An Examination of "The Diagnosis and Management of Primary Hypothyroidism" and other Hypothyroidism Practice Guidelines

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This essay attempts to resolve the dispute between medicine and patients in the diagnosis and management of the symptoms of hypothyroidism. This rather convoluted language is used to open minds to the potential for mimics of hypothyroidism, which become more and more possible and eventually a certitude through the reading of this essay. Studies demonstrate the existence of a problem. A Quality Assessment of Life Years (QALY) demonstrates there is a problem, potentially costly problem. The ubiquitous lack of linguistic clarity in this medical niche is a problem that hides the lack of recognition for the physiological potentials for the causes of this dispute between medicine and unhappy patients. There are numerous publications, too numerous to refute, in medical science journals describing the physiological aspects of these mimics. Further, there are many compelling ethical and indefensible legal reasons for finding a solution to this dispute. Thus, the question has to move from the proscription of triiodothyronine containing hormone replacements to their proper diagnostic and therapeutic prescription. This can be done easily by recognising that medicine is an iterative art. Or the demands of science must be met. Whatever the solution or solutions, they need to be addressed quickly because medical science indicates a depravity: People are suffering needlessly.

This conclusion is driven home with an allegory of a patient with a bewildering infection that was finally resolved with: "When examining the whole patient, examine the ***whole*** patient."

Section 1 – The Existence of a Problem

The recent release of "The Diagnosis and Management of Primary Hypothyroidism" [1] has exacerbated the frustration with other British hypothyroidism guidelines [2,3] and hypothyroidism guidelines in general.[4-9] This frustration was studied [10] and found to exist in 13% of all those being treated for hypothyroidism. They were not satisfied with their treatment. Their symptoms are not being mitigated. This problem is further demonstrated in website polls [11,12] and a physician poll. [13]

Survey Symptom	Thyroid Patient Advocacy Sheila Turner (n=1500) [11]	Thyroid About Mary Shomon (n=907) [12]
Extreme Tiredness / Fatigue	85.9%	91.7%
Weight Gain / Can't Lose It	81.9%	64.5%
Lethargic / Sluggish	72.4%	62.1%
Joint & Muscle Pain	67.0%	50.8%
Sleep Problems	64.5%	46.1%
No Motivation / Depressed	63.4%	45.4%
Cold Intolerance	62.5%	39.7%
Mood Swings	60.3%	45.2%
Lack of Stamina / Weak	56.9%	47.7%
Brittle & Ridged Nails	53.7%	32.4%

Memory Loss / Foggy Mind	53.7%	58.3%
Dry, Sore, Scaly Skin	50.1%	31.3%

However, the existence of a problem is routinely denied or excused by blaming the inadequacies of medicine with “nonspecific symptoms” [14] or by blaming the patient by diagnosing her with “functional somatoform disorders.” [15] These excuses are derived from studies of functional somatic syndromes. [16,17] However, an inspection of these papers reveals no effort to exclude subjects with hypothyroidism. Consequently, the excuses attempt to distance the inadequacies of the medicine of hypothyroidism with data tainted by hypothyroidism. This is not proper logic as it is a form of a circular argument. Additionally, since these claims/diagnoses can not be proven, they must remain as a last resort after all physical potentials have been eliminated via evidenced based medicine and differential diagnostics. [18-22] But these physical potentials are not investigated in spite of medical practice admitted to their existence. [14,23,24] Unfortunately, these potentials are subsequently dismissed without citation. “When examining the whole patient, examine the ***whole*** patient” is an obvious admonition that is being ignored under direction of the endocrinology establishment.

Incredibly, the American Thyroid Association [14] dismisses the need for any triiodothyronine (T3) replacement because the peripheral metabolism is “regulated.” However, since the peripheral metabolism supplies 80% of the T3, it must also operate perfectly in all people during their entire life times. This supremely counter-intuitive statement begs for proof, but none is offered or cited. Similarly, Dr. Garber [23] of the American Association of Clinical Endocrinologists claims that there is no need to assay T3 because it “generally” follows the level of thyroxine (T4). Unfortunately, the satisfaction rate of thyroid therapy is “generally” good [10] but not completely good – not good enough. It is that last 13% that presents the problem. Is there enough T3? Is the T3 being used? Finally, Dr. Gossel’s continuing medical education course describes the history of thyroid related discoveries in Table 1, [24] but then dismisses the post thyroid discoveries of peripheral metabolism and peripheral cellular hormone reception. No diagnostics or therapies are offered for the post thyroid abnormalities, excesses or deficiencies.

