The Linguistic Etiologies of Thyroxine-Resistant Hypothyroidism

by Eric K. Pritchard

Abstract

The thyroxine resistant victims of hypothyroidism are not suffering because there is no treatment available – the Food and Drug Administration approved and indicated them long ago. These victims are suffering because the proper treatments are not considered – linguistic etiologies keep the science of exo-endocrine (outside of the endocrine system) hypothyroidism beyond the reach of the practicing physician with the confusion of overinclusion (identical treatment of two classes that burdens one excessively). This confusion begins with two definitions for "hypothyroidism," thyroid-centric and symptom-oriented, which are related to thyroxine (T4) and triiodothyronine (T3) respectively. Although the hypothyroidism paradigm concentrates upon the thyroid and thyroxine, the active hormone in the tissue cells is triiodothyronine. The claimed intimate relationship between inadequate thyroid secretion and the symptoms of hypothyroidism has been distanced by scientific discoveries of exo-endocrine hormone operations. Symptoms don't imply endo-endocrine (within the endocrine system) etiologies only.

The confusion of overinclusion hides the exo-endocrine hormone operations, their potential for failure, and their importance. Different hormones and etiologies demand different diagnostics and hormone supplements.

The confusion of overinclusion continues in the coverup for thyroxine-resistant treatment failures. Counter intuitively and contrary to research, laboratory assays and the exo-endocrine hormone operations are declared infallible. Fallacious logic reassigns continuing symptoms to "normal" somatic etiologies. Definition confusion allows endo-endocrine test results to be erroneously applied to exo-endocrine etiologies.

Baisier, et al., published an empirical study of the thyroxine resistant and found success in using an indicated, FDA approved, and available therapy. These sufferers need the resolution of this confusion so they can regain their lives. The linguistic etiologies must be eliminated so that people needing different treatments are treated differently and properly.

Keywords: hypothyroidism, exo-endocrine, thyroxine-resistant, Baisier, continuing symptoms, basal temperature, nonspecific symptoms, combination therapy, guideline, overinclusion

The Linguistic Etiologies of Thyroxine-Resistant Hypothyroidism

by Eric K. Pritchard

The victims of hypothyroidism who are thyroxine resistant are not suffering because there is no treatment available – the Food and Drug Administration approved and indicated them long ago. These victims are suffering because the proper treatments are proscribed by the endocrine establishment, its paradigm, and its treatment guideline. Consequently, they are rarely considered. The proper treatments are not considered because linguistic etiologies keep the "medical science" of exo-endocrine hypothyroidism (hypothyroidism caused by etiologies outside of the endocrine system) beyond the rationale of the practicing physician by improperly (over) including them with endo-endocrine hypothyroidism (hypothyroidism caused by etiologies within the endocrine system). The first etiology is in the title: "Thyroxine-Resistant Hypothyroidism." The second is in the description: "exo-endocrine hypothyroidism." The endocrine establishment would claim that both are oxymorons. But are they? That depends upon the definition of "hypothyroidism." Is it thyroid-centric or symptom-oriented? This essay explores the consequences of these choices in the light of the physiology to show that the thyroid-centric is only applicable to the recognized class of endo-endocrine hypothyroidism, while the symptom-oriented definition is applicable to both endo-endocrine hypothyroidism and the unrecognized class of exo-endocrine hypothyroidism.

Patient suffering should be sufficient to demand the linguistic treatment of these etiologies. But it has not. Many respondents to a minuscule outreach effort [32] have been suffering for decades. However, since medical guidelines are used by state boards of medicine to regulate, discipline, and prosecute physicians, the notion of Overinclusion, a derivative of the Equal Protection Clause of the Fourteenth Amendment and the antithesis of Due Process, appears to be applicable and compelling. [see the definitions below]

The physiology of hypothyroidism begins within the endocrine system (the hypothalamus, the pituitary, and the thyroid) continues through the exo-endocrine peripheral metabolism sites in various organs (predominately, the liver) to the hormone receptors of the peripheral cells, where the hormones are used and the symptoms begin to be sensed. Certainly, symptoms are not produced directly by any hormone when in the serum. Since the assays are serum based, they are indirect measurements. The subsequent conclusions, then, depend upon inferences and correlations.

