

Below is a copy of a covering letter I have today sent to Professor Jayne Franklyn (President of the British Thyroid Association), with the attached paper '*Hypothyroidism Mimics Require Consideration*' by Eric Pritchard (US Researcher). He discusses the mimics of hypothyroidism which must not continue to be ignored. It is extremely important that an urgent reassessment should be made of the recommended TFT Guidelines. I would be very pleased to receive any comments.

Kind regards,

Sheila Turner
Thyroid Patient Advocacy
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Eric Pritchard's paper follows this letter

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29 July 2008

Dear Professor Franklyn,

As you are aware, for the past four years, I have been running the website 'Thyroid Patient Advocacy-UK and alongside, an Internet campaigning and support forum for sufferers of thyroid disease and its mimics - a place where sufferers can discuss their particular thyroid problem and get the help and support they are not receiving within the NHS. During these 4 years, I have done research and gained information from patients, doctors, and various studies, and know that I am putting some of the pieces of the jigsaw into the right spaces and seeing parts of the picture that are giving me insight into some of the reasons why many sufferers are not being given a correct diagnosis, are not being treated with the level or type of thyroid hormone that would give them back their normal health, or are being refused the diagnostics and therapy they need.

The diagnosis and treatment of hypothyroidism should be straight-forward: a few blood tests and visits to a GP, after which many should be helped to regain their normal health. However, other patients and their doctors find it a mystery that is inappropriately excused with "nonspecific symptoms" or "functional somatoform disorders." Amazingly, the symptoms that prompted the assays in the first place are not regarded as sufficient for further investigation - but the mimics of hypothyroidism require consideration too and must not be ignored. Ignoring medical science has produced a 13% failure rate amongst those treated for hypothyroidism as indicated in one study, and yet more, in another.

Is the "ism" in hypothyroidism the deficient secretion by the thyroid gland, or the deficient thyroid hormone levels? If the former, do the guidelines apply to post-thyroid effects on thyroid hormones? And if not, why isn't this stated in the guidelines to eliminate any risk of misinterpretation? If the latter, why do the guidelines recommend diagnostics and therapies that only apply to the thyroid gland, and why don't the guidelines contain any diagnostics and therapies for any post-thyroid operation, which are known to medical science, and have been known for the past 38 years?

The problem appears simple. Hypothyroidism and hypoposthypothyroidism deficiencies are not the same, although they have the same symptoms, and the overarching name that is given to them - hypothyroidism. Medical ethics, the protocol of differential diagnostics, and the regulations upon valid consent require that the T3-requiring post-thyroid deficiencies be considered. They are different physiologically. They occur in different places. The dominant hormone is different. The feedback "mechanisms" are different. The appropriate assays are different.

I am enclosing a paper written by Eric Pritchard, a US researcher, entitled '*Hypothyroidism Mimics Require Consideration*', and I hope that you will be able to devote sufficient time to read it.

Eric Pritchard makes the point that many patients appear to be suffering from "thyroid" deficiencies when, in fact, they are suffering from replaceable post-thyroid hormone deficiencies, i.e., deficiencies of peripheral metabolism of T4 to T3 or the reception and use of T3 by cells and their nuclei.

There are seven arguments for the proper diagnosis and treatment for the other causes that produce symptoms, which could suggest hypothyroidism:

1. Medical science (Braverman, Refetoff et al) discovered the mimics of hypothyroidism around 1970.
2. The TFT guidelines are NOT pertinent to these mimics.
3. The support for thyroxine-only therapy is NOT pertinent to these mimics.
4. The protocol for differential diagnosis requires consideration of known causes with similar symptoms, prior to concluding 'non-specific' symptoms or 'somatoform' disorders.
5. Informed consent for any proposed medical treatment should be a matter for full discussion between doctor and patient, covering all the potential courses of action, not just the preferred therapy.
6. Declarations concerning patient's care, as issued by the General Medical Council, must be strictly adhered to.
7. Ignoring certain elements of proper care is probably a civil rights issue.

Thus, the proper diagnosis and treatment of these mimics of hypothyroidism is paramount. They MUST not continue to be ignored. The needless suffering of undiagnosed, untreated, and undertreated patients must stop.

Please will you help?

Yours sincerely,

Sheila Turner
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Copies to:

- Members of the BTA Executive Committee
- NHS Endocrinologists
- Trustees, British Thyroid Foundation
- Rt Hon Gordon Brown PC MP, Prime Minister
- David Nicholson, NHS Chief Executive
- Sir Liam Donaldson, Chief Medical officer
- Mr Alan Johnson MP, Secretary of State for Health
- Lord Sir Ara Darzi
- Dr Fiona Adshead, Deputy Medical Officer
- Ms Claire Whittington, Head of Long Term Conditions

- Professor Roger Boyle, National Clinical Director – Heart Disease/Stroke
- Professor Christine Beasley, Chief Nursing Officer
- Flora Goldhill, National Director of user Experience and Involvement
- Harry Cayton, National Director Patient and Public Involvement
- Dean and Head of all UK Medical Schools

Hypothyroidism Mimics Require Consideration

by Eric K. Pritchard, MS

Introduction

She wakes tired. Struggles to get up. Dresses slowly. Now exhausted, she falls back into bed. Once again she will have an after-nap breakfast. She wakes for her doctor's appointment. Once again, he claims her tests were normal. Once again she claims she is sick. Once again, he writes the same old prescription, saying that she is really suffering from functional somatoform disorders. Once again, she is reduced to tears. "Why again? Must I suffer more? Why can't you help me?"