Dr. Gossel had reiterated the error that the Joint Committee on Higher Medical Training had produced the “Higher Medical Training Curriculum for Endocrinology and Diabetes Mellitus.” [25] The training of endocrinologists completely ignores the medical science of post thyroid functions and their impact upon symptoms of hypothyroidism. Consequently, in this niche of medicine, physicians are trained to ***not*** examine the whole patient. Consequently, the confusion we know exists in a substantial minority of cases.

Routinely, the low basal temperature measurement, discovered by Dr. Barnes, [26] is dismissed [14] by citing Mackowiak. [27] However, Mackowiak, et al., did not exclude subjects with hypothyroidism. So this interpretation creates another illogical circular argument of attempting to distance the result from hypothyroidism with data tainted by hypothyroidism. And while the low basal temperature measurement does not uniquely indicate hypothyroidism, [26] it might suggest the person is not totally healthy for some reason, but that reason is not sought.

A comparison of medical practice guidelines for hypothyroidism [1-9] with the medical practice guideline for hypothermia [28] creates two comments. The hypothermia guideline [28] provides far more differential diagnostic information and consequently appears to be focusing upon

finding a way to get the patient back to health. The hypothyroidism guidelines [1-9] do not provide such a service. In fact, most of them [2-9] do not suggest that there are any alternative solutions to the symptoms of hypothyroidism. They recommend only levothyroxine sodium. Only the one by the Royal College of Physicians [1] suggests that there might be non-thyroid etiologies. Unfortunately, this guideline also precludes any diagnostics and therapy for non-thyroid etiologies which are post thyroid.

At some point the following quotation by Dr. E. Chester Ridgway must be considered:

> *"T_4 . . . is not the active ingredient. T_3 is the active ingredient and it's the thing that accounts for the thyroid hormone action. As I've been reminded many times, there are no intracellular events that we know that can be described by T_4 at the level of the nucleus. Only T_3. T_4 is not the active compound. Likewise, the site of action is in the nucleus. The site of action is not T_4 in the plasma."*
> – Dr. E. Chester Ridgway [29]

But the hypothyroidism guidelines [1-9] do not address the T3 in the nuclei, where the action is, but T4 in the serum, and usually then only if TSH is high. Thus, the typical assays are merely statistical correlations that can not demand exactitude. However, guidelines in the presence of enforcement are not voluntary. They are effectively mandatory and consequently imply exactitude where there is none. And the most mandatory guideline is the latest by the Royal College of Physicians. [1] These guidelines are mandatory even if they use words like "generally" because of the financial-ethical tradeoff physicians find themselves in. Is it worth the thousands upon thousands to defend the ethical decision to treat a patient properly? Hardly. Guidelines in the presence of enforcement, even merely peer pressure, are mandatory according the logic displayed in American courts. [30-31]

The guidelines [1-9] do have significant flaws. These will be explored further below.

Endnotes Section 1

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Section 2 – Two QALY Views of the Problem

Quality Adjusted Life Years (QALY) is used in the assessment of healthcare interventions. Nominally, it is used to appropriately distribute limited healthcare resources so that these resources have the maximum benefit. [1] What should be inherent in the QALY figure for a proposed intervention is that it should not produce a negative change. A negative change would not allow any monies being spent on that intervention. Thus, such interventions are both financially and ethically contraindicated. Consequently, a QALY view of two cases are particularly interesting:

Patient S has been prescribed Armour for some years. She was prescribed Armour because all of the other therapies relevant to the symptoms of hypothyroidism did not manage her chronic and debilitating symptoms. But Armour did. However, physicians are generally not willing to prescribe any triiodothyronine (T3) containing hormone replacement since they are either not recommended or they are proscribed by guidelines. [2-10] The advent of the Royal College of Physicians guideline "The Diagnosis and Management of Primary Hypothyroidism," [2] has further reduced the numbers of physicians willing to prescribe Armour or any T3 containing hormone replacement with its unquestionable proscription. Nominally, her case as already demonstrated that she does not have primary hypothyroidism, yet statements in the conclusion of this guideline [2] drive physicians to deny new prescriptions for Armour. By her own history,

we know that her QALY figure for denying Armour is negative – her life will become horrible, since Armour is the one therapy that works.

