and well-being of many patients.

The Metabolic Treatment of Fibromyalgia
by Dr. John C. Lowe
Readers' Comments

I hope that it's acceptable to you that I've replied.

In your email, you reach two conclusions: (1) that no patient needs to use T3 alone, and (2) that using T3 is dangerous for patients. Below, I address the implied or explicit evidence you give for these two conclusions. My point is to show that your conclusions don't validly follow from your evidence, and that your conclusions are false.

Your First False Conclusion: You first imply that no patient needs to take T3. Your reason is that except for those who are deathly ill, all patients effectively convert T4 to T3. That being the case, according to you, patients have nothing to gain by taking T3.

Many doctors mistakenly believe that patients should use T3 because they have impaired T4 to T3 conversion and, as a result, low T3 levels. But this has never been the reason for our patients using T3. Unfortunately, we're not certain why some patients must use T3 to free themselves from hypothyroid-like symptoms. Nonetheless, some patients must; T4 alone, and in some cases T4/T3 combination medicines, don't relieve their symptoms and signs; only T3 in fairly high dosages does.

One such patient was reported by Kaplan and colleagues in 1981.[1] To be healthy, the patient had to take 500 mcg of T3 per day. Testing showed that she didn't have impaired T4-to-T3 conversion, and she didn't have mutated beta-thyroid hormone receptors. Despite this, extensive testing showed that when she took this large amount of T3—and *only* then!—her metabolism was normal and she was free of symptoms. Importantly, she also had no tissue overstimulation.

We have studied and treated similar patients for the past seventeen years. We've reported our work with these patients as case reports and open systematic clinical trials,[2][3][4][5] double-blind, placebo-controlled trials,[6][7][8] and a long-term follow-up.[9] The patients' T3 dosages ranged from 50 mcg to 500 mcg—although I hasten to add that most require dosages only between 100 and 150 mcg. None of these patients had lab test results consistent with blocked T4-to-T3 conversion. Also, among those whose T3-receptor genes we sequenced, none had mutations. As with Kaplan's patient, our patients overall have remained healthy for years on these supraphysiologic dosages. None have experienced adverse health affects from their continued use of T3, and none have had evidence of tissue overstimulation upon serum and urine biochemical testing, electrocardiography, or bone densitometry. It's noteworthy that most of our patients who've recovered with T3 therapy previously failed to benefit from the use of T4 replacement.

I must comment parenthetically on one of your arguments for patients not needing to use T3. You wrote, "Several ICU studies have not shown any better survival with the addition of T3 to these patients and is not recommended." Some thyroid researchers agree with you about this,[18] including our research group. The low T3 levels of most critically ill patients have a protective effect, aiding the patients in resting and recovering from their illnesses.

There is, however, a notable exception that the other researchers and you neglect to consider: critically ill cardiac patients who benefit from T3 treatment. Studies show that T3 improves these patients' heart function in a variety of ways. It also decreases the severity and incidence of their cardiac abnormalities, and increases their survival rate.[10][11][12][13][14][15][16] Endocrinologists might not recommend T3 for these heart patients, but some cardiologists and cardiac surgeons definitely do. Rather than doctors such as you roundly denouncing T3 therapy, you'd better serve patients' welfare by reading the relevant studies and then rectifying your judgment of T3 to reflect its safety and the potential benefits for select patients.

Your Second False Conclusion: Your second conclusion is that T3 can be

dangerous. Your evidence is that Thyrolar, which contains T4 and T3, impaired Mohammed Ali's athletic performance. However, the Ali case doesn't at all logically lead to the conclusion that T3 is dangerous. I'll explain why.

Both Thyrolar and Armour Thyroid contain 38 mcg of T4 and 9 mcg of T3 per grain (60 mg). Many millions of patients have used and continue to use these products with no harm to themselves. Moreover, before the advent of the TSH test in the early 1970s, patients used these products in much higher dosages than nowadays. Yet the record does not show that patients were harmed by the higher dosages.[17] Most of our hypothyroid patients recover within the higher dosage range used before the early 1970s. Our meticulous safety monitoring for many years has revealed no adverse effects whatever; instead, the patients in general remain extraordinarily healthy.

However, some patients clearly do occasionally use dosages of the products that are too high for them individually. Ali was one such patient; *an excessively high dosage* left him weak, fatigued, and a poor match for Joe Frasier. The overstimulating dosage was indeed dangerous for him—he was in the ring with Frasier, a *powerful* slugger. But Ali's overstimulating dosage apparently didn't endanger his health in any other way. Consequently, the situational danger Ali faced certainly doesn't justify you inferring that using T3—regardless of the dosage—is dangerous in general.

Prejudice Against T3: Your conclusion that T3 is dangerous is unsound in another way. You imply that it was the T3 in the Thyrolar that impaired Ali's boxing performance. Indeed, too much T3 can impair athletic performance and can have other adverse affects. But so can too much T4. In fact, for patients whose cells effectively convert T4 to T3, too much T4 causes exactly the same adverse effects as too much T3. In that Thyrolar contains both T4 and T3, why did you attribute Ali's impaired performance only to the T3?

I suspect the answer to my question is that you hold a prejudice against T3—a prejudice you inculcated into your belief system while studying with Ingbar and Braverman. Working under the supervision of prominent conventional thyroid specialists such as Ingbar and Braverman carries a risk: The student doctor may unquestioningly accept prejudicial pronouncements by the prominent specialists—pronouncements that aren't based on research findings but instead on financial incentives from corporations. One such pronouncement is that the only thyroid hormone any patient ever needs to use is T4. Another is that T3 shouldn't be used because it's dangerous.

