

Muddling through with non-compliant biology

An ethnographic investigation the meaning and practice of evidence in an NHS thyroid disease out-patients clinic.

A thesis submitted for the degree of Doctor of Philosophy

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In loving memory of Neula Flo Warren and Arnold Kilgour Ferguson

Abstract

Thyroid diseases number amongst the most widespread and chronic conditions in the population. They are symptomatically vague, difficult to diagnose, and are more prevalent in women than men. Since the hormones produced by the gland control cell metabolism in the body, symptoms of glandular failure are elusively diverse and non-specific and are easily attributable to other diseases or no disease at all.

Consequently, a definitive diagnosis conventionally relies on a blood test to measure the function of the gland. However, as observed in the clinic, such a diagnosis is often partial and is not compatible with the clinical symptoms some patients present. 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 study investigates the indeterminate aspects of thyroid dysfunction by exploring how a contest over meaning is manifest in the clinic, particularly with regards to how embodied clinical symptoms and dis-embodied biochemical measures are used as evidence for and against decisions over diagnosis and treatment. The thesis identifies that, due to the vague nature of both symptomatic and biochemical evidence, thyroid disease is a location where medicalisation is impossible. Subsequently, using the available discourses of the clinic, specifically EBM and patient centred medicine, clinicians and patients construct various orders of thyroid biology and pathology, in an attempt to satisfy diagnostic strategies and treatment needs.

As a consequence the thesis can add to the analytical purchase of the anthropologist Paul Rabinow's concept of biosociality and theories of governmentality more generally. That is, it demonstrates how practices of re-making thyroid biology are possible, not because of a new found molecular control over thyroid biology, but due to the non-compliant nature of thyroid biology and the technical deficiency of current diagnostic and treatment strategies deployed to attend to it within the clinic.

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Chapter 1

Introduction: the accuracies and vagaries of thyroid disease

Tanya, a woman in her mid forties who had attended a thyroid clinic for a thyroid swelling provided a typical account of her experience when I interviewed her for this study at her home in London in June of 2006. She had visited her GP during 1989/90 because she noticed that her neck had started to swell. Her regular GP was away on holiday so she saw a locum who, after examining the swelling, referred her to a consultant surgeon. During the consultation with the surgeon, in which Tanya thought that he was 'rather showing off' in front of the medical students who were observing the clinic, he told her that her thyroid gland was swollen and that it would 'have to come out'. He also offered to conduct a biopsy in order to determine whether the gland was cancerous, but warned that such a test could only show the presence of cancer if the biopsy needle hit the 'right spot'. He also said that he couldn't tell what type of goitre it was – smooth or nodular¹. Therefore, removal of the gland was considered to be the best option.

Tanya recounted having been 'fairly horrified' by the prospect of the removal of her thyroid gland because people she knew whose thyroid glands had been removed had encountered problems returning to full health after the operation. After its removal, the hormones produced by the thyroid gland proved hard to replace, causing chronic problems such as continued lethargy, depression and excessive weight gain. After the appointment with the surgeon, Tanya went back to her regular GP to express her concerns. Her GP had returned from holiday and was surprised to learn that the locum had referred her to a surgeon and not to a specialist endocrinologist, who would be more familiar with diseases of the thyroid gland. She referred Tanya to an endocrinologist for a second opinion. After waiting six months, Tanya was scheduled for an appointment during which the endocrinologist scanned her neck and told her that it was a 'simple goitre' and of no medical consequence. Not entirely reassured by

¹ A smooth goitre is a uniform swelling of the gland that is 'normal', but also, can be a sign of a thyroid problem. A nodular goitre occurs when a number of lumps develop in the gland. Again these can be normal and asymptomatic, but can also be a sign of a thyroid problem. The relationship between thyroid abnormalities and thyroid disease is discussed in detail in Chapter five.

this second diagnosis, Tanya agreed with her GP that she would have an annual thyroid function test in case her gland failed in the future – a risk for individuals who have goitres, according to the current literature concerning thyroid disease.

Tanya had annual tests, but after fourteen or fifteen years she decided to have her thyroid checked again more thoroughly. It worried her that her neck appeared to swell at times of stress, and she found this symptom troubling. In preparation for an appointment with the endocrinologist, she conducted some research on the Internet where she discovered patient-run websites claiming that the blood tests were 'a rather blunt instrument' for measuring the health of the gland. This discovery strengthened her concern that there might in fact be something wrong with her thyroid gland that had eluded diagnosis. Given her past experience of a range of conflicting diagnoses, Tanya's doubts did not seem far-fetched. For example, in addition to the confusion about her abnormal thyroid gland Tanya had her case of appendicitis misdiagnosed as a urinary tract infection and then ovarian cysts. Before the correct diagnosis was reached her appendix ruptured making her very unwell and consequently she has 'light worries about the reliability of the NHS'.