Is the diagnosis and treatment of hypothyroidism trivial? Or, is it more complex, even mysterious? For most patients, it is trivial – a few laboratory assays and office visits and they are back to an energetic, more attractive, disease resistant life. The other patients and their physicians find it a mystery that is inappropriately excused with "nonspecific symptoms" or "functional somatoform disorders." Amazingly, the symptoms that prompted the assays are not sufficient for the further investigation demanded by medical science and basic medical protocols, but these mimics of hypothyroidism require consideration too. The ignoring of relevant medical science has produced a 13% failure rate among those treated for hypothyroidism ^[1] and an unknown rate of false negatives among other patients. Another study paints a bleaker picture, see List 1. ^[2] A patient petition demanding better diagnosis and treatment has been signed by just under 900 patients so far. ^[3] Without a doubt, there is a distinct need.

Beyond kindness to fellow humans, there are seven arguments for the proper diagnosis and treatment for the other etiologies that produce the symptoms of hypothyroidism. First, medical science discovered the mimicking etiologies circa 1970 – they exist. Second, the medical practice guidelines are not applicable to these mimics of hypothyroidism, but their lack of clarity suggests that they are and that is their interpretation. Although, the cause of this confusion is proscribed by a guideline authorship protocol, this protocol has been ignored universally. Ultimately, this confusion by the lack of clarity may be settled by the definition of hypothyroidism proffered in patient information by the guideline issuing organizations.

Third, the support for the thyroxine-only therapy will also be clarified with the stipulation of the definition of "hypothyroidism," and consequentially discredited for the mimicking post-thyroid etiologies.

Fourth, the protocol for differential diagnosis requires the consideration of scientifically known etiologies with symptoms similar to the patient's symptoms. However, this protocol was trumped by the pre-1970 paradigm of hypothyroidism, which had become established earlier. It eclipsed the possibility of any mimics, their diagnosis, and their treatment. This improper trumping has subsequently produced the improper diagnostic conclusions of "nonspecific symptoms" and "functional somatoform disorders."

Fifth, informed or valid consent for a proposed medical treatment requires disclosure of all material facts and all of the potential courses of action, not just the preferred course. Without complete diagnostics upon potential etiologies, informed or valid consent is not possible.

Sixth, proper care is required by various statements of medical ethics. Medicine's prime consideration for the patient is too often quite secondary to other considerations. The mimicking etiologies of hypothyroidism consequently go undiagnosed and untreated. The unfortunate patient/victim is consequently cursed with chronic misery. Hopefully, for the patients' sakes, medical ethics will inspire recognition of the science of the mimics of hypothyroidism, peripheral metabolism and peripheral cellular hormone reception, comply with differential diagnostic protocols, informed consent rulings, and treat the mimicking etiologies properly. A physician consensus petition, with 1600 signatories, demanding the right to prescribe the physiologically required therapy. ⁽⁴⁾

Seventh, since the self regulation and patient concern in this realm seems insufficient, the proper care is demanded by civil rights and equal treatment by the law – a concept that is active in the US Constitutional Fifth and Fourteenth Amendments, is active in corresponding British Law, and is quite active in International Law. This notion harks to the days of Henry II and the Magna Carta, 1215.

The History of the Greater Thyroid Realm

The history of the medical science of the greater thyroid realm, both inside and outside of the classic endocrine system may be found in the Milestone List, List 2, below. Prior to the introduction of synthetic thyroxine, levothyroxine sodium, properly treated patients generally did not continue to have symptoms of hypothyroidism. However, with the virtually pure synthetic replacement for thyroxine (T_4), some patients continued to present symptoms of hypothyroidism. Since desiccated thyroid delivers all of the thyroid related hormones, and levothyroxine is a virtually pure thyroxine replacement, the other thyroid related hormones must have been doing something for some patients. Nonetheless, the entrenched hypothyroidism paradigm did and still does not recognize the possibility for any other thyroid related hormone operations between the thyroid and the symptom producing cells. The discoveries by medical science in 1967 and 1970 of resistance to thyroid hormone reception by the peripheral cells and of the peripheral conversion of thyroxine (T_4) to triiodothyronine (T_3) have not changed this paradigm. However, they do explain the continuing symptoms and the need for T_3 -containing hormone replacements.

A sampling of studies provide greater proof of the existence of peripheral conversion⁽¹¹⁻¹²⁾ and peripheral hormone reception resistance.⁽²³⁻²⁸⁾ Other references can be found in these references as well as Dr. Lowe's extensive tome⁽⁸⁾ and other reports on this website.

Thus, without a doubt, there are post-thyroid operations upon thyroid hormones.

