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Abstract

Normal Abnormalities: An investigation into the role of non-specific symptoms in the diagnosis, treatment and management of thyroid conditions within an NHS Thyroid Clinic.

Thyroid diseases number amongst the most widespread chronic conditions. They are difficult to diagnose and ten times more prevalent in women than men. Since hormones produced by the thyroid gland control cell metabolism in the body, symptoms of glandular failure are elusively diverse and non-specific. For instance a 'classic clinical symptom' of thyroid disease is the swelling of the gland into what is known as a goiter, which concurrently may or may not be a result of disease.

Consequently a definitive diagnosis conventionally relies on blood tests to measure the function of the gland. However, such a diagnosis is self-evidently partial. From participant observation conducted in a Thyroid Clinic in the United Kingdom and 20 interviews with patients who attended the Clinic, the research has explored the tension between the wide-ranging and non-specific symptoms that patients complain of and the highly precise biochemical measurements used by clinicians to diagnose disease. Predictably, many patients feel such tests are inaccurate measures of the extent of their disease, and do not match their own interpretations of clinical symptoms or experiences of suffering. In response, many clinicians express concern that patients are incorrectly attributing their symptoms to thyroid disease and as a consequence are demanding treatments which are not necessary.

This qualitative sociological study investigates the thyroid 'treatment gap' by exploring how a contest over meanings of pathology is manifested in the Clinic, and how the boundaries of 'actual' thyroid disease are co-constructed by clinicians and patients. Through an investigation of how the boundary of thyroid disease is embedded in and generated through broader social relationships, this project demonstrates an inherent tension in the biomedical paradigm(?) -between the 'cause and effect' model of how disease is biologically defined and the practice of treating disease as a process within the social context of clinical medicine. This study explores the idea that the suffering of the patient is co-produced by both the symptoms of physical disease and the tension between the pre-clinical model of ***knowing*** disease and the clinical ***process*** of treating it, where social and physical symptomatology are separated.

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Introduction

Seeing disease as being situated and performed within broader social relationships, via the production of specialist and professional knowledge and practices, is a common conceptual framework within medical sociology and anthropology. Countless ethnographies of health and disease (Lock 1994, Martin 1994, Mol 2002, Rapp 1999, Stacey 1997) and social studies of biomedicine (Berg and Mol 1998, Canguilhem 1978, Foucault 1976, Latour, Woolgar, and Salk 1986) have identified that bodies, pathologies and subsequent interventions are as much cultural artefacts and relationships, as they are biological entities. What this awareness of the 'situatedness' of the body, biology and pathology allows an analysis to do is see more - albeit through an often complex and contradictory collection of knowledges, languages and practices. As science-studies theorist, Annemarie Mol, points out in her analysis of Atherosclerosis, a disease is not a thing "…just by pointing at it, saying what it is, where it is, or whether it is, but also by handling it. Acting upon it. Transforming it…They perform it" (Mol 2002: 10). Although not an end in itself, this way of seeing a disease, as an assemblage of objects and practices across a number of contexts, is an approach that has been able to do justice to and make sense of the data collected by this study of thyroid conditions. The ability of 'seeing as an assemblage' to perceive the performance, complexity and 'mess', is something that resonates with how the disease is understood, treated, managed, experienced and made sense of by the current ethnographic study.

From the data collected during participant observation in an NHS Out Patient's Thyroid Clinic and interviews conducted with 20 patients who attended the Clinic, a common theme has emerged with regards to the tension between pre-clinical model of *knowing* thyroid disease and the clinical *process* of treating it. As will be described, in the process of diagnosing and treating thyroid disease, factors beyond the confines of the disease model are crucial to the manifestation of the condition. Firstly, I will provide a brief description of the field of thyroid conditions. Secondly I will present three instances of aetiology, symptomology and diagnostic practice, where this knowing and doing tension arises and subsequently shapes how thyroid conditions are commonly brought into being.