The history of hypothyroidism begins after the discovery of hyperthyroidism with the discovery of thyroid extract in 1891, the realization that the thyroid controlled the metabolic rate in 1895, and Hashimoto's disease in 1912. The structure of thyroxine (T4) was discovered in 1926 and triiodothyronine (T3) was identified in 1952. Thus, by the time that the exo-endocrine peripheral conversion of T4 to T3 was discovered in 1970, sufficient time had passed for the hypothyroidism paradigm to form and solidify. This paradigm includes the rigid belief that there was an intimate connection between the thyroid secretion and symptoms. Thus, all hypothyroidism etiologies belong to the same class. However, the thyroid and symptoms were separated by the discoveries, in 1967 and 1970, of two intervening hormone operations, the exo-

endocrine peripheral conversion of T4 to T3 and the identification of T3-binding receptors in tissue. Then in 1989 and 1990, mutations in the thyroid-hormone receptor accounted for increasing hormone reception resistance. [1] Thus, there are exo-endocrine etiologies for the symptoms of hypothyroidism. Unfortunately, they are substantially ignored and hypothyroidism remained singularly classed in spite of significantly different etiologies, different diagnostics, and different hormone supplements.

Multiple, Undeclared Definitions for Hypothyroidism

The inevitable tension created by the peripheral conversion and subsequent exo-endocrine discoveries are reflected in the once believed similar, but now distinct definitions for "hypothyroidism." The thyroid-centric definition is quite similar to the definition of "hypothyroid": "The clinical consequences of inadequate secretion of hormones by the thyroid." The symptom-oriented definition removes the intimate connection between the thyroid gland and symptoms: "The clinical consequences of inadequate levels of thyroid hormones in the body." (Taber's Cyclopaedia Medical Dictionary) And that definition may be reasonably and should be extended to "The clinical consequences of inadequate ***usage*** of thyroid hormones by the body" – particularly in the light of Baisier, et al., finding that a 24-hour urine diagnostic correlates best [2] or Brady's preference for the rT3 test. [3]

A fallacious attempt was made to mitigate this tension by contextually assuming the infallibility of exo-endocrine hormone operations. [4] However, not only is this somatic infallibility quite counter intuitive, but pre-dating research [5, 6] disputes this contextually alleged perfection. The tension is valid since the definitions have different associations. The thyroid-centric definition, being similar to "hypothyroid," is more related to the secretion of thyroxine (T4). On the other hand, the symptom-oriented definition is more related to the availability or usage of triiodothyronine (T3) as suggested by the following quotation [7]:

> *"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

As a consequence of no intracellular events being directly related to thyroxine, the implications of the apparently similar definitions are significant. The thyroid-centric definition ignores the potential of exo-endocrine etiologies while the symptom-oriented definition is inclusive of both endo- and exo-endocrine etiologies. Thus, the following lament by Anthony Taft and Geoffrey Beckett [8] is, then, quite 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."*

This lament is even more understandable by the author's research of dictionary definitions of "hypothyroidism." There were 29 citations for the thyroid-centric version and 22 citations of the symptom-oriented. However, to seemingly sustain the confusion, 11 of each were cited simultaneously. Confusion reigns.

Overinclusion reigns. Since the dominate endo-endocrine hormone is thyroxine (T4) and the dominate exo-endocrine hormone is triiodothyronine (T3) and since their etiologies are different, the treatment of these etiologies are different, these forms of hypothyroidism should be divided into two classes: endo-endocrine and exo-endocrine hypothyroidism. There is precedent for this concept, type 1 diabetes which suffers from the lack of insulin and type 2 which suffers from the inability to use it. This is quite similar to endo-endocrine hypothyroidism not producing sufficient thyroxine and exo-endocrine hypothyroidism not being able to use it by deficient peripheral metabolism or deficient hormone reception.