Patient K was examined for thyroid deficiencies more than 40 years ago. She was told that she did not have a thyroid problem – it was just a little low. One day she fainted while walking and broke her leg in two places. Upon examining her, the emergency room physician claimed that he would fix her leg immediately and her "thyroid" the next day. In fact, pictures of Patient K from that time demonstrate the classic appearance [11] of someone quite lacking in thyroid related hormones – she was a textbook example. The physician went on to claim that she was quite lucky that she did break her leg because if she had, she would have died shortly without treatment. Indeed, Patient K was planning her own funeral. Consequently, her pretreatment QALY index was undoubtedly less than 1. The physician prescribed liothyronine sodium (one of the currently banned/boycotted hormone replacements). In retrospect, her post treatment QALY index was greater than 40 as she is still quite alive and in good health. So if the present guidelines were followed, they would have killed her. By doing proscribed diagnostic (observation only) and prescribing one of the proscribed hormone replacements the QALY index went up more than 40 points.

But 25 years later, the physician retired. Patient K was now forced to find another. The physician applied the philosophy of the present guideline, and prescribed levothyroxine sodium. Soon she began to circle life's drain again. But with the knowledge of her prior experience, she searched for a physician who would prescribe liothyronine sodium. Her well being rapidly returned. Once again the proper application of the guideline reduced her QALY index and disobeying it raised her QALY index. Thus, in two series of applications, of the spirit of the subject guideline, actually lowered the QALY index of the patient -- the first time would have, absent the accident, brought death.

There are other QALY considerations, for example: [12-18] Hypothyroidism, and presumably its mimics, produce numerous chronic and repeating health problems [11-20] ranging from reoccurring headaches and colds to life's great killers, heart attacks and diabetes, which significantly impact pre-treatment QALY. Dr. Barnes did a study of his patients' heart attacks [20] and reported that proper treatment with desiccated thyroid reduced heart attacks far below the Framingham study. His study group, treated with this thyroid extract had only four heart attacks. A Framingham group of the same size and duration would have had 72 ([20], page 180). Further, dropouts from the Barnes study had a high heart attack rate.

Unfortunately, contemporary thought dismisses the science of thyroid pioneers because their science was not produced via extremely expensive, competition limiting, randomized, double-blind, placebo-corrected clinical studies. However, Benson and Hartz [21] found expertly performed legacy studies produced quite similar results. Consequently, these pioneers' results regain credence and cannot be summarily dismissed, as they routinely are.

These findings, [11-20] absent therapy, reduces the patient's pre-treatment QALY index, and consequently demands earlier low-cost thyroid hormone replacement in lieu of greater expenditures for treating the myriad of unnecessary disease.

Conclusion: The hypothyroidism guidelines mis-diagnose and mismanage some patients with the symptoms of hypothyroidism. Deficiencies in the greater thyroid realm, particularly in the post thyroid realm, should be more aggressively pursued.

Endnotes Section 2

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Section 3 – The Scope of the Dispute

The scope of this dispute can be found in the difference in the meanings for "hypothyroidism." Knowing the difference in these meanings help to limit the extent of the support of the numerous studies used to support the hypothyroidism guidelines.

The definitions for hypothyroidism generally fall into two groups. [1] The first group focuses upon the deficiency in the secretion by the thyroid gland as the "hypo" prefix and the "ism" suffix imply. The second group focuses upon the deficiency of thyroid related hormones in the blood or body. Logically then, this second group encompasses any somatic function that impacts the thyroid related hormones in the blood or body, not just the thyroid gland. Routinely, the existence and nature of these extra-thyroidal or post thyroidal functions are neither disclosed nor explained. [2,3] That view was reasonable prior to the discovery of thyroxine resistance by

investigators in the 1950's. [4] However, as medical science began unraveling the physiological tangle of thyroxine resistance, they found the answers by 1970 [5] and began characterising the peripheral metabolism and peripheral cellular hormone reception more exactly. [references in 6 for example] Consequently, the two definitions are not equivalent. [1]