The history of the greater thyroid realm also includes successful treatment of the failures of the thyroxine-only therapy paradigm. The empirical study by Dr. W. V. Baisier, et al., *Thyroid Insufficiency? Is Thyroxine the Only Valuable Drug?*⁽²⁹⁾ demonstrates that a T_3 -containing hormone replacement is needed for clinical euthyroidism when T_4 -alone fails to significantly reduce symptom intensity. There is further positive evidence for using T_3 -containing hormone replacements.⁽³⁰⁻³⁴⁾ The explanation for these observations is in the history of thyroid related discoveries in List 2 below and medical studies.^(11, 28) It is simply the matter of replacing the deficient hormone, triiodothyronine, the active hormone.

Please note that there is a significant difference between clinical euthyroidism and chemical euthyroidism just as there is a difference between the broad and narrow definitions of "hypothyroidism." (See the Stipulated Definitions, List 3, below.) Clinical euthyroidism is the absence of the symptoms of thyroid abnormalities and consequently implies that there is no net abnormality from the hypothalamus to the nuclei of the symptom-producing peripheral cells. On the other hand, chemical euthyroidism is demonstrated by the assay of thyroid gland behavior that shows normality of that gland. This difference is the source of substantial confusion noted in the following lament by Anthony Toft and Geoffrey Beckett.⁽³⁵⁾

"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."

Accompanying this confusion is the seemingly unexplainable pair of anecdotes: The first patient's assay of thyroid stimulating hormone (TSH) indicates severe hyperthyroidism, but the patient is clinically euthyroid. The second patient's assay of TSH indicates severe hypothyroidism, but the patient is clinically euthyroid. These examples may be explained by post thyroid deficient and excessive efficiencies, respectively. However and unfortunately, since post-thyroid, exo-endocrine-system etiologies are not considered by the existing paradigm and exiting medical practice guidelines, they generally remain medical mysteries as these examples do.

Medical Practice Guideline Issues

Medical practice guidelines should describe applicable physiology, the symptoms of the etiologies, the applicable differential diagnostics, and the therapy approaches as dictated by the diagnostic results.

Unfortunately, medical guidelines do not live up to their purpose. Studies of medical guidelines⁽³⁶⁻³⁸⁾ give the average guideline a seriously failing grade. Very few are excellent.

A logical examination of a continuing medical education course⁽⁵⁾ and the medical practice guidelines for hypothyroidism⁽³⁹⁻⁴⁶⁾ finds that they are not clear.⁽⁴⁷⁾ They, like virtually all papers relating to hypothyroidism, do not stipulate a definition for “hypothyroidism.” Although the two definitions for hypothyroidism were believed to be equivalent prior to 1967, subsequent medical science has definitively separated them. The two definitions are no longer equivalent:

1. The narrow, thyroid-centric definition is as the “-ism” suffix implies: “The clinical consequences of deficient secretion by the thyroid gland.” This definition suggests potential deficiencies in the thyroid gland (primary hypothyroidism) or a preceding gland, the pituitary (secondary hypothyroidism) or the hypothalamus (tertiary hypothyroidism).
2. The broad, symptom-oriented definition, “The clinical consequences of inadequate levels of thyroid hormones in the body.” (Taber’s Cyclopaedia Medical Dictionary), suggests deficiencies in the hypothalamus-pituitary-thyroid axis *plus* the potential deficiencies in the peripheral metabolism and peripheral hormone reception, which are recognized by science.⁽¹¹⁻²⁸⁾

The usual hypothyroidism guideline draws patients into its protocol by the existence of symptoms, i.e., the broad definition of hypothyroidism. However, these guidelines then diagnose and treat these patients according to the narrow, thyroid-centric definition.^(5, 38-46) For hypothyroidism, the only indicators assayed and considered are the following:

1. Thyroid Stimulating Hormone (TSH or Thyrotropin) – the “input” to the thyroid.
2. Free Thyroxine (fT₄) – the primary “output” of the thyroid.
3. Thyroid antibodies – an indicator of internal thyroid problems.

The dominant post-thyroid hormone, triiodothyronine (T₃), is not assayed. Additionally, an indicator of post-thyroid deficiencies, reverse triiodothyronine (rT₃) is not assayed such as suggested by Brady in Table 2 of *Functional Thyroid Disorders*.⁽⁴⁸⁾ Neither is the T₃ level in a 24-hour urine sample as suggested by Baisier, et al.⁽²⁹⁾ Further, the clinical diagnostic algorithm upon a combination of symptoms by Baisier, et al.,⁽²⁹⁾ is completely ignored – probably because individually the symptoms are “non-specific.”

But then, if the symptoms were sufficient to prompt a testing for hypothyroidism, why are they not sufficient to prompt an investigation for post-thyroid etiologies as well? For patients who are being treated with levothyroxine sodium, why aren’t continuing symptoms sufficient to prompt an investigation for post-thyroid etiologies?

The indicated therapy provided by the medical practice guidelines is simple: “a high-quality brand preparation of levothyroxine.” And “desiccated thyroid hormone, combinations of thyroid hormones, or triiodothyronine should not be used as replacement therapy”.⁽³⁹⁾ In other words, the only viable therapies are the ones that only address thyroid gland secretion deficiencies.