When Tanya mentioned her suspicions about her thyroid gland to her GP, her GP said that she did not know enough about the subject to comment on the claims, so Tanya left some information with her. When Tanya next saw the consultant, she mentioned this literature to him and he 'rather poo poed it'. Tanya resigned herself to the fact that both her GP and the consultant appeared to believe that there was nothing wrong with her. However, on reading about some of the symptoms of an underactive thyroid gland (one of the diseases that can affect the thyroid) Tanya suspected again that there might be something wrong with her. She has problems losing weight (although admits to 'slightly over-eating') and has dry skin and thinning hair, both of which are classic clinical symptoms of the disease.

So, is there anything wrong with Tanya? She has been monitored for an abnormality in her thyroid gland for over fifteen years, however, there has been no diagnosis of a disease so far. So, is she just unnecessarily anxious because she has read scientifically suspect claims on the internet? Or are

the blood tests currently used in the diagnosis of thyroid disease insufficient? Is thyroid disease 'too vague' a condition to measure accurately - is its diagnostic profile too similar to the metabolic strains of modern urban life? As this thesis demonstrates, one thing is apparent: none of these clinical diagnoses, professional opinions, or patient concerns can either be wholly discounted or positively affirmed. It is instead the dynamic between them that largely defines the experience of thyroid disorder for patients and health professionals alike.

This dynamic between clinical diagnoses, professional opinions and patient concerns demonstrated in the case of thyroid disease has significant consequences for the concept of medicalisation (Zola 1972), insofar as this term is taken to refer to the power of authoritative expert knowledge to subordinate other truth claims, and discipline the bodies of its subjects. As is described by this ethnographic study of thyroid disease, a tidal wave of changes affecting health professions, identities and management has resulted in both a pluralisation and disintegration of this familiar sociological concept. Caught in the interstices of conflicting models of care and cure, Tanya's goitre was alternately identified as something that required examination, 'that had to come out', that 'needed to be monitored', that may have been wrongly diagnosed, and that was 'nothing to worry about'. The multiple and contradictory accounts of thyroid disease typified by Tanya's case are repeatedly confirmed by the data collected for this ethnographic study based in a weekly NHS thyroid clinic, and drawn from over 20 interviews with the clinicians and patients who ran and attended the clinic. As in Tanya's case, depictions of thyroid disease, its diagnosis and its treatment vary greatly between medical professionals, among internet sources, and in formal accounts of thyroid disease found in clinical practice guidelines and textbooks. There is a corresponding feeling amongst clinicians and biochemists that diagnose and treat thyroid disease that this once 'simple' disease has become increasingly complicated and frustrating. They suspect that there is something about the contemporary clinical encounter that makes this well established, common and straightforward disease unnecessarily complicated. But what would this be? Has thyroid disease changed as a result? Is it atypical, or does it instead represent a pattern that is becoming more common, as the progress of what Marilyn Strathern calls a 'post pluralist' society transforms formerly unassailable truth claims into matters of choice?

It used to be so easy...

In the preface to his 1947 book *The Conquest of the Unknown: The Story of the Endocrine Glands*, doctor and author George Bankoff claims that the mysterious nature of the endocrine glands, the relationship between our biology and our behaviour, had been well acknowledged even before the dawn of the Christian era where the magic of human organs was embedded with folklore. Bankoff describes how tribal warriors ate animal organs to increase strength and banish fear before battle; cowards were made to eat the hearts of enemies to give them courage. He also refers to the doctrine of the medieval Swiss physician Paracelsus: 'Heart cures heart, spleen cures spleen, lungs cure lungs' (1947 :vi) and suggests he was the first to realise that the human body possesses substances that 'Science' (capitalization in the original) later named hormones. Therefore, Bankoff is implying that when he published his history of the endocrine glands this once unknown, mysterious and magical system had been conquered by modern science and was a basic unequivocal fact of human physiology. In fact, Bankoff considers knowledge about the endocrine system so certain and straightforward it can be communicated to the 'lay man' with confidence in order to help him decipher the mysteries and workings of the body:

All medical writers have one paramount aim: to bring out the mysteries of medicine for the layman.. I will not enter into the debatable question of whether the layman ought to know about the mysteries of his body and the workings of his mind and glands. No doubt some will say that he must remain ignorant of his ills and his body organs. They will tell you that a little knowledge is dangerous and that giving the layman a superficial knowledge of the functions of his body might lead him astray and make him imagine all the possible ills that can befall him. That may be so, but I am not convinced and I am entirely of the opposite view. I believe that the sooner we give our patient – the layman- sound knowledge of the working of his body, the better we will serve him[...]He will be more intelligent and will seek the advice of his doctor more readily, thus avoiding perhaps many grave consequences (Bankoff 1947: v – vi)

Bankoff's assumption that if doctors are open with patients medicine will 'serve them better' though the communication of 'sound knowledge' assumes that medical knowledge advances in a straightforward and progressive manner. Bankoff assumes that through biomedical research

and innovation, complex systems will be revealed and fit neatly into our increasingly complete understanding of the mechanisms of the body, health, disease and illness. However, as this study demonstrates, the march of medical progress is by no means straightforward. Often, ironically, the more we 'know' about the body and disease and the more techniques we develop to manipulate them, the less we understand as a result. For instance, in the case of thyroid disease, biochemical diagnostic tests have been developed that can detect not only overt disease but also subclinical states of thyroid pathology which, according to current professional wisdom, 'may or may not' result in symptoms for the patient. More generally, innovations that can save, extend and 'enhance' our lives, such as genetic screening, new reproductive technologies, organ transplantation and mood altering drugs have also resulted in the emergence of new questions and challenges, or what might be described as a sense of deficit that is directly engendered by technological progress. Improved medical knowledge has enabled unprecedented control over human health by facilitating the prevention, treatment, and often cure of a wide array of chronic and life threatening, as well as simply inconvenient pathology – from heart disease to headaches. However, as this thesis will demonstrate, such changes have at the same time both continuously and dramatically remodelled the medical landscape, creating new challenges for the medical profession and patients. As Nikolas Rose claims in the introduction to *The Politics of Life Itself* such technical and social shifts 'have generated hopes and fears, expectations and trepidation, celebration and condemnation' (2006:1).

The sociological complexity of contemporary medical and scientific progress - and its implications for professional and patients' understandings and experiences of disease - have understandably emerged as one of the most important contemporary arenas of social and cultural theory. The sociologists Ulrich Beck (1992) and Anthony Giddens (1990) have, in different ways, identified that modernization has produced a number of risks, such as the discovery of new diseases, pollution and crime, that have led to a distrust in government and experts, including the medical profession. Anthropologists such as Donna Haraway (1990), Margaret Lock (1994), Emily Martin (1987), Rayna Rapp (1999), Marilyn Strathern (1992a) and Paul Rabinow (1992) have identified the constantly shifting meanings of the biological and natural across a range of biomedical contexts. The

ability of such studies to draw attention to the fluctuating and situated boundaries of biology, health disease and illness inform the approach used in this study of thyroid disease. In particular, they have helped to develop a theoretical and methodological approach that can grapple with, and account for, the non-compliant and indeterminate nature of thyroid pathologies.

The anomalous nature of thyroid disease, which is the subject of this thesis, can be said to represent the 'new typical' idiom of disease - where 'at risk' epidemiological populations grapple with uncertain competing and disparate medical knowledge (e.g. biochemical, genetic, immunological and dietary) in order to acquire and maintain acceptable levels health. It is these practices of grappling with health that make thyroid disease not only 'typical' in the context of the contemporary clinical encounter, but also, mundane and often unsatisfactory for both patients and clinicians. Often the treatment for thyroid disease does not result in a triumphant 'cure' but an uneasy strategy of disease management negotiated by doctors and patients that contradicts the perceived precision of diagnostic tests and the medical profession's knowledge of thyroid hormone action. In addition, as is described in the next section of this chapter, thyroid disease and its treatments were the result of a flurry of research and innovation during the nineteenth and early part of the twentieth century. During this period the endocrine system, and its role as a regulatory system that enabled the body to maintain a balanced internal environment, was assumed to be synonymous with the 'internal secretions' - what we now call hormones. Although thyroid disease has been redefined under subsequent medical metanarratives, such as immunology, development, and more recently molecular biology (Porter 1997, Borell 1985, Medvei 1993) its treatments have remained largely unchanged since the late nineteenth century and there is very little research or innovation in the field (Walsh 2002a).