The lack of a single definition for "hypothyroidism" and the general, but routinely ignored, demand for clarity, requires the stipulation of its definition. Such stipulations were recognised by 17th Century linguists, are part of the standard of care in legal documents, are a part of required clarity for medical practice guidelines, [7] and specifically required by a protocol for the authorship of guidelines. [8]

> *Careful use of definitions can clear up many disputes arising from the ambiguity of expressions which one disputant takes in one sense, and other in another sense. . . When the idea we want to express by some word is not indicated precisely and clearly enough, it is almost impossible to avoid equivocating in the course of an argument.* – Antioine Arnold – 17th Century

If this linguistic standard of care were met and logical consistency were maintained then patients would not suffer needlessly and physicians would not be prosecuted wrongly. [1] If definition of "hypothyroidism" were stipulated narrowly to the thyroid gland, then the scope or jurisdiction of the guidelines would be limited to the thyroid gland and the physician could then treat the patient ethically for non-thyroid and post-thyroid etiologies. Alternatively, if the definition of "hypothyroidism" were broad and logical consistency maintained, the medical practice guidelines must embrace the post-thyroid etiologies with appropriate diagnostics and maintenance measures. Unfortunately, for both patient and physician, neither is done. This makes the observation by Anthony Toft and Geoffrey Beckett [9] quite reasonable and understandable:

> *"It is extraordinary that more than 100 years since the first description of the treatment of hypothyroidism and the current availability of refined diagnostic tests, debate is continuing about its diagnosis and management."*

Examinations of the guidelines of the Royal College of Physicians [10] and the British Thyroid Association [11] demonstrate two faults. The RCP guideline [10] uses the well defined term "primary hypothyroidism" but then does not maintain logical consistency in the conclusions as it makes proscriptions that exceed the bounds of primary hypothyroidism. On the other hand, the BTA guideline [11] offers the broad definition of "hypothyroidism" but then does not follow through with diagnoses and therapies suitable for the post thyroid functions that are associated with broad definitions of "hypothyroidism." The other guidelines simply do not offer any definition for hypothyroidism, but all focus on the thyroid gland without disclaiming jurisdiction over the post thyroid deficiencies. In all these cases the post thyroid deficient patient is neither diagnosed nor treated properly. They simply continue to suffer needlessly.

These linguistic standards of care impact the interpretation of guideline supporting studies, particularly those that demonstrate the ineffectiveness of the active hormone, triiodothyronine (T3), and support the thyroxine-only therapy for hypothyroidism, for example: [12-15] They suffer from several issues:

1. Many studies were done with subjects that had primary hypothyroidism or had thyroidectomies. These subjects nominally fit the narrow, thyroid-centric definition. The subjects showed little if any benefit with the addition of triiodothyronine (T_3) to their therapy in lieu of some of the thyroxine (T_4). Consequently, these results proved nothing for patients suffering from post-thyroid or exo-endocrine deficiencies. [1]

2. The low occurrence rate of subjects that have post-thyroid or exo-endocrine deficiencies permitted the authors to round off the low rate of improvement to nil and conclude "no improvement."

3. The triiodothyronine doses were quite low, usually about 5 micrograms per day. This dose in a patient that actually suffers from post-thyroid deficiencies did not produce sustained noticeable benefits. Indeed, that dose is less than 5% to 10% of the usual replacement dose of 50 mcg to 100 mcg daily. In fact, 5 micrograms is the recommended ***starting*** dose for more sensitive patients, infants and the elderly. [16]

4. These studies produce the contradictory position that the active hormone, T3, [17] is not effective, while the relatively inactive hormone, T4, [17] is effective.

5. Taking a further contradictory position, the so-called ineffective hormone, T3, is also dangerous. [18]

Additional, but error filled, support for the thyroxine-only therapy may be found in *Wilson's Syndrome*, [18] a position paper by the American Thyroid Association. It claims that no triiodothyronine-containing therapy is needed because the peripheral metabolism or conversion is "regulated." In other words, this peripheral, post thyroid process never fails. This is a substantial statement from allopaths, whose adult lives have been spent in medicine learning how and treating somatic failures. Considering the lack of supporting references and its extremely counter-intuitive nature, this is a substantially misleading statement.