Medical Practice Guideline Repair

A guide for the authorship and “repair” of the medical practice guidelines has been written.⁽⁴⁹⁾ Table 4 of this protocol for authoring medical practice guidelines⁽⁴⁹⁾ demands the stipulation of definitions of words and terms that are “unfamiliar, critical, or subject to misinterpretation.” In other words, the medical practice guidelines for hypothyroidism should have stipulated the definition for “hypothyroidism” because it is both subject to misinterpretation and critical to the guideline even though they use “thyroid” in their title.^(40, 45, 46) Of course, common sense suggests making important matters clear.

Patients with post-thyroid or exo-endocrine deficiencies would be diagnosed and treated properly if the definition of “hypothyroidism” were stipulated and logical consistency maintained in one of two ways.⁽⁴⁷⁾ If

the broad, symptom-oriented definition were stipulated, then the medical practice guideline would need expansion to include the additional diagnostic and treatment protocols. If the narrow, thyroid-centric definition were stipulated, then the medical practice guideline would not be applicable to post-thyroid or exo-endocrine deficiencies.

The patient-physician relationship would then be changed from win-lose to win-win by stipulating either definitio.⁽⁴⁷⁾ If the broad definition were stipulated and logical consistency maintained, then the physician would be guided towards the proper therapy. The patient would recover his/her well-being and the physician would not suffer any liability to the state boards of medicine. If the narrow definition were stipulated, then the post-thyroid deficiencies would not be in the guideline's jurisdiction. The physician, since there is no guideline for post thyroid etiologies, could use his best judgment without undue liability to the state boards of medicine.⁽⁴⁷⁾

However, the choice has already been made. Patient information for hypothyroidism already stipulates the narrow, thyroid-centric definition:

"Hypothyroidism (underactivity of the thyroid gland) occurs when the thyroid gland produces less than the normal amount of thyroid hormone".⁽⁵⁰⁾

Unfortunately, the British patient information⁽⁵¹⁾ uses the broad definition. This reinforces the confusion noted by Toft and Beckett, see quote above⁽³⁵⁾. More unfortunately, because the basic science of post-thyroid operations are ignored^(5,6), all interactions between the post-thyroid realm and the thyroid are also ignored. At a minimum, the guidelines should acknowledge the possibility of this interaction.

Thus, the existing medical practice guidelines should not be used to guide or influence the medical care of the post-thyroid etiologies of deficient peripheral metabolism or deficient peripheral hormone reception. Further, care should be exercised in the use of existing guidelines because post-thyroid behaviors do affect thyroid behavior.

Support for the Thyroxine-Only Therapy for Hypothyroidism Clarified

Numerous studies in support of the thyroxine-only therapy for hypothyroidism have been published⁽⁵²⁻⁵⁵⁾. They suffer from several issues:

1. Many studies were done with subjects that had primary hypothyroidism or had thyroidectomies. These subjects fit the narrow, thyroid-centric definition exactly. The subjects showed little if any benefit with the addition of triiodothyronine (T₃) to their therapy in lieu of some of the thyroxine (T₄). Consequently, these results proved nothing for patients suffering from post-thyroid or exo-endocrine deficiencies.⁽⁴⁷⁾
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 and conclude "no improvement."
3. Anecdotally, 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.⁽⁵⁶⁾

Additional support for the thyroxine-only therapy may be found in *Wilson's Syndrome*,⁽⁵⁶⁾ 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 process never fails. This is a substantial statement from allopaths, whose entire adult lives have concentrated on somatic failures. Considering the lack of supporting references, its counter-intuitive nature, and existing contrary references,^(1, 8, 18, 29-33) This is a substantially misleading statement.

The routine medical excuse for continuing symptoms, i.e. “nonspecific symptoms” is also proffered and supported by referencing Barsky, et al.⁽⁵⁸⁾ Unfortunately, the logic of this situation fails⁽⁴⁷⁾ because the study did not eliminate subjects with hypothyroidism. This circular argument desires to distance hypothyroidism from somatic symptoms tainted by the subjects with hypothyroidism. “Nonspecific symptoms” are further discredited by *Hypothyroidism Presenting with Musculoskeletal Symptoms*.⁽⁵⁹⁾

Similarly, the discrediting of the low basal temperature indicator for hypothyroidism discovered by the hypothyroidism pioneer, Dr. Broda Barnes⁽⁶⁰⁾ shares the same logical problem.⁽⁴⁷⁾ The study of body temperatures⁽⁶¹⁾ neglected to eliminate subjects with hypothyroidism also. Although hypothyroidism is not the only reason for lowered body temperatures, it is the main cause.^(7, Pages 12-17) Thus, it is an indicator that demands further investigation.^(18, Page 884)

Wilson’s Syndrome⁽⁵⁷⁾ also warns of the dangers of T₃ replacements. The half-life of triiodothyronine in the serum is only approximately eight hours and therefore suggests substantial physiological variations. However, the effect of that T₃ lasts much longer – about 50 hours according to Danzi, et al.⁽¹⁶⁾

Similarly, a recent paper⁽⁶²⁾ claims that the “nonspecific” symptoms of *Wilson’s Syndrome*⁽⁵⁶⁾ are “functional somatoform disorders” by citing a study⁽⁶³⁾. Inspection of this study found the same issue as found with Barsky, et al.,⁽⁵⁸⁾ – the subjects with thyroid hormone deficiencies in the greater thyroid realm from the hypothalamus to the nuclei were not excluded. Thus, again, the effort to distance symptoms from hypothyroidism is tainted by subjects with physical deficiencies.