The major change ushered in by twentieth century medicine is the extent to which endocrinology, combined with clinical biochemistry, can be used to detect, and calibrate, the levels of specific substances such as thyroid hormones. The isolation of thyroid hormones and the subsequent mass production of synthetic replacements² and blood tests that measure thyroid

² Thyroid hormones were first synthesized by British biochemists Charles Harrington and George Barger in 1927 Charles Harrington and George Barger.

function have resulted in a robust biochemical model of the disease corresponding to standardised diagnostic and treatment practices. However, as will become clear, the 'straightforwardness' of thyroid disease as the measurable malfunction of a biochemical mechanism comes at a cost. That is, the disciplining of thyroid disease through the idiom of biochemistry clashes with the experiences of some patients as well as additional contemporary bureaucratic and political discourses that aim to rationalise health services resources and place the patients at the centre of the medical encounter. More specifically, it will be demonstrated that whilst this model does account for a biomedical 'truth' of thyroid disease, it does so in a broader community of truths that jostle for position in the context of the 'post-pluralist' medical encounter. Therefore, as this thesis concludes, it is the relationships and interactions between these varied truths and their corresponding practices which ultimately constitute thyroid disease within the contemporary National Health Services (NHS) in the United Kingdom. In the following section the development of the formal biomedical account of thyroid biology and pathology will be summarised. Towards the end of this chapter, this formal account of the endocrine system, and specifically thyroid disease, will be used as an empirical location where a number of more general questions will be posed with regards to the politics of contemporary biomedical practice within western healthcare contexts.

A brief history of thyroid disease

In 1854 the French physiologist Claude Bernard developed the concept 'milieu interieur' or the 'environment within' that described the way in which the body produces chemicals – what we now call hormones- that maintain certain internal conditions necessary for survival. Bernard suggested that;

All of the vital mechanisms, however varied they may be, have always one goal, to maintain the uniformity of the conditions of life in the internal environment[...].The stability of the internal environment is the condition for the free and independent life (Bernard 1974: 84)

The idea that there was a 'vital mechanism' that resulted in the 'uniformity' and 'stability' of the body's 'internal environment' enabled physiologists such as Bernard to ascribe a communication-like function to the glands of the body and to surmise that they were the mechanisms that secreted

substances that controlled this regulatory system. Before the concept of internal regulation, the organs of the endocrine system had been comprehensively described anatomically, but not in terms of physiological function. As the medical historian Ted Porter claims, in the seventeenth century a flurry of anatomical studies described organs but were unable to visualize the function of, 'enigmatic tube like ducts carrying off the secretions of such organs as the liver' (1997: 562). However, the introduction of Bernard's concept meant that a hypothesis of function of these previously mysterious organs was developed and formed the basis for a number of gland-specific experiments that could investigate and ascribe function to them. For instance, in 1856 the Mauritian physiologist Charles-Édouard Brown-Séquard demonstrated that Addison's disease was a result of the failure of the adrenal glands by removing them in animals and studying the consequences (Porter 1997: 563). In 1902 in England, physiologists William Bayliss and Ernest Starling (Borell 1985: 11) found that when they injected hydrochloric acid into a dog's duodenum the pancreas produced pancreatic juice. They concluded that the duodenum must secrete a substance that sent a message to the pancreas and prompted it into action. In 1905, following his work with Bayliss on the function of the pancreas, Starling adopted the word 'hormone' meaning 'I excite' in Greek as a name for these secretions (Porter 1997: 563).

The research triggered by Bernard's hypothesis also prompted further investigation into the function of the thyroid gland. The medical historian T.C. Golding notes that descriptions of the thyroid gland and swellings of the neck caused by thyroid abnormalities have appeared in writings for thousands of years. For instance, Marco Polo mentions such abnormalities in certain areas of Asia during the thirteenth century and Galen and the Roman author Juvenal both mentioned the gland, its propensity to swell and a number of hypothetical functions for it (Golding 1952:55). In the United Kingdom, 'Derbyshire Neck' was a common occurrence in the Peak District - a phenomenon that also occurred in mountainous areas around the world due to the low levels of iodine in the water supply that impaired the glands' ability to produce hormones (Bayliss and Tunbridge 1998a: 115). Moreover, in the first half of the nineteenth century the Irish physician Robert Graves (Graves 1835) and the German physician Karl von Basedow (Bayliss and Tunbridge 1998a: 183) independently described a number of symptoms, such as bulging eyes and palpitations that occurred along side

swelling of the thyroid gland – what we would today diagnose as hyperthyroidism (or Graves/ von Badesow disease depending on your geographical location). However, in 1844 conjecture about the function of the thyroid became associated with the concept of internal regulation when the English surgeon John Simon claimed that the gland secreted a substance that passed into the circulatory system. Moreover, at around this time in Bern the physiologist Moritz Schiff demonstrated that when the thyroid glands of guinea pigs and dogs were removed they died, suggesting that the thyroid gland was necessary for life. (Porter 1997: 563). In 1850, the English physician Thomas Blizard Curling performed autopsies on two 'mentally deficient' children which revealed that they didn't have thyroid glands (Medvei 1993: 135). And finally, in 1873 the Queen Victoria's personal physician Sir William Gull formally described a set of symptoms that occurred in a woman whose thyroid gland had become swollen and atrophied