The student doctor, enchanted by his teachers' seeming authority, may believe the pronouncements throughout his medical career, and he may reflexly defend them without open-mindedly considering evidence that contradicts them. His reflex defense of the pronouncements may ensure that he doesn't treat patients with T4/T3 products or T3 alone. If so, he'll never learn how safe and effective these products are. He'll therefore remain miseducated about the products, and he'll lack clinical experience with them. Because of this, his conclusion that T3 is dangerous will be nothing more than a statement of prejudice that's completely without merit.

As a final note, I assume you've studied the book you mentioned, Ingbar and Braverman's *The Thyroid*. The book, which I've scrupulously studied, contains much good academic and some practical information. But the only method of thyroid hormone therapy the authors advocate and describe in the book (except in the thyroid hormone resistance chapter) is T4 replacement. So the book contains paltry little that's useful about thyroid hormone therapy. For the most comprehensive coverage of T4, T4/T3, and T3 therapies ever published, I strongly recommend my book *The Metabolic Treatment of Fibromyalgia*.[19] I suggest that you read the exhaustive information in the book on all forms of thyroid hormone therapy. Doing so is the single best way to fill the gaps and inaccuracies in your knowledge left by Ingbar and Braverman's tutelage.

—Dr. John C. Lowe (December 5, 2003)

References

[1] Kaplan, M.M., Swartz, S.L., and Larsen, P.R.: Partial peripheral resistance to thyroid hormones. *Am. J. Med*., 70: 1115-1121, 1981.

[2] Lowe, J.C.: Improvement in euthyroid fibromyalgia patients treated with T3 (tri-iodothyronine). Journal of Myofascial Therapy, 1(2):16-29, 1994.

[3] Lowe, J.C.: T3-induced recovery from fibromyalgia by a hypothyroid patient resistant to T4 and desiccated thyroid. Journal of Myofascial Therapy, 1(4):21-30, 1995.

[4] Lowe, J.C.: Results of an open trial of T_3 therapy with 77 euthyroid female fibromyalgia patients. *Clin. Bull. Myofascial Ther*., 2 (1):35-37, 1997.

[5] Honeyman, G.S.: Metabolic therapy for hypothyroid and euthyroid fibromyalgia: two case reports. *Clin. Bull. Myofascial Ther*., 2(4):19-49, 1997.

[6] Lowe, J.C., Garrison, R., Reichman, A., Yellin, J., Thompson, M., and Kaufman, D.: Effectiveness and safety of T_3 therapy for euthyroid fibromyalgia: a double-blind, placebo-controlled response-driven crossover study, *Clin. Bull. Myofascial Ther*., 2(2/3):31-57, 1997.

[7] Lowe, J.C., Garrison, R., Reichman, A., Yellin, J.: Triiodothyronine (T_3) treatment of euthyroid fibromyalgia: a small-n replication of a double-blind placebo-controlled crossover study. *Clin. Bull. Myofascial Ther*., 2(4):71-88, 1997.

[8] Lowe, J.C., Reichman, A., Yellin, J.: The process of change with T_3 therapy for euthyroid fibromyalgia: a double-blind placebo-controlled crossover study, *Clin. Bull. Myofascial Ther*., 2(2/3):91-124, 1997.

[9] Lowe, J.C., Reichman, A., Yellin, J.: A case-control study of metabolic therapy for fibromyalgia: long-term follow-up comparison of treated and untreated patients (abstract). *Clin. Bull. Myofascial Ther*., 3(1):23-24, 1998.

[10] Goichot, B. and Schlienger, J.L.: Les indications extrathyroVdiennes des traitements par hormones thyroVdiennes. *Rev. Méd. Interne*, 19:720-725, 1998.

[11] Dyke, C.M., Yeh, T., Jr., Lehman, J.D., et al.: Triiodothyronine-enhanced left ventricular function after ischemic injury. *Ann. Thorac. Surg*., 52:14-19, 1991.

[12] Klemperer, J.D., Zelano, J., and Helm, R.E.: Triiodothyronine improves left ventricular function without oxygen wasting effects after global hypothermic ischemia. *J. Thorac. Cardiovasc. Surg*., 109:457-465, 1995.

[13] Vavouranakis, I., Sanoudos, G., Manios, A., et al: Triiodothyronine administration in coronary artery bypass surgery: effect on hemodynamics. *J. Cardiovasc. Surg*. (Torino), 35(5):383-389, Oct., 1994.

[14] Wechsler, A.S., Kadletz, M., Ding, M., et al.: Effects of triiodothyronine on stunned myocardium. *J. Card. Surg*., 8(Suppl.2):338-341, Mar., 1993.

[15] Salter, D.R.: Acute severe post-ischemic myocardial depression reversed by triiodothyronine. *Ann. Thorac. Surg*., 54(2):301-305, Aug., 1992.

[16] Hamilton, M.A., Stevenson, L.W., Fonarow, G.C., et al.: Safety and hemodynamic effects of intravenous triiodothyronine in advanced congestive heart failure. *J. Cardiol*., 81(4):443-447, 1998.

[17] Pearch, C.J. and Himsworth, R.L.: Total and free thyroid hormone concentration in patients receiving maintenance replacement treatment with thyroxine. *Brit. Med. J.*, 288: 693-695, 1984.

[18] Burman, K.D. and Wartofsky, L.: Thyroid function in the intensive care unit setting. *Crit. Care Clin*., Jan;17(1):43-57, 2001.

[19] Lowe, J.C.: *The Metabolic Treatment of Fibromyalgia*. Boulder, McDowell Publishing Co., 2000.