“The only common feature of somatoform disorders is that they show somatic symptoms without an associated general medical condition”.⁽⁶⁴⁾ However, the association to a general medical condition depends “on the state of medical science”.⁽⁶⁵⁾ Unfortunately, it is not medical science that is at fault here. The fault lies with medical practice, which has failed for decades to recognize the relevant medical science, see List 2 and note representative studies.⁽¹¹⁻³⁴⁾ It still fallaciously claims that thyroid assays represent the totality of thyroid hormonal operations from the hypothalamus to the symptom-producing cells.⁽⁶²⁾

There also seems to be an assumption that all of the available T₃ is used by the peripheral cells and hence the danger. However, this, too, is not correct since there is another destination for serum T₃. It also leaves by way of the urinary system through a potentially additional somatic regulatory means. The evidence is simple: Baisier, et al. measure T₃ in 24-hour urine samples for an indication of the need for a desiccated thyroid or a hormone combination therapy.⁽²⁹⁾

Thus, efforts to discredit post-thyroid deficiencies and triiodothyronine (T₃) containing therapies have themselves been discredited.

Differential Diagnosis Requirements

“Differential Diagnosis” is “the distinguishing of a disease or condition from others presenting with similar signs and symptoms” (Merriam-Webster). Obviously, then if hypothyroidism is suspected, the potential of peripheral deficiencies, post-thyroid must be considered also because the symptoms are similar. They are similar because the site of the action is nuclei of the cells and the action is described with triiodothyronine, not thyroxine in the test sample:

“T₄ . . . is not the active ingredient. T₃ 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 T₄ at the level of the nucleus. Only T₃. T₄ is not the active compound. Likewise, the site of action is in the nucleus. The site of action is not T₄ in the plasma.” – Dr. E. Chester Ridgway.⁽⁶⁶⁾

The symptoms are similar because every operation from the hypothalamus to the peripheral cells affects the behavior of the cells. Just like the seemingly ancient, unimproved series-wired Christmas tree lights

will be out if only one bulb is out, the peripheral cells will produce symptoms of deficiency if there is a deficiency anywhere in that path. This is an over simplification because there are many peripheral metabolism sites operating together, but the general concept still holds. Post-thyroid or exo-endocrine deficiencies must be considered.

Thus, and particularly when the symptoms of hypothyroidism are not mitigated by thyroxine-only therapy, post-thyroid etiologies must be considered to satisfy the differential diagnosis protocol.

Implications of Informed Consent

The concept of informed consent is based upon personal autonomy and has grown from litigation. Informed consent was created by its need, see List 1. A recent informed consent case in US law, *Matthies v. Mastromonaco* (160 NJ 26 and affirmed by the New Jersey Supreme Court), produced a ruling that requires the physician to present the material facts so that the patient can make an informed decision even if there is no surgery involved. (For the UK, see the visit www.medicalprotection.org/uk/factsheets/consent-basics for more information and see below for the definition for "consent, valid.") From studying Baisier, et al,⁽²⁹⁾ one can readily imagine that the subjects, who continued to suffer with the symptoms of hypothyroidism in spite of thyroxine replacements, were never allowed an informed or valid decision. Ultimately 40 of the 89 subjects, who were studied over thirteen years, became subjects of a follow up study. Here they recovered their energetic lives by taking the century-old desiccated thyroid hormone replacement. Had informed consent been a part of their lives, they would not have had to suffer so long, so much.

Thus, a question must be asked: How many of the 13% of all those treated for hypothyroidism⁽¹⁾ are also suffering from the lack of informed consent? How many other patients are not informed of potentials for false negative results?⁽²⁾

Medical Ethics

Medical ethics deplores the continuing suffering by the victims of post-thyroid deficiencies imposed by the current medical practice. Patients are supposed to be medicine's first priority:

Make the Care of Your Patient Your First Concern. The UK General Medical Council (2006)

The Health of my Patient Will Be My First Consideration. The Declaration of Geneva (1949, 1968, 1983)

A Physician Shall, While Caring for a Patient, Regard Responsibility to the Patient as Paramount. American Medical Association (2001)

A Physician Shall Owe His/Her Patients Complete Loyalty and all the Scientific Resources Available to Him/Her. World Medical Association (1949, 1968, 1983)

If the patient's well-being is paramount, then the discovery of the etiologies of continuing symptoms should be found according to the protocols of Differential Diagnosis and Informed Consent. And should the etiologies require a triiodothyronine (T₃) containing hormone replacement, it should be prescribed.

Be Honest and Open and Act With Integrity. The UK General Medical Council (2006)

A Physician Shall Be Honest in all Professional Interactions. American Medical Association (2001) Denying further medical investigation by claiming the patient has "non-specific symptoms"⁽⁵⁷⁾ is not honest if post-thyroid, exo-endocrine, peripheral deficiencies have not been investigated as they can be⁽⁸⁾.