Miss B...became insensibly more and more languid, with a general increase of bulk...Her face altering from oval to round,...the tongue broad and thick , the voice guttural, and the pronunciation as if it were too large for the mouth (creatnoid)...There has been a distinct change in the mental state. The mind, which had previously been active and inquisitive, assumed a gentle, placid indifference, corresponding to the muscular languor, but the intellect was unimpaired[...]The change in the skin id remarkable. The texture being peculiarly smooth and fine, and the complexion fair, at a first hasty glance there might be supposed to be a general slight oedema of it...The beautiful delicate rose-purple tint of the cheek is entirely different from what one sees in the bloated face of renal anasarca. (Gull 1873- 1874)

Gull named the swelling and wasting of the gland and these associated symptoms myxoedema, or what we would now call hypothyroidism or an underactive thyroid gland. Subsequently, in 1891, Gull's fellow English physician George Redmayne Murray began to develop a cure by linking the failure of the thyroid gland with an impaired ability to produce secretions (hormones). In an experimental treatment, he administered an extract of a sheep's thyroid gland by injection to a 46 year old woman with myxoedema and her symptoms improved (Murray 1891).



Figure 1: Clinical photographs of a 55 year old woman with myxoedema (Wellcome Collection)

This practice of 'organotherapy'³ soon became a common approach to treating endocrine disorders and throughout the early twentieth century a programme of research was established that attempted to isolate the properties of endocrine glands and their hormones. For instance, in 1921, with the help of the chemist J.B. Collip, medical scientists Frederick Banting and Charles Best (2007) identified and then isolated insulin and thus developed a cure for diabetes. Also, in 1929 G.W. Corner and Willard Allen (1929) identified the role of the corpus leuteum in the production of the pregnancy hormone progesterone. Over the next few years a number of other sex hormones were discovered and in the early 1940's progesterone was synthesized by American biochemist Russell E. Marker using extracts from a wild Mexican Yam (1947). This field of research would eventually result in the development of the contraceptive pill (Borell 1987), and techniques used in reproductive medicine (Borell 1985, Parkes 1966), as well as number of physiological theories that attempted to establish the biological basis of sexual difference (Oudshoorn 1994) .

The isolation of thyroid hormones began with the identification of iodine in the thyroid gland by the German chemist Eugene Baumann in 1885. After this discovery it became clear that iodine deficiency caused an overgrowth

³ Organography was first developed by Brown-Séquard in 1889 and took the form of injecting men with the invigorating substance contained in the testes as a rejuvenating agent to improve physical strength and mental activity. Although a practice that was designed to enhance the physical performance of men didn't become established, it did pave the way for the development for future hormonal treatments (Medvei 1982: 289).

in thyroid cells resulting in goitres, and soon after the Austrian psychiatrist Julius Wagner von Jaure began to successfully treat goitres with iodine (Medvei 1993:140). This identification of the iodine-rich nature of the thyroid secretions directed research that would eventually isolate thyroid hormones, and in 1927 British biochemists Charles Harrington and George Barger (1927) synthesized thyroxine, and synthetic thyroxine, or Levothyroxine finally became commercially available in 1950 (Wiersinga 2001). Interestingly in contrast to the perceived wisdom of contemporary prescribing practices, the medical profession was reluctant to prescribe Levothyroxine and continued to use thyroid extract derived from pig, sheep and cow thyroid glands until the 1970's and early 1980's (Macgregor 1961, Jackson and Cobb 1978).

In sum, the 'unknown world' of the endocrine system which, according to Bankoff had been enshrined in folklore for centuries, was gradually conquered throughout the mid 18th to the mid 19th century. Scientifically, Bernard's concept of 'milieu interieur' presented a rationale that formed the basis for the experimentation that eventually assigned the specific functions to the glands of the endocrine system with which we are familiar today. Long associated with the birth of modern physiology, Bernard's model of endocrinology gave much needed precision to the study of metabolic processes, and was key to the development of the theory of homeostasis, first posited by the American physiologist Walter Cannon in 1932. The concept of homeostasis explains the body's capacity to maintain a stable internal environment by the regulation of hormones, which had clear implications for definitions of health and illness, and medical understandings of both. Interestingly, as Roy Porter (1997: 562) notes, Cannon's concept not only crystallized the field of endocrinology but also provided a scientific basis for his conservative political philosophy that valued social stability and order. To be more precise, Cannon's 'discovery' of the body's ability to maintain itself through internal regulation provided evidence that such order and stability was present in biology and therefore an *a priori* principle that should govern human society.