Then, this dense fog of overinclusion confusion is maintained. Inspect the only closest applicable guideline. [9] The choice of definitions for "hypothyroidism" is not indicated (or stipulated, see definitions), but the text implies both. The guideline description of symptoms implies the broad, symptom-oriented definition. But, the limited diagnostics and treatments in the guideline imply the narrow thyroid-centric definition. If the practicing physician has a thyroxine-resistant patient with persistent symptoms of hypothyroidism in spite of thyroxine treatment and euthyroid-indicating serum levels, the physician is left in a quandary. There is no other recommended treatment. This leaves the physician with two choices:

1. Adapt the standard practice excuses provided by the admonition against any treatment containing T3 [4, 9], deny the existence of continuing hypothyroidism, and deny the potential existence of a second class of hypothyroidism – exo-endocrine hypothyroidism. In this way, the physician avoids the potential wrath of a board of medicine inquiry. The physician wins, but the suffering patient loses.

2. Recognize that the excuses for thyroxine-only treatment failures are fallacious, exercise the treatment disclaimer ("In general ...," page 6, left-hand column of [9]) and the completeness disclaimer at the end of the guideline (page 9 of [9]), and successfully treat the patient with a hormone replacement that is specifically discouraged and proscribed, but is applicable to a second class of hypothyroidism – exo-endocrine hypothyroidism. While the patient wins, the physician loses to the liability of the draconian wrath of a board of medicine for unnecessarily exceeding the recommended treatment (as measured by inconclusive diagnostics, see below) and endangering the patient since they believe that there is only one class of hypothyroidism victims.

Linguistic etiologies create this quite unprofessional win-lose situation. First, the guideline [9] does not specify or stipulate which definition is operative. If the definition were specified, as required by protocol [27], the patient-physician relationship would become win-win for either of the following two reasons:

1. If the thyroid-centric endo-endocrine-only definition were specified in the guideline [9],

then the physician and the board of medicine would realize that the guideline did not apply to the thyroxine resistant, exo-endocrine patients. The physician could then treat the patient properly without fear of fallacious hypothyroidism guideline action by a board of medicine.

2. If the symptom-oriented definition were specified in the guideline [9] and the guideline maintained logical consistency, then the physician would be appropriately guided by an expanded and revised guideline that addressed both classes of hypothyroidism. The patient would be treated properly without fear of fallacious action by a board of medicine.

Logical Faux Pas

The lack of definition for hypothyroidism and the lack of recognition of a second class of hypothyroidism are hardly the last linguistic faux pas in this realm. For example, the guideline (the lower, right-hand column of page 6 of [9]) refers to a dispute between studies of combinations of thyroid hormones. Some studies [10-13] take advantage of the lack of definition of "hypothyroidism," to form the studies and then to make overly broad conclusions or inspire overly broad interpretations. Using subjects with primary hypothyroidism is not representative of subjects with deficient peripheral metabolisms or increased peripheral hormone receptor resistance because they are physiologically different. Concluding that a T3-containing treatment is of no use is not proper when only most showed no improvement. The difference between "all" and "most" is "rare" or "few," precisely the occurrence rate of thyroxine-resistant, exo-endocrine hypothyroidism, the other class of hypothyroidism victims. Thus, in either case, professionalism and patients are not served properly.

If the definitions were known, then those studies could be properly categorized according to the classes of hypothyroidism. The detractions to combination hormone treatments are formulated under a narrow definition of hypothyroidism, endo-endocrine hypothyroidism, and their conclusions must be so treated and limited. The proponents of combination hormone treatments [14-18] are formulated under a broad definition of hypothyroidism and their conclusions must be examined in a different, inclusive light.