^{29, 47)} and should be by the differential diagnostic protocol and informed consent rulings. Similarly, claiming the patient has “functional somatic disorders” ⁽⁶²⁾ and denying further medical investigation is not honest.

Provide a Good Standard of Practice and Care. Keep Your Professional Knowledge and Skills up to Date. The UK General Medical Council (2006)

A Physician Shall Continue to Study, Apply, and Advance Scientific Knowledge, Maintain a Commitment to Medical Education, Make Relevant Information Available to Patients, Colleagues, and the Public. The American Medical Association (2001)

The medical knowledge of post-thyroid deficiencies, peripheral metabolism and peripheral hormone reception, has generally not been applied. The withholding of this information from patients and the public may be the cause of substantial suffering by people being treated for hypothyroidism.⁽¹⁻³⁾ There is no commitment to the medical education of these post-thyroid, exo-endocrine etiologies. In fact, this knowledge is being dismissed or ignored completely.^(5, 6, 39-46, 57)

Medical knowledge gained by long-term observational studies are routinely ignored in favor of the gold-standard, double-blind, randomized, placebo-corrected trials. In areas that cannot demand the great investment of such trials, observational studies should be considered because they do get approximately the same result.⁽⁶⁷⁾ Ignoring history for the sake of the trial ideal is quite wasteful of life-long dedication ^(7, 8, 60) and the well-being that it produced and can continue to produce.⁽²⁹⁾

A Physician Shall Act Only in the Patient's Interest When Providing Medical Care Which Might Have the Effect of Weakening the Physical and Mental Condition of the Patient. World Medical Association (1949, 1968, 1983)

Prescribing a thyroxine replacement (levothyroxine sodium) for a patient who is suffering from post-thyroid, exo-endocrine deficiencies does not produce well-being. The post-thyroid deficiencies in the peripheral metabolism of thyroxine (T_4) to triiodothyronine (T_3) and/or in the peripheral cellular reception of T_3 work against the somatic use of the exogenous thyroxine. The patient's suffering continues essentially unmitigated. Consequently, the patient's increased susceptibility to life's great killers ⁽⁶⁸⁻⁷⁴⁾ is not reduced.

Barnes reported in his 1976 book ⁽⁶⁰⁾ 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.^(60-Page 180) Further, dropouts from the Barnes study had a high heart attack rate.

Danzi and Klein ⁽¹⁶⁾ not only verify the cellular need for triiodothyronine (T_3), but also discuss the beneficial regulation of cardiac genes and vasculature.

The adverse effects of hypothyroidism are not limited to the heart.^(7, 60) The effects are quite widespread and documented. The proper treatment of hypothyroidism (broad definition) is long overdue.

Thus, the lack of proper ethics that needlessly maintains chronic suffering demands immediate repair. People should not be doomed to decades of needless suffering that the thyroxine-resistant patients have endured when their proper hormone supplement is indicated and approved by the Food and Drug Administration and is available at the local pharmacy.

Civil Rights

Although US government insured rights do not apply to actions by private individuals, there is an exception for private acts that are done in a symbiotic relationship with the government.^(75, 76)

Undoubtedly, this view is expressed in other countries as well. The major governmental encouragement surrounding boards of medicine, medical councils, etc. is the federal Health Care Quality Improvement Act and the National Practitioner Data Bank. These symbiotic relationships give private acts in the medical arena the legal status of state action. As such, they become subject to various quite prevalent civil rights protections that originated with the Magna Carta. Besides the fairness demanded by due process, there are Equal Treatment and Overinclusion. Presumably, other countries have similar provisions.

Unequal Treatment by Enforcement of Hypothyroidism Guidelines

Hormone replacement rests on a simple concept: repletion of the deficient hormone. This is done for a long list of hormones with the exception of triiodothyronine (T₃) for post-thyroid deficiencies. So in the class of people having deficiencies in endogenously produced hormones, those with post-thyroid deficiencies are not given the deficient hormone, but a different hormone, thyroxine, via levothyroxine sodium.

Overinclusion by Enforcement of Hypothyroidism Guidelines

When we logically analyze the medical guideline directed action upon victims of post-thyroid deficiencies (a.k.a. exo-endocrine deficiencies), we see an undesirable result. Patients with physiologically different etiologies are diagnosed and treated alike. The post-thyroid deficiencies are not assayed and are not treated. In general, the patients with post-thyroid deficiencies then continue to suffer chronically.