With this destruction of support for the thyroxine-only therapy, hopefully minds will be open to the use of triiodothyronine (T3) containing hormone replacements [19-25] as well as many other references, such as in: [6] There is no doubt that peripheral hormone reception exists. [26-36] Additionally, there is no doubt that the peripheral metabolism of the pro-hormone, thyroxine (T4), to the active hormone, triiodothyronine (T3), exists. [37-43] Further, there are many more references in books, papers, and websites. [6,28,44-46]

Dr. Anthony Toft, a highly respected endocrinologist, resolved the quandary [9] with a philosophical reversal on combination hormone therapies. [47]

> *"It would appear that the treatment of hypothyroidism is about to come full circle. . ."* – Anthony Toft

Conclusion: The reality of medical science and the need for linguistic care has yet to penetrate the hypothyroidism guideline authorship committees. The thyroxine-only therapy is decidedly not applicable to hypothyroidism in its broad meaning and may be questionable in its narrow, thyroid gland oriented meaning. [47,48]

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Section 4 – The Reasons for Resolving this Dispute

There are ethical and legal reasons for resolving this dispute. Both are compelling. There are many statements of medical ethics from several sources in the UK, and the rest of the world. However, the following two should suffice:

> ***Make the Care of Your Patient Your First Concern*** The UK General Medical Council (2006)
>
> ***Provide a Good Standard of Practice and Care. Keep Your Professional Knowledge and Skills up to Date.*** The UK General Medical Council (2006)

The organised lack of training and the enforcement of incomplete diagnostics and treatment proscriptions belies the stated ethic of patient priority. The question of the level of professional knowledge required by the second statement of ethics above has been answered in *Bolitho v City and Hackney Health Authority* [1997] 4 All ER 771:

> In *Bolitho* the court declared that it was not bound to find for a defendant simply because he leads evidence from a body of experts who genuinely believe that the defendant's practice conformed to sound medical practice. The court will require further evidence that the practice proclaimed has a logical basis, and that the defendant practitioner has weighed up the benefits and risks.

From the above discussion, there is no logical basis for a universal thyroxine-only therapy. The thyroxine-only therapy is particularly inappropriate for patients with deficiencies in one or both of the post thyroid functions of peripheral metabolism of thyroxine (T4) to triiodothyronine (T3) or the peripheral cellular hormone reception of T3. There is, on the other hand, a logical basis in medical science for supplying T3 for continuing symptoms of hypothyroidism if one of these deficiencies exists.

The Human Rights Act of 1998 enacts Articles 2 to 12 and 14 of the European Convention on Human Rights. The court in NHS Trust A v. M and NHS Trust B v H [2001] found “a positive obligation to give treatment where that is in the best interests of the patient - but not where it would be futile.” The minimal expense and the high value of all appropriate thyroid related hormone replacements demands this obligation be fulfilled.

Article 3 becomes a requirement for positive action in the lights of (a) the cases of both Patients S and K, (b) the inhuman and degrading treatment indicated in the above cited surveys, and (c) for all those these people they represent. Indeed, more than a third of the respondents [1] felt they had not been dealt with very well or not very well at all. Unquestionably, post thyroid physiology needs to become a part of endocrinology, medical practice guidelines, and a part of the standard of care.

Valid consent is violated by the withholding of pertinent scientific knowledge. Of 1500 patients taking the survey [1] 93.8% had not been informed of any hormone replacement other than levothyroxine sodium. This appears to be an Article 8 violation as well as a tort.

Other potential Article 8 issues taken from the survey [1] are 15.5% had given up employment for lack of appropriate treatment, another 20% were forced to take leave for lack of appropriate treatment, and a third felt their close relationships had been adversely affected.