The Thyroid and its Diseases

The thyroid gland is described as a butterfly-shaped structure that sits over the top of the windpipe in the neck, an association that is not lost on the various professional and patient groups who chose a butterfly as their motif. The two halves, or wings, that sit either side of the windpipe are called lobes (left and right) and the body of the butterfly is called the isthmus, the entire structure sits just below the larynx (Bayliss and Tunbridge 1998)1. It averages approximately 20 grams in weight (Toft 1995)

Figure 1 and 2: Illustration of the 'butterfly' shaped thyroid gland showing position around the trachea and the butterfly logo of the British Thyroid Association (BTA), the professional organization of clinical specialists who deal with thyroid disease.

The gland is part of the endocrine system that is responsible for the production and distribution of hormones around the body. Particular glands produce specific hormones that, in order to facilitate a number of different functions and processes in cells around the body. The endocrine system, its glands and their subsequent hormones, are therefore, conceptualized as a communication system that consists of glands that manufacture messenger chemicals or hormones which 'regulate' and 'tell cells what to do' to ensure that various processes and conditions are sustained in order to facilitate a state of 'normal function' within the body. The thyroid gland produces the thyroid hormones T4 and in much smaller quantities, T3. The role of these hormones is to regulate metabolism, i.e. the speed at which cells within the body function. So, if there is too much thyroid hormone the body cells work too fast and if not enough the body cells work too slowly – resulting in an array of non-specific symptoms located throughout the body, both physical and psychological.

Disease of the gland, is therefore, present in the form of Hyperthyroidism when the gland works too quickly and Hypothyroidism when the gland works too slowly. However, these terms are descriptions of the level of function of the gland and can be caused by a number of factors including an autoimmune attack on the gland, side effects of some medications, cancerous tumours and over or under treatment of one or other of the abnormal levels of thyroid function.

Thyroid function itself is defined by the relationship between the thyroid gland and the pituitary gland. The pituitary gland sends a hormone called thyroid stimulating hormone (TSH or Thyrotrophin) to the thyroid gland, which 'tells' it to make thyroid hormones (T4 and T3). Via a process of negative feedback[1] the amount of TSH regulates the level of production of thyroid hormones ensuring that there

[1] Negative feedback – feedback that reduces the output of a system, i.e. the TSH reduces or increases the production of thyroid hormones in order to retain normal level and subsequently normal function.

is always the right amount of 'fuel' for metabolism to take place. As the British Thyroid Foundation explains:

> The mechanism is very similar to that which regulates the central heating in a house where there is a thermostat in, say, the living room, which is set to a particular temperature and which activates the gas- or oil-fired furnace or boiler that heats the hot water. In the case of the thyroid, the 'thermostat' consists of a little gland, called the pituitary gland, that lies underneath your brain in your skull (The British Thyroid Foundation 2006)