In view of the state enforcement of medical practice guidelines, the like treatment of dissimilar groups that causes undue suffering in one of the groups is the Constitutional issue of Overinclusion (see definitions below). In US law, this is a derivative of the Equal Protection Clauses of the Fifth and Fourteenth Amendments, predominately the latter.^(47, 77)

Conclusions

1. Medical science circa 1970 discovered somatic operations upon "thyroid hormones." The thyroid's relatively inactive hormone thyroxine (T₄) is metabolized by the removal of a certain iodine atom to produce the active hormone triiodothyronine (T₃). This triiodothyronine is then received by the peripheral cells via hormone receptors. The cellular behavior is related more closely with the T₃ in the cells' nuclei, than with T₄ in the serum.
2. The medical language has not been updated as a result of the discoveries of potential post thyroid etiologies. The once believed identical meanings of the definitions of hypothyroidism are no longer identical. They are different.
3. The medical language used in medical practice guidelines needs to be clarified so that patients are properly diagnosed and treated. The proper definition for "hypothyroidism" has been proffered by medical associations in their patient information. It is the narrow, thyroid-centric definition. For example, "Hypothyroidism (underactivity of the thyroid gland) occurs when the thyroid gland produces less than the normal amount of thyroid hormone."
4. Since medicine knows of these post-thyroid or exo-endocrine mimics of hypothyroidism, the differential diagnosis protocol demands that they be considered when hypothyroidism is being considered. In other words, patients with normal thyroid assays should be tested for post thyroid deficiencies before claiming "nonspecific symptoms" or "functional somatoform disorders."

5. Informed Consent requires the disclosure of all pertinent and material facts to the patient so that the patient can make an informed decision. This would include information on the post-thyroid deficiencies when hypothyroidism is being considered.
6. Medical ethics require the following:
 - Making the patient's well-being the physician's primary consideration.
 - Honesty.
 - Continuing study, application, and advancement of scientific knowledge.
 - Making relevant information available to patients.
 - Acting in the patient's best interest.
7. The post-thyroid deficient patient may have his civil rights violated if she/he is not diagnosed and treated properly. Indeed, this should be a violation if there is any justice for these people suffering quite needlessly.
8. Thus, medicine has effectively no choice but to properly diagnose and properly treat the post-thyroid deficiencies in the peripheral metabolism of thyroxine (T₄) to triiodothyronine (T₃) and in the peripheral cellular reception of triiodothyronine (T₃).

Thyroid Patient Advocacy Survey Results – List 1 [2]

Percentage of Responses	Description
93.8%	Were not given a complete disclosure of their treatment options. In fact, only levothyroxine sodium was disclosed.
38.8%	Were not satisfied with their interaction with their physician.
15.5%	Had resigned from their employment.
20.0%	Had taken time off from work as a result of their “thyroid” illness.
33.3%	Believed that their relationships had suffered as a result of their “thyroid” illness
42.1%	Had reduced or stopped exercising.
78.4%	Believe that they have not regained their optimal state of health.

Milestones in Thyroid Hormone Behavior Studies, List 2 [5-10]

Circa	Event
1786	Association between hyperthyroid state and changes in heart and eyes noted
1820	Iodide therapy used in Europe to treat goiters [Marine & Kimball]
1871	Cretinism described
1874	Myxedema (Gull's disease) described [Gull]
1883	Myxedema discovered after thyroidectomy [Kocher]
1891	Thyroid extract therapy for myxedema [Murray]
1892	Oral administration of fresh thyroid gland and thyroid gland extract was found to be effective in mitigating hypothyroidism [Fox and MacKenzie]
1895	Effect of thyroid on controlling metabolic rate discovered
1912	Hashimoto's disease described
1914	Thyroid hormone discovered and crystallized [Kendall]
1926	Structure determination of thyroxine (T ₄) [Harrington]

1952	Identification of triiodothyronine (T ₃), the much more active thyroid-related hormone [Gross & Pitt-Rivers] <i>The thyroid-centric hypothyroidism paradigm became entrenched.</i>
1950's	Hypothyroidism-like malady that only responds to T ₃ First synthetic thyroxine, Synthroid®, marketed without patent protection. <i>Some patients using synthetic thyroxine continue to exhibit symptoms in spite of being assayed as "normal."</i>
1958	
1963	Beginning of efforts to discredit desiccated thyroid and all T ₃ -containing hormone replacements. Faux desiccated thyroid containing no thyroid hormone but only iodine (the object of the indirect quality test) were placed into the medication supply train to encourage the prescription of levothyroxine sodium and other synthetics. [9] Desiccated thyroid never recovered from this hoax.
1963	Thyrotropin (TSH) purified
1960's	Thyrotropin (thyroid stimulating hormone) assay developed [Utiger & Odell]
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] <i>Medical science now knows that there are post-thyroid causes of hypothyroidism – but they are ignored in practice – medical science is trumped by the established hypothyroidism paradigm.</i>
1971	Thyrotropin immunoassays for diagnosis of hypothyroidism
1972	Identification of T ₃ -binding receptors in tissue
1990	Demonstrations that point mutations in the thyroid_hormone receptor accounted for hormone resistance