For example, *Thyroid Insufficiency? Is Thyroxine the Only Valuable Drug?* [2] primarily deals with the thyroxine-resistant patient, i.e., those that fall within the broad, symptom-oriented definition of hypothyroidism but that fall outside of the narrow, thyroid-centric definition, i.e., exo-endocrine hypothyroidism. As a consequence, the conclusion of successful treatment for the thyroxine-resistant patients should only apply to the thyroxine-resistant, although this particular study also treated undelineated patients in the same way with success. Baisier, et al., had success with both classes of hypothyroidism because they used natural desiccated thyroid, which does address both classes of hypothyroidism.

In spite of the success by Baisier, et al. [2] in diagnosing and treating thyroxine-resistant patients, the expression of the paradigm [4], made the errant assumption that no one could be thyroxine resistant because the peripheral metabolism was "regulated" and contextually infallible. Although this "regulation" was refuted by counter examples in the literature [2 (by

inference), 5, 6], these counter examples are simply ignored. They are ignored because it is human nature to preserve paradigms. [19] People want the consistency of prior decisions, i.e., *starie decisis*.

The expression of the hypothyroidism paradigm [4] also takes advantage of other logical faux pas to cover up or explain away the failures of thyroxine (T4) only therapies. The first cover up discredited the basal temperature diagnostic found by Barnes [14] and used by many others. However, this was falsely discredited by citing a study by Mackowiak. [20] Mackowiak had not excluded subjects with hypothyroidism. Thus, the lower basal temperature diagnostic was attempted to be dissociated from hypothyroidism with a conclusion influenced by hypothyroidism. This logical faux pas is a form of a circular argument and consequently proves nothing.

Similarly, the paradigm expression [4] attempts to reassign somatic symptoms when the thyroxine treatment for hypothyroidism fails by describing them as "nonspecific." This attempt cited Barsky [21] who also did not exclude subjects with hypothyroidism and consequently used a conclusion influenced by hypothyroidism to dissociate a connection with hypothyroidism. Additionally, Baisier, et al., [2] presents a counterexample in the form of an accurate somatic diagnostic technique based upon a combination of coarse evaluations of each of eight somatic symptoms. First, this clinical evaluation values each of the eight symptoms with 0, 1, or 2 for none, some, and substantial presence. Second, these valuations are summed for an overall value between 0 and 16 inclusive. Finally, the sum of these eight clinical evaluations is compared to limits. The ranges of values for euthroid subjects and those with hypothyroidism are substantially separated. Therefore, symptoms, although in combination, can successfully diagnose hypothyroidism – a substantial counter-example to the hypothyroidism paradigm.

Thus, the establishment deflection of blame for the failures of the single-class, thyroxine-only prescription [9] is both illogical and disproved.

Further, neither the guideline [9] nor the paradigm expression [4] acknowledges the existence of any syndrome of the resistance to thyroid hormones [22, 23] or its potential for treatment. [24] There are hormone receptors everywhere. Any abnormality in the reception at the glands or organs is indistinguishable from immediately subsequent hormonal operation abnormalities. However, in the peripheral cells, the abnormality is the basic operation of the cell and the creation of symptoms.

Medical Failures Hidden by the Corruption of Evidence-Based Medicine

The failure of the thyroxine-only treatment prescription is made more confusing by enshrining its short comings in the gold standard of a laboratory assay protocol. This corrupts the vaunted Evidence-Based Medicine protocol. [25] Such a corruption is promoted by the now-discredited, mind-distrusting Behaviorist philosophy [26] and ignores the "checks and balances" counsel of governance of the great James Madison:

"*That if all men were angels, no government would be necessary. If angels were*

to govern men, neither external nor internal controls on government would be necessary. In framing a government which is to be administered by men over men, the great difficulty lies in this: you must first enable the government to control the governed and, in the next place, oblige the government to control itself." – The Federalist Papers

This corruption of Evidence-Based Medicine defies the non-angelic fallibility of mankind as it assumes that every element of the test from its creation and the establishment of normality to the delivery of test data is always perfect. The excessive faith in this illogical corruption is a substantial component in the systemic maltreatment of the victims of thyroxine-resistant, exo-endocrine hypothyroidism. It improperly keeps physicians from listening to their patients and considering the validity of their symptoms. It improperly claims people do not have "thyroid" hormone issues when it does not measure the results of where the action is [7]: in the *intracellular* spaces or any result or indication thereof.