Since the patient is dependent upon the physician, who, by the enforcement by the General Medical Council, is particularly dependent upon the medical practice guidelines written by professional medical associations, the authoring associations are liable to the patients via vicarious liability for their suffering. [2] Such liabilities are broadened by the ubiquitous tort phrase of "knew or should have known." Obviously, experts in the medicine surrounding the thyroid "knew or should have known" of the medical science of the post thyroid functions – their descriptions have been in medical science literature for decades:

Relevant Milestones in Thyroid Hormone Behavior Studies [3-7]

Circa	Event
1952	Identification of triiodothyronine (T_3), the much more active thyroid-related hormone [Gross & Pitt-Rivers]
1950's	Hypothyroidism-like malady found that only responds to T_3
1967	Identifies patients with resistance to T_4, but respond to T_3 [Refetoff]
1967	Resistance to thyroid hormone reception found [Refetoff, Dewind, & DeGroot]
1970	Evidence that circulating T_3 was derived largely from peripheral monodeiodination (conversion) of T_4 [Braverman, Ingbar, & Sterling]
1972	Identification of T_3_binding receptors in tissue
1990	Demonstrations that point mutations in the thyroid_hormone receptor accounted for hormone resistance

Finally, the general proscription of all triiodothyronine may also be considered an anti-competitive boycott or refusal to deal that inappropriately deprives vulnerable consumers from necessary hormone replacements with a variety of deceptions that distort competition improperly.

Conclusion: Medical ethics and society's expectations demand the proper training and guidance of physicians in the diagnosis and maintenance of post thyroid deficiencies so that the vulnerable patient might lead a reasonable life free of inhumane and degrading treatment.

Endnotes – Section 4

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Section 5 – Peripheral Metabolism Efficiency & Bioequivalency?

This is a note of caution. The variability in the efficiency of peripheral metabolism can affect the alleged bio-equivalency of different classes of hormone replacements and the clinical resolution of this dispute.

First, a counter example to the established paradigm: Two women have opposite characteristics that can only be explained by substantially different efficiencies in the post thyroid realm. The first has an over-suppressed TSH (=.002) but presents no hyperthyroidism characteristics. The second has a high TSH (=60) but presents no hypothyroidism characteristics. These counter examples can not be explained by the endocrinology paradigm. Differences in the efficiencies in the post thyroid functions of peripheral metabolism and peripheral cellular hormone reception must be considered.

The potential differences in post thyroid efficiencies questions bioequivalency of different classes of thyroid hormone replacements, which some sources claim to exist.
If these replacements contain different quantities of thryoxine (T4) and triiodothyronine (T3) they can only be possibly equivalent if with a particular peripheral metabolism conversion efficiency. The present problem, however, considers that the peripheral metabolism conversion efficiency may be different than average. Consequently, these supposed equivalencies can not be considered. Instead, when changing prescriptions, there should be a weaning period followed by the gradual increase of the new replacement.

Section 6 – How to Resolve this Dispute

The resolution to this dispute is simple – consider the whole patient. There are two ways, either by considering medicine as an art or as a science.

Artfully, the physician who did recognise and treat Patient K's symptoms of hypothyroidism did so according to the arts of endocrinology – the visual recognition of the problem, discussing the problem with the patient, and making trial prescriptions. In the light of known post thyroid deficiency potentials, this is better

than halting the diagnostic process with the diagnosis of “functional somatoform disorders.” Obviously, this is not scientific, but medicine is also an art, and this approach has the distinct advantage of being able to be implemented rapidly. Patients are suffering needlessly.

Unfortunately, scientific certainty is not available either. The laboratory assays which focus upon the thyroid gland only, do not produce a scientific image of the condition of the whole of the greater thyroid realm, particularly for the post thyroid functionality. The thyroid stimulating hormone (TSH) assay is at least three operations away from the cells’ nuclei. This separation is created by thyroid secretion, peripheral metabolism, and hormone reception. There are many unknowns along the way – clearance rates, enzyme levels, functional efficiencies etc. Even with the optional additional assay of free T4 (fT4), there are still two intervening operations, peripheral metabolism and peripheral cellular hormone reception.

To make the situation more complex, the peripheral metabolism is also a regulatory function, so its output is not simply a function of its input alone. This regulatory function also isolates, at least partially, the thyroid side of the peripheral metabolism from the reception side. Thus, it is possible to have “normal” thyroid function test results and the symptoms of hypothyroidism simultaneously. Indeed, that was Patient K’s case. Even today’s thyroid function tests would not have found Patient K’s deficiency.

Measuring free T3 (fT3) does not provide the whole picture either because the hormone reception is not simply a function of the T3 level. The receptors can mutate [1] to reduce the reception rate or increase the reception resistance. The tests for measuring the end use of T3 are generally discredited and somewhat non-specific.