Stipulated Definitions, List 3

Chemically Euthyroid	Describes the laboratory assay of thyroid gland activity that is within normal ranges.
Clinically Euthyroid	Describes the clinical presentation of a patient which has no symptoms of hyperthyroidism or hypothyroidism. Note: since the discovery of potential post thyroid etiologies, this definition is not the same as Chemically Euthyroid.
Consent, Informed	The consent for medical therapy, surgical or non-evasive, that, for the autonomy of the patient, demands complete disclosure of material facts relating to the patient's condition. (US law: Matthias v. Mastromonaco, 160 NJ 26 and affirmed by New Jersey Supreme Court)
Consent, Valid	The patient must have sufficient information to make a choice. Without adequate information, patients are unable to make decisions about their treatment. The General Medical Council and the courts expect patients to be given all information material to their decision, with the proviso that it would not cause the patient serious harm. Further, the patient must be able to give his/her consent freely. Pressuring patients into consenting to treatment invalidates the consent. Visit this site for more information: (www.medicalprotection.org/uk/factsheets/consent-basics)
Differential Diagnosis	The distinguishing of a disease or condition from others presenting with similar signs and symptoms. – Merriam-Webster
Endo-endocrine	Pertaining to the classical endocrine system, i.e. the glands that secrete into the blood stream: the hypothalamus, the pituitary, the thyroid, the parathyroid glands, the islets of the pancreas, the adrenal glands, the testes, and the ovaries.

Exo-endocrine	Pertaining that which is not within the classical endocrine system, also described as peripheral.
Hypothyroidism, Symptom-Oriented	The clinical consequences of inadequate levels of thyroid hormone in the body. (Tabor's Cyclopedic Medical Dictionary). This definition was thought to be equivalent to the thyroid-centric definition, but basic medical science has made them different. It confusingly relates to the greater thyroid realm from the hypothalamus to the nuclei of the peripheral cells.
Hypothyroidism, Thyroid-centric	The clinical consequences of inadequate secretion by the thyroid gland. "Hypothyroidism (underactivity of the thyroid gland) occurs when the thyroid gland produces less than the normal amount of thyroid hormone." [49]
Overinclusion	Is the treatment of two different groups similarly in spite of such treatment producing a significant burden to one group. This concept was derived from the Equal Protection Clause of the Fourteenth Amendment to the US Constitution
Peripheral Hormone Reception	The means for a cell to accept a certain hormone into the cell for use by the cell. There are hormone receptors at the receiving end of every hormonal communication that goes through the serum. The Peripheral Hormone Receivers accept triiodothyronine (T ₃) for use by the cells nuclei.
Peripheral Metabolism	Peripheral Conversion. The somatic chemical operation that removes a particular iodine atom from a thyroxine molecule to produce triiodothyronine (T ₃) and a different particular iodine atom to produce reverse triiodothyronine (rT ₃).
Post-Thyroid	Refers to the Peripheral Metabolism and Peripheral Hormone Reception
Thyroid	Thyroid may be narrowly specific when it refers to the thyroid gland. Unfortunately it can also be broadly vague, as commonly used, when it refers to the greater thyroid realm from the hypothalamus to the nuclei of the peripheral cells.
Thyroid hormone	A term for any hormone in the thyronine class. This term can be misleading since the individual hormones, T ₁ through T ₄ , rT ₃ , TRH, and TSH are physically different and have different properties particularly in their somatic interactions. For example, T ₄ is the semi-active prohormone. T ₃ is the active hormone. RT ₃ is the inactive alternative product of peripheral metabolism. TRH communicates from the hypothalamus to the pituitary and TSH communicates from the pituitary to the thyroid.
Thyroxine-Resistant	The lack or reduction of response to thyroxine, endogenous or exogenous

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About the Author

Eric K. Pritchard has degrees in mathematics and electrical engineering and more than forty years of experience in these fields. In addition to a published medical paper, he has thirty-nine U.S. Patents and many foreign in seven disciplines. After researching and designing circuits to operate through a strategic nuclear threat, he began researching and designing solutions for specialized production problems. The "impossible" task of translating the artistry of vacuum tube amplifiers to solid state, a task that many had tried and all failed, intrigued him and ultimately demanded a reinspection of engineering theory.

According to Pritchard, "Like medicine, engineering predominately fails to maintain a tightly logical connection with firm knowledge. Unlike mathematics, both make sweeping assumptions and approximations, use imprecise arguments, and readily excuse failures. Although failures in engineering usually only cost money, failures in medicine cause or extend pain and suffering and can cause or hasten death."

Mr. Pritchard began studying the etiologies of and treatments for hypothyroidism after his wife recounted to him visits with physicians that suggested "an errant philosophical structure in the diagnosis and treatment of hypothyroidism." He writes, "This suggestion was made by the excessive dependence upon the 'objective' laboratory assay and the total depreciation of the 'subjective' clinical presentation. The laboratory assay claimed euthyroidism while the clinical presentation disagreed completely. From earlier research in audio, where the objective and subjective clash also and where the 'objective' is overly glorified and the "subjective" is under

appreciated, the existence of an errant technical assumption was probably being hidden by the excessive valuation of 'objective' testing. Just as the audio paradigm assumes that the human hearing process does not produce harmonics of audible tones, the endocrine paradigm assumes that all somatic operations on 'thyroid' hormones outside of the classical endocrine system, if they exist, are infallible. Just as the audio paradigm made the Total Harmonic Distortion test and the Intermodulation test the gold standard of objectivity, the endocrine paradigm made the thyroid-stimulating hormone (TSH) test its gold standard."

Pritchard poignantly concludes: "The difference between these situations is substantial. The audio debate does not adversely affect health."

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