Counter Examples

The author knows of two people presenting counterexamples to the thyroid-stimulating hormone concepts:

1. A subject with an extreme hyperthyroidism indicating TSH level of .002 exhibits clinical hypothyroidism.
2. A subject with a substantial hypothyroidism indicating TSH level of 60 exhibits clinical hyperthyroidism.

The thyroid paradigm cannot explain these characteristics, however, deficiencies and excesses in the exo-endocrine behaviors can. The failure of the thyroid-stimulating hormone paradigm to explain these counterexamples demands consideration of a second class of "thyroid" etiologies – exo-endocrine etiologies.

The Etiologies of Linguistic Etiologies – Non-Compliant Guidelines

The etiologies of linguistic imprecision stem from the apparent assumption that the problem does not exist. The immediate guide to medical authors, *The Manual of Style*, produced by the American Medical Association, does not mention the concept beyond a single sentence admonition for clarity. It spends scores of pages upon abbreviations and statistics but only one sentence for clarity. Unfortunately, *The Manual of Style* is not alone. Other such manuals are also quite lacking in lessons of clarity. More unfortunately, the etiologies of vagueness are generally not explored in the classroom. Somehow, clarity is supposed to arise in the mind like wild flowers in the spring – naturally.

The protocols for the authorship of the guidelines have been written, even by the authoring organization of the hypothyroidism guideline [27, 9] but were ignored. The most important admonition to this case that is in the protocol [27] but is not written into the guideline [9]: the stipulation of definitions of critical and readily misunderstood words and terms [see the

definitions].

Studies of the authorship of medical guidelines have shown them to be substantially lacking. Dr. Shaneyfelt, et al., gave an average grade of 43% to 279 medical guidelines. [28] Dr. Grilli, et al., found that only 5% of 431 guidelines met all three criteria of their grading system and that most were unsatisfactory. [29] Dr. Burgers, et al., graded 100 guidelines and found that oncology guidelines were better (42.2%) than other guidelines (29.4%). [30]

Additionally, a study [31] estimates that 4% of all hospital patients will suffer from an "adverse" event that produces an estimated 200,000 deaths annually. Since half were preventable and a fourth are negligent, then the remaining causes are either beyond medical science or merely beyond practice guidelines, or in the confusion within practice guidelines.

Since it appears that human suffering is an inadequate motivation to transform vagueness to clarity, perhaps the civil rights guaranteed by the Constitution of the United States will. When medical missives meet the law at the government's boards of medicine, the concept of "Overinclusion," a derivative of the Equal Protection Clause of the Fourteenth Amendment, becomes applicable. (See the definitions, below.) Overinclusion occurs when two distinct classes, of different etiologies that require different treatments, are included in the same class and the treatment for one of these classes is improper and consequently places undue and excessive burden upon the members of that class.

Certainly, the inconsistent implications of the two implied definitions of "hypothyroidism" in the guideline [9] and the expression of the paradigm [4] creates two classes, those people included in the narrow definition and those included in the broad definition but not included in the narrow definition. There is an endocrine precedent: While excessive urination is a general symptom of diabetes, its somatic nature separates the various distinct subclasses. The major subclasses, type 1 and type 2, are similar to the classes of hypothyroidism: the inability to produce and the inability to use respectively.

Conclusions and Final Implications

Baisier, et al. [2], have empirically shown that thyroxine-resistant patients exist, can be treated properly, and regain their former selves with natural desiccated thyroid. This original treatment for hypothyroidism [1] has served many quite well [14, 18] in the past and presently. Other forms of combination therapies have been used successfully as well. [16, 17] The Baisier, et al., empirical results are not flukes. Physiological theory agrees. The exo-endocrine hormone operations, peripheral metabolism and peripheral hormone receptors, exist and are fallible. [5, 6, 22 - 24]

Accepting the fallibility of exo-endocrine operations upon the thyroid hormones, which common sense and science demands, also demands acceptance of the treatment with the active exo-endocrine hormone, triiodothyronine (T3), simply because that is the hormone that requires replacing in case of such failings. Unfortunately, the oligarchical powers bestowed on the medical associations via the state boards of medicine have far greater influence over physicians

than truth and reality. Of the 120 plus thyroxine-resistant sufferers found by our minuscule outreach effort – letters to the editors of West Virginia newspapers [32], only six have been able to use our research to persuade their physician to prescribe any physiologically necessary T3-containing therapy.