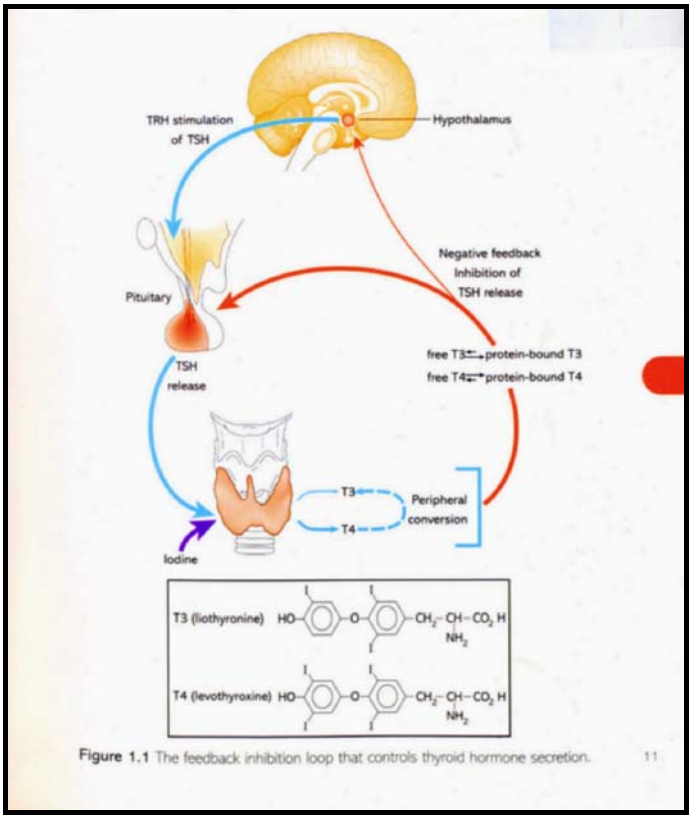


Figure 3: Diagram representing the feedback mechanism or the 'conversation' between the hypothalamus, pituitary gland and the thyroid gland. As this diagram demonstrates the regulation model of thyroid function is represented rather than the anatomical position of the relevant organs.

Although the health of the thyroid is mediated around this feed back mechanism, which in turn provides the basis for the diagnosis and treatment of thyroid disease, as will be demonstrated in the following examples, this model is continually fluctuates in significance across various contexts.

The Vaguely Genetic

Although the causes for thyroid dysfunction can vary, the most common cause is an autoimmune attack on the gland that can take the form of either stimulating it into over production (hyperthyroidism) or damaging it so it under produces (hypothyroidism). As the patient information and support group organisation The British Thyroid Foundation states, this autoimmune attack is 'sort of ' hereditary;

> *Are thyroid disorders hereditary?*
> It depends on how you define "hereditary". It is not handed down from parent to child in every generation. It is "hereditary" in the sense that autoimmune disease in your immediate family or predecessor's means that you have an increased risk of a thyroid disorder yourself (The British Thyroid Foundation 2006)

Moreover, although there is an array of literature attempting to develop an understanding of the complicated genetic aetiology of predispositions to autoimmune thyroid conditions (Chistiakov 2005, Hall and Stanbury 1967, Tomer et al. 1999, Tomer et al. 2001) within the Clinic it is not really significant. Although they may be of interest to and understood by a consultant physician in an NHS Out Patient Clinic, it is not necessarily used in their process of developing a diagnosis or treatment. Within a clinical consultation, when the Consultant questions a patient about a family history of the disease, it is not only to aid in assessing the likelihood of the patient having the condition, but also acts as a way for the clinician to work out how familiar the patient is with the condition and what it might entail symptomatically and in terms of treatment. i.e. if the patient already knew what the condition was through the experience of a family member, the clinician tended not to go into as much detail in terms of how he explained causes and symptoms to them. So whilst a molecular biologist could be more specific and demonstrate more specialist knowledge than the 'sort of' answer supplied by the British Thyroid Foundation with regards to genetic susceptibility towards thyroid conditions, in a clinical environment this detail is not particularly useful. Firstly, it does not offer a superior diagnostic tool to the standard thyroid function blood test, and secondly, knowledge about the disease process at a molecular level does not change or influence the available treatments.

Moreover, the familial disposition to thyroid disease was conceptualised by patients in yet another way. Often the vagueness of the hereditary nature of the predisposition to thyroid disease and the slow onset of the non-specific symptoms, were made sense of by embedding them in specific formulations of kin relationships, such as shared physical characteristics or personality traits. As Carole explains, her thyroid disease makes sense due to the likeness to her father's side of the family.

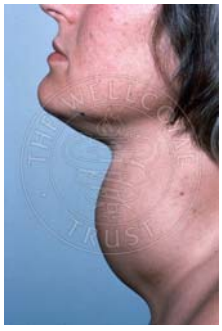
> **C:** My elder cousin on that side of the family [paternal], he died actually, um, but I think he had a problem and also my female cousin…they both had thyroid problems like their Mum. It didn't occur to me in a million years I would ever get it [a thyroid problem] although I am quite like that side of the family. Tall like my father, um, my mother's side are very short .My mother is 4' 10''…Well, she is now, she was a bit taller. My Grandmother was 4' 10", she was tiny…minute, so they are quite short, they all tend to be short on my Mum's side.
>
> **M:** So you feel like you are more like your Dad's side?
>
> **C:** Oh yes, definitely. I am more like him as a person too…