This connection between biology, the endocrine system, and styles of knowledge was also apparent to the French physician and philosopher Georges Canguilhem who stated that 'interpreted in a certain way, contemporary biology is, somehow , a philosophy of life' (1994: 318).

Canguilhem's claims that the development of concepts such as that of internal regulation, and the consequent emergence of disciplines such as endocrinology and biochemistry, produced a specialist and standardized understanding of biological processes were a primary influence on Foucault, and his theory of biopower⁴ (Foucault 1981). The idea that there is an inherent system of regulation within biological organisms that can be known by the medical profession provides a baseline of 'normal function' which can then be used to epistemologically discipline the material body through rational and authoritative categories of normal and pathological or healthy and diseased. Moreover, for Canguilhem such approaches fundamentally ignore the relationship between states of illness as an ability to adapt to one's environment, that is, the ways in which disease and illness are socially and biologically intertwined in context. In turn, as Canguilhem's pupil Michel Foucault would later claim in *The Birth of the Clinic* (1973), concepts such as the endocrine system developed throughout the 18th and 19th century by the emerging medical profession were part of a broader revolution in the structure and production of knowledge, with direct consequences for systems of governance. Through the expert gaze of the medical profession, a rational and scientific 'truth' of the body, health and disease was established whose authority could in turn be deployed as a range of management strategies over individuals and national populations. As Nikolas Rose (2001: 3) notes, following Foucault, various biopolitical strategies were developed during the 20th century, such as free milk in schools and health visits for new mothers, in order to promote a healthy population and instil an ethic of 'care of the self' through both state intervention and the 'responsible' practices of individual citizens.

However, although the idiom of biological regulation still plays a pivotal role in the diagnosis and treatment of thyroid disease, it is increasingly apparent that the authority of this model - and medical authority more generally - is threatened, and has altered in response to a number of technical and social arrangements. As demonstrated by this thesis, far from making the diagnosis and management of thyroid disease simpler and more effective, thyroid function tests often result in dissatisfaction for both clinicians and patients. Often, these tests discipline the biological function of the thyroid

⁴ Foucault famously defines Biopower as 'an explosion of numerous and diverse techniques for achieving the subjugations of bodies and the control of populations.' (1981: 140).

gland 'to a fault', isolating biochemical activity as the key explanatory factor that informs diagnosis and treatment. The result is an impoverished view of the general health of the patients, in which their symptom reporting is undervalued and subjugated to the results of standardised thyroid function tests. In many ways this demonstrates Canguilhem's assertion that understandings of biology, health, disease and illness achieved through mechanical and technical measures based on fixed concepts and standards reduces the patient to an isolated organism whose health is not considered in terms of broader environmental milieus. Therefore, as the title of this thesis suggests non-compliance in the context of thyroid disease has meaning the beyond how individual patients interpret and perform the advice of their doctor through, for example, the ingestion of prescribed medicines and the observance of regimes disease management. Thyroid biology and pathology is itself is non-compliant because often when it is experienced by patients and grappled with by clinicians it does not behave how it is 'supposed to'. It does not follow the mechanical and technical rules established by biomedical epistemologies and as a consequence is constantly negotiated in an array of contradictory ways by the patients and professionals who encounter it.

Consequently, this study will investigate how the traditional biomedical model of thyroid disease operates in the 'post plural' context of the contemporary clinical encounter. Specifically, how the value of biochemical evidence provided by formal biomedical knowledge and practices is negotiated by patients who have sought and developed alternative models of thyroid biology and pathology that account for their personal experiences of thyroid disease. After a review of the appropriate theoretical literature and an outline of the methodological approach of the study (chapters two and three), chapter four introduces thyroid disease through an analysis of the relevant clinical and patient support group literature and data collected during a twelve month period of fieldwork. In chapter five, the evidence used to diagnose and guide the treatment of thyroid disease is presented and analysed using data from interviews, participant observation and secondary materials. On the basis of these findings I argue that much of the factual evidence encountered in the context of thyroid treatment is highly indeterminate and is deployed by various patients, patients' support groups and clinical professionals to construct competing orders of thyroid biology and pathology that fulfil a wide range of multiple and contradictory