However, the regulatory nature of peripheral metabolism gives us some insight to the use of T3 since the metabolism sites produce reverse T3 (rT3) if there is sufficient T3 available. [2] Brady claims that this test provides the needed information when the other tests suggest normality.

Baisier, et al., offer two tests, a clinical evaluation algorithm and a 24-hour urine T3 assay. Good results are claimed for both. [3] However, these results are undoubtedly dismisses as they were not the product of randomized, placebo-corrected, double-blind studies. However, again Benson and Hartz found that well-constructed studies by practicing physicians are reliable. [4]

Obviously, the medical practice guidelines for hypothyroidism require substantial revision.

Endnotes – Section 6

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Section 7 – Conclusion

The endocrinology establishment faces the results of ignoring medical science for the past 40 years and dismissing pioneering results in the unnecessary suffering of a significant minority of patients with the symptoms of hypothyroidism. Substandard medical education and care are covered with linguistic fog and improper excuses. The appropriate technology for scientifically diagnosing patients completely has not yet been accepted, but is needed quickly. Ethics, torts, fair trading, and human rights demand action – immediate action. The evidence for change began appearing a half century ago. The outline of the relevant science was known four decades ago. The time for action began decades ago. People are suffering needlessly. The proper hormone replacements are safe and effective, per the US Food and Drug Administration. They need and deserve relief from their unnecessary miseries. They want and need their lives back. Endocrinology needs to rise to the challenge of caring for the last 13%.

Section 8 – An Applicable Allegory

Medicine By Wishful Thinking

Excerpted from an Article
by Dr. Alan Eshleman, www.SFGate.com

The intern can't figure out why Mrs. Riley has a fever. He's listened to her lungs, her heart, and her belly. He's checked her legs for blood clots. He's sent blood and urine samples to the lab and reviewed all the medications she's taking to see if this could be a drug fever. Ms. Riley's white blood count is high with a significant proportion of immature white cells, one of the hallmarks of infection, but he can't find a source. "Let's go examine her," I say.

Ms. Riley is 87 years old and quite demented. She communicates by grunting or withdrawing in pain. She has lived in a convalescent hospital for the past two years after a stroke rendered her unable to care for herself in her tiny, downtown apartment. She was brought to the hospital late last night by ambulance after the nurses aides at the convalescent hospital found Ms. Riley even more withdrawn than usual and unable to take spoon feeding.

The room reeks of stale urine. There are three other patients in the room, all elderly, demented women. Ms. Riley's bed is closest to the window. A beam of sunlight plays on her blankets. Ms. Riley is thin and tall with unruly black hair shot with gray. The right side of her face droops and her right arm does not move when I take it to feel her pulse. She stares blankly at the ceiling with her mouth open. I go through the formalities of introducing myself, though I may as well be conversing with a box of rocks.

I look at the bedside chart that shows an erratic fluctuation of temperature between 99.5 and 101.6. Her urine draining from a catheter is clear and yellow. I agree with the intern that Ms. Riley's legs and thighs appear normal. I use the controls to raise the back of the bed and with the intern pulling on both of Ms. Riley's wrists slide my stethoscope down the back of her chest. The lungs sound normal meaning pneumonia is unlikely, so I pause to think.

"Did you roll her over?" I ask the intern. He thinks for a second and answers "no." In fact, Ms. Riley has been flat on her back for her entire hospital stay. She came directly from her bed at the convalescent hospital to a gurney in the ambulance to another gurney in the emergency room, and from there was wheeled to a bed on one of the medical floors. So, with Ms. Riley resisting all the way, we roll her from a supine to a prone position and we find our answer: at the base of her spine there is an angry, red ulcer, draining pus, and extending down through fat and muscle to glistening white bone. Ms. Riley has an infected decubitus ulcer. The decubitus ulcer is a common problem among people who are so debilitated that they cannot adjust their own position in bed or in a wheelchair. The weight of the person's body compresses nerves and blood vessels, starving the tissue and causing it to break down. Most decubitus ulcers develop over the lower back, buttocks, hips, and heels.

The take home message (as we are fond of calling it on the wards) is that when you examine the whole patient you should examine the whole patient. Otherwise you will miss something. I let the message sink in without further comment.