The Normal/Abnormal Goitre

A goitre or swelling of the gland is considered a 'classic' sign of thyroid disease, but concurrently, goitres are also highly ambiguous entities with regards to the manifestation of disease:

> Many disorders of the thyroid gland may make it bigger. The enlargement may be associated with a normal output of thyroid hormones, and the patient is then said to be euthyroid. The goitre may be associated with and increase secretion of thyroid hormone, and the patient is then said to be hyperthyroid. Or it may be associated with decreased deficient output and the patient is then hypothyroid. From this you will see that the size of the thyroid gland bears little relationship to its secretory activity or function (Bayliss and Tunbridge 1998) 10

The examinations of all patients with a goitre, for whatever reason, followed a simple protocol. First a physical examination of the gland would occur. This consisted of the Consultant standing behind the sitting patient and reaching round to the front of the lower part of the neck and locating the gland. The Consultant would then gently feel and press the gland to get to know the form and size of the swelling or the number and size of individual nodules. The Consultant would then ask the patient to swallow, in order to further ascertain the size and positioning of the thyroid abnormality. Sometimes he would comment how the patient had a 'nice slim neck' making the goitre easy to see, however, most of the time this physical examination of touch was necessary in order to 'see' the swelling more clearly and confirm the contours of the enlarged gland. Frequently patients had not realised they had a swelling, and sometimes it was a relative or their G.P who had first noticed it. Again this is contradictory to much of the literature that represents goitres particularly with regards to there manifestation as a discernable entity. As the images below demonstrate, in text books goitres are noticeable abnormalities. However, as the third image demonstrates, they are also subtle swellings that, within the Clinic, were picked up by the Consultant, who knew what he was looking for, via palpation.



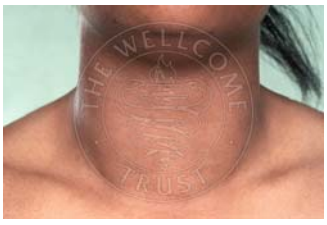


Figure 4,5 and 6: The goitres depicted in figures 4 and 5 are far more distinct and sizeable than those commonly seen in the clinic (figure6). In this sense these images provide 'ideal type' goitres, as usually palpation was also needed to 'see' them within the clinic.

During an interview one patient explained to me that during late 1940's early 1950's she had heard that one of her aunts on her father's side had had a breakdown and been institutionalised because, she claimed, she felt like she was being strangled. It later transpired that this aunt actually had hypothyroidism and it was a goiter (that was apparently un-noticeable), in combination with the depressive symptoms of the disease, that led to this subsequent misdiagnosis and hospitalization. This problem of the goiter, both in terms of its pathology (or not) and visual manifestation (or not), again demonstrates the tension of how thyroid disease is known and how it behaves within practice. Again it

is a factor of the disease that misbehaves, it is not as visible both in terms of its proximity to the diagnostic process and its manifestation as a sizeable swelling of the gland that is visible in the neck of the patient.

Biochemistry *is* the Disease

As mentioned previously, disease of the thyroid is defined as the malfunction of the feedback mechanism between the pituitary gland and the thyroid gland, although the causes for this can be many. The health or function of the thyroid is calculated biochemically through a thyroid function blood test (TFT) that measures the levels of thyroid hormones (T4 and T3) in the blood, as well as the thyroid stimulating hormone (TSH) generated by the pituitary gland. Within the Clinic these tests are considered to be the definitive diagnosis of thyroid disease, due to the many and wide ranging and non-specific symptoms that thyroid function can cause. So whilst during the consultations the clinician would ask the patient questions about their bowel movements or sleeping pattern and look for visible signs, such as a goitre, shaky hands, a fast or slow pulse or hair loss, the blood tests were what secured the definitive diagnosis of thyroid dysfunction. Essentially, clinical symptoms are secondary to biochemical measures and are not considered proof of thyroid disease in and of themselves due to their non-specific nature. According to the first consultation draft of the UK Guidelines for the Use of Thyroid Function Tests:

> The symptoms of thyroid disease can be relatively non-specific and one of the first challenges for the G.P is to consider thyroid disease in the differential diagnosis of the symptoms of the individual patient. Once this consideration has been made the GP would like TFT either to exclude or confirm the diagnosis of thyroid disease." (Association for Clinical Biochemistry 2005)

As well as diagnosing disease, the measurement of the mechanism can also be used as an indicator of disease occurring in the future. Often a borderline measure of TSH can occur in a blood test result, thereby inferring that the patient has sub-clinical thyroid disease. This means that the TSH level is at a low or high level, depending on the type of dysfunction, (Hypo or Hyper) but is just about maintaining 'normal function'. For example, in the case of Hypothyroidism a blood test result that is sub-clinical is often described as the brain (TSH producing pituitary gland) working at 'full pelt' to keep the thyroid going.

Another predictor of disease, specifically with regards to auto-immune thyroid dysfunction, is measuring the presence of thyroid antibodies (TPOs) in the gland. If they are present in high numbers an attack is occurring and as a result the gland will eventually stop working. Again, this can identify the cause for dysfunction before the gland actually fails. Patients at this stage are deemed to have a sub-clinical form of disease and are therefore not supposed to be suffering from any symptoms, although some may have a thyroid swelling (goitre).

The function of 'my thyroid' is something that continuously emerged as an area of contention in the clinic, which was specifically mediated around the relationship between the blood test results and the symptoms the patients were suffering from. Patients' experiences of 'symptoms' that fitted a diagnosis of thyroid dysfunction were often attached to a pathology within their gland that they did not feel was being acknowledged by the Consultant. Many patients felt that they had their own unique level of thyroid function that the reference ranges of the blood test could not account for - often these patients were subclinical, or had a thyroid swelling but not biochemical proof of disease. Moreover, they were often suffering from the list of symptoms, that albeit non-specific, were considered 'classic' indicators of the disease, such as weight gain, lethargy, depression and a short temper, or in the case of Alex, a Hypothyroid woman in her early thirties, thinning of the hair.

> "I've only got to look at the vacuum cleaner to see how much hair is falling out, and it's full of my hair. I run through my hair and I just think oh, it does fall out in surprising amounts, my hair is very thin. According to the Doctor, your hair is on a two year cycle, so it will be another year before I get it back, or once I have found a right dosage for me. But as I say, I only have to turn the vacuum and look at the rollers and it's stuffed full of my hair, and it does fall out in large quantities, so it can't be right. If you speak to other people who have thyroid problems and they say their hair only falls out when their tablets aren't right, and they have been taking their tablets for years so, err…I've never found a happy medium, so I don't really know. A year down the line I'm still no closer…perhaps I'm a bit closer. My current dosage…I don't know the average, but mine isn't right"

Conclusion

The difficulty in diagnosing thyroid disease purely on the presentation of it's 'classic' yet non-specific symptoms, seems to be escalated by the practical biomedical response deployed in order to treat the disease. Whilst the clinical texts and health care professionals can embrace the vagaries of the disease when they elucidate its symptomatic presentation, they cannot maintain this understanding when reaching a definitive diagnosis with regards to the idiom of biochemical feedback. Therefore, thyroid disease is not only an imbalance in the production in thyroid hormones, or aching limbs, or low mood, or a puffy face, or bulging eyes, or a swollen neck, it is also the specific process through which it is, but also, is not eventually diagnosed. The inability of the model of thyroid function to get at the vagaries of the disease, beyond its biochemical boundaries, is therefore, as much a part of suffering from the disease as, for example, a goitre is. This is a situation that is acutely recognisable for those patients who exist in the border between normal and abnormal states of thyroid function, where their disease is not considered to be 'present enough' to receive treatment - in spite of the fact that they have been diagnosed with an abnormality within a clinical context.