rights, needs, responsibilities, duties, obligations, and desires. This chapter concludes by asking how thyroid disease is experienced and constituted by these various groups, and speculates on what kind of sociological model of 'medicalisation' can comprehend such a diverse range of activities. Which evidence is most highly valued by whom, in which context, and why? What forces discipline and shape these forms of evidence – especially when they are in tension, or even in direct conflict? And what are the practical implications of these competing evidentiary discourses for the categorisation and treatment of thyroid disease within the NHS? In chapter six, these issues of evidential indeterminacy are further examined through an analysis of how thyroid disease is practiced by clinicians and patients in order to justify various diagnostic and treatment outcomes. Drawing on the work of Annemarie Mol, this chapter concludes that thyroid disease is not a singular entity (i.e. a malfunction of the biochemical function of the thyroid disease that can be picked up by thyroid function tests), but a nexus of practices and 'modes of ordering' thyroid biology that interact with each other. By widening Mol's thesis to engage with Strathern's models of context in a post-plural world, I suggest a means of re-conceiving the relationality at the heart of medicalisation, asking what are the implications of these relational modes of ordering on the definitions and actual conditions of thyroid health?

Chapter 2

Literature review: knowing and doing the body

Within social studies of biomedicine, the body is increasingly recognized as being in a continuous state of flux, a site through which various social relationships, political constellations and deployments are enacted (Martin 1994, Martin 1987, Lock and Gordon 1988, Lock 1994, Fausto-Sterling 1985, Duden 1991, Rose and Novas 2004). Feminist analysis of the relationship between reproduction and embodiment (Oakley 1980: 40, Oakley 1984, Tew 1990, Boston Women's Health Book 1974, Ehrenreich and English 1973, Martin 1987) has been one of the most important strands in what has later come to be known as the 'sociology of the body' (Turner 1996, Shilling 2003, Featherstone et al. 1990). Current trends within the social sciences including an interest in the body, embodiment, as well as knowledges about the body such as medicine anatomy and biology owe a great debt to the insights provided by this literature. For example, the feminist sociologist Charlotte Perkins Gilman vividly demonstrated in her semi-autobiographical account of post-partum depression, *The Yellow Wallpaper*, how pregnancy and childbirth became the preserve of the male dominated medical profession as it emerged during the nineteenth century and became progressively pathologised, medicalised, and associated with mental illness. Subsequently, over the course of the twentieth century, an array of technologies, such as prenatal scans and diagnostic tests (see Rapp 1999a), have been introduced in order to manage perinatal care more extensively. In a pattern that has been repeated in other areas⁵ conception, gestation and childbirth have become 'medical problems' for which the monitoring of women before, after and during pregnancy is not only standard practice but has also become a social responsibility in order to secure the health of their child and future generations.

⁵ For example see Horwitz and Wakefield (2007) on depression, Martin (2007) on depression, Young (1995) on post traumatic stress disorder and Lane (2007) on social anxiety disorder.

This example demonstrates, as the sociologist Irving Zola (1972) claimed nearly forty years ago, the concept of medicalisation, which can be understood as a process through which the normal aspects of everyday life and individual behaviour came to be placed under the control of medical expertise and subject to various biomedical and technical solutions. In addition, as Ivan Illich famously claimed 'Professionally organised medicine has come to function as a domineering moral enterprise that advertises industrial expansion as a war against suffering' (1975: 133). As a result, he argues that we have been stripped of our ability to cope with disease, even when there are many instances in which medical interventions actually cause disease and suffering through a number of iatrogenic interventions. However, although a number of technologies have aided in the process of medicalisation, over the last two decades it is also clear that they have not colonised, but rather disrupted, traditional formulations of the body, health and illness. As the sociologist Aditya Bharadwaj states, a number of anthropological studies located at the sites of biomedical innovation (Rabinow 1992, Latour 1993, Strathern 1992b, Haraway 1990), have 'conceptualised the constructed nature of the biological, organic, natural and their shifting meanings' and thus 'problematized the post/ late modern bioscapes' (2008: 98). That is, technologies that have enabled us to 'engineer our vitality' (Rose 2006: 4), such as genetic diagnosis, mood altering drugs, new reproductive technologies, organ transplantation and regenerative medicine, have also resulted in multiple accounts of the body and concurrent practices, networks and identities (see Gibbon 2007, Rose 2003, Franklin 1997, Lock 2002).