Hopefully, the Equal Protection of the citizens of the United States, guaranteed by the Fourteenth Amendment to the Constitution and Due Process, will, as it should, prevail. Hopefully, the existing Overinclusion of all hypothyroidism victims into a single class will demand an end to the linguistic etiologies that imprecisely treat all types of victims in the same way. Hopefully, the seemingly endless suffering by thyroxine resistant patients will end with their return to energetic lives by the proper treatment of exo-endocrine hypothyroidism.

Hopefully too, the linguistic methodology shown herein will spread throughout medicine to improve guidelines and medical care.

Albert Einstein recommends that problems be simplified to clarify them. However, they cannot be overly simplified, since that creates a different type of confusion. The early lack of knowledge plus the use of universally applicable natural desiccated thyroid gave single-class hypothyroidism credence. This over simplification became evident with the advent and use of levothyroxine sodium. Levothyroxine sodium left some hypothyroidism (broad, symptom-oriented definition) victims untreated, thereby demonstrating the confusion of over-simplification and overinclusion. Thus, the use of levothyroxine sodium exposed the existence of a second class of hypothyroidism victims – those with exo-endocrine hypothyroidism, who have different physiological etiologies that physiologically, rightfully, and lawfully demand different treatments.

Definitions

due process	The conduct of legal proceedings according to established rules and principles for the protection and enforcement of private rights, including notice and the right to a fair hearing before a tribunal with the power to hear the case.
endo-	A prefix meaning within or inside of.
exo-	[Gr. exo, outside] Combining form meaning without or outside of.
equal protection	The constitutional guarantee under the 14th Amendment that the government must treat a person or class of persons the same as it treats other persons or classes in like circumstances.
hypothyroidism	Thyroid-centric meaning: the clinical consequences of inadequate secretion by the thyroid. Symptom-oriented meaning: the clinical consequences of inadequate levels or usage of thyroid hormones. Potential future meaning: The clinical consequences of inadequate usage of thyroid hormones. [This definition encompasses all etiologies of hypothyroidism.]
overinclusion	Extention beyond the class of persons intended to be protected or regulated; burdening more persons than necessary to cure the problem. [Legally this concept requires judgement based upon the percentage of

	persons overincluded, the extent of their burden, and the potential for removing the burden.]
proscribe	Outlaw or prohibit; to forbid
starie decisis	[Latin for "to stand by things decided"] The doctrine of precedent, under which it is necessary for a court to follow earlier judicial decisions when the same points arise again in litigation.
stipulative definition	A definition that, for the purposes of the document in which it appears, arbitrarily clarifies a term with uncertain boundaries or that includes or excludes specified items from the ambit of the term.
vagueness	Uncertain depth of meaning. Vagueness raises due-process concerns if legislation does not provide fair notice of what is required or prohibited, so that enforcement may well become arbitrary. [However, amazingly, vagueness can be eliminated if there is a consensus in the community of the meaning.]

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32. Our minuscule outreach effort has consisted of only four sets of letters to the editors of West Virginia newspapers. We have no idea how many were printed, but we do have respondents from all over the state. We sent the respondents research papers to give to their physicians. The success rate was about 5%. The other reason for the outreach effort was done to impress the state government to act on behalf of the unnecessarily suffering. West Virginia demonstrated that it is a most obedient servant of the medical association rather than a protector of the weak.

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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. He has thirty-nine U.S. Patents in seven disciplines, some of which also have foreign counterparts. 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 euthroidism 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 endocrine system 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."