Suffering thyroid disease, therefore, extends far beyond the manifestation of biochemically attributable symptoms. It is also produced by how disease is seen and interpreted within a biomedical paradigm. That is, any symptoms that are not attributable to 'biochemical thyroid disease', become attributed to

illness – the ways in which patients evaluate and experience their disease or abnormality in terms idioms that are considered to sit in the realm of imprecise culture. Moreover, due to the scientific *"culture of no-culture"* (Traweek 1988) embedded in the paradigm of biomedical disease categories that claim to be a distinct domain *"from morality and aesthetics, religion and politics and social organization"* (Hahn 1983) this disease/illness distinction is stratified as real and imagined, true and false. Subsequently, the suffering of the patients with thyroid disease is ghettoized into aetiology, symptomology and diagnostic definitions and practices, that as has been demonstrated, are themselves imprecise messy and context dependant.

However, as the anthropologist Margaret Lock states, this contextualizing of the truth claims of biomedicine through a dissection of natural categories of health and illness, is not where an analysis of biomedicine, such as this study of thyroid conditions, should end. The overwhelming question should be *"how and why certain representations become dominant at specific times and the exposing the hegemony they exert over everyday life and practices associated with health and illness"* (Lock 1993).

For example, the retreat into an overtly mechanical mode of delivering treatment by the Consultant, was often a response to the way in which the patients perceived his specialist knowledge and professional role, as well as their own status as sufferer. Often when a patient entered the clinic asking for particular diagnostic techniques or treatments for their condition, which they had read about, for example, on the internet, the Consultant would show resistance. This response could be attributed to the patients asking for treatments and medications that were simply not available through the NHS, but also because, he felt that if that is what they really wanted, there was 'no point in him being there' and treating them through the Clinic'. That is, if they wanted to indulge in treatments for which, in his opinion, there was no sound evidence base - such as dietary measures or herbal remedies - what was the point in him taking the time to treat them in his way? Whilst this opinion could be initially construed as a medical professional protecting his professional judgement is was also apparent that this view was embedded in the pressures on NHS resource allocation and a belief in a doctors' duty of care to their patients. Often his expression of resignation at the demands of the patients were commented upon in between the many consultations in the highly pressured Clinic, where there was simply not the time to discuss unsubstantiated and biomedically unfamiliar theories. In addition, the attitude that some patients seemed to reproach the Consultant with, in response to his detached reception to their preferences for treatment, assumed that he was behaving as such because he was philistine when it came to treating thyroid disorders. However, often this clash of professional and lay biomedical models would mask another key reason for the Consultant being so dogmatic with regards to the treatment algorithm. He did not entertain some treatments because he believed, and in some cases had seen, how these other methods had actually made patients unwell. For example, a number of patients attending the clinic were doing so because they were being treated for hyperthyroidism, due to the over

administering of thyroid replacement therapy, by either themselves or other clinicians. It is perhaps also not irrelevant to note that this competition between professional and patient models of thyroid disease is occurring at a time within the NHS where policy initiatives such as 'Patient Choice' are encouraging individuals to become fully engaged and responsible for their own health in order to facilitate better use of health care resources.

As this paper has demonstrated, when thyroid conditions are described through specialist and professional texts, patient support group literature, observations of clinical practice and patient accounts, they lose their shape and are difficult to see as just the result of a straightforward pathological process. In fact, as has been demonstrated, to understand thyroid conditions and to understand the experience of people who suffer from thyroid conditions, is to encounter the grey areas between: normal/abnormal, diseased/healthy, euthyroid/hyperthyroid, euthyroid/hypothyroid and text/practice. Moreover, these spaces in between the pre-clinical knowledge and clinical practice, both represent and generate the suffering of thyroid disease. That is, they give thyroid disease a recognisable cultural form via a biomedical way of knowing, but also, sideline other ways of knowing and experiencing disease, which in turn generates suffering.

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