Thus, as the medical anthropologist, Margaret Lock states with regard to the social scientific investigation of the body and the medical sciences, such projects are embarking on 'a rigorous questioning of what are so often assumed to be natural categories of thought and classification' (2001: 480). However, as Lock also notes, this re-conceptualizing of the body by contextualizing the truth claims of medicine is not the only aim of such projects. Lock states that it is also critical to investigate 'how and why certain representations become dominant at specific times and exposing the hegemony they exert over everyday life and practices associated with health and illness' (2001: 480). This project will attempt to contribute to this trajectory of social scientific enquiry. Specifically, in terms of how the technical innovation (or lack thereof) that currently constitutes the diagnosis

and treatment of thyroid disease within the NHS shapes the diagnostic categories of thyroid pathologies and the practices and experiences of clinicians and patients.

The following review will sketch out the key literatures that provide a theoretical framework through which these questions can be explored. First, the changing relationship between the body, systems of governance and subjectivity will be discussed. Second these relationships will be linked to debates that address the changing nature of medical authority, clinical practices and broad definitions of health, disease and illness. Finally, the empirical location of thyroid disease, specifically the relationship between professional and patient accounts and experiences, will be introduced.

Bodies, governance and subjectivity

In her account of the emergence of specifically female physiological drawings of skeletons, the feminist historian Londa Schiebinger (2000) asserts that, in 1796, the German anatomist Samuel Thomas von Soemmering published what he claimed to be the first illustration of the female skeleton. This claim, as Schiebinger notes, was remarkable as not even the father of anatomy; Andreas Vesalius claimed that sex was the determinant of bodily difference. For Vesalius, the organs and skeletons of the sexes were understood to be the same, excepting the reproductive organs and the shape of the male and female bodies. In this sense, the differences between the male and female body were neither essential nor fixed. In other words, as Schiebinger points out, although sex difference was acknowledged in the anatomical illustrations of the Renaissance, the need to differentiate between the sexes was not as urgent. The question then, as Schiebinger poses it, is why was the female skeleton discovered at this point? That is, what were the conditions that led von Soemmering and his contemporaries to distinguish 'sex differences in every bone, muscle nerve, and vein of the human body'? (2000: 25)

As Schiebinger goes on to discuss, the need to differentiate between the sexes anatomically was intrinsically linked to broader power relationships embedded in the natural law philosophy that shaped the political landscape of the time, and that was espoused by political philosophers such as John Locke (Locke and Goldie 1994). This style of political philosophy looked to

nature as a means of defining and sanctioning social conventions and roles. For instance, the female pelvis was depicted as noticeably larger than the male so as to confirm women's innate ability to nurture children. The female skull was represented as distinctly smaller in comparison to the male, in order to confirm women's inferior intellectual capacity. In this way, women were deemed 'naturally' to belong to the private sphere of the home, as they did not have the intellectual capacity to perform a role in the public sphere. The fabric of their very being was shown to be designed for another function altogether, to reproduce and rear children. Both of these 'anatomical facts', reinforced the exclusion of women from the public sphere and their confinement in the domestic sphere.

The 'natural facts' of the body, therefore, came to be used as evidence of the natural inequality between men and women and their positioning as innately 'rational' (men) and 'natural' (women) creatures. This 'feminization of feeling and masculinisation of reason' (Schiebinger 2000: 25) during the early development of modern science reflected and reproduced broader divisions of labour and power within European society. That is, the gender relationships embedded in the emergence of disciplines such as physiology, became a 'natural irrefutable fact' of scientific knowledge more generally. In this context, science became, as the Science Studies scholar Sharon Traweek (1988:162) phrases it, a 'culture of no culture' in which the masculine political culture, that was so fundamental to its inception and reproduction, was made to disappear by a dialect of objectivity and nature. Therefore, as the medical anthropologists Robert Hahn and Arthur Kleinman point out, the claims of biomedical practitioners, that their domain is distinct 'from morality and aesthetics, religion and politics and social organization' (1983: 306) are rooted in this historical cultural idiom. Consequently, biomedicine can be defined as day-to-day medical practice that is embedded in a 'value system characteristic of an industrial-capitalistic view of the world in which the idea of science represents an objective and value-free body of knowledge' (Lock and Gordon 1988: 3). Biomedicine, therefore, can be interpreted as an ethnomedicine (Hahn and Kleinman 1983), a socio-cultural system that makes, and is constrained by, nature – 'the product of a dialect between culture and nature'. (Lock 2001: 479)

The multiple and situated understandings of the body described above are consolidated in sociological and anthropological approaches to the body that can broadly be identified as 'social constructionist' (Nettleton 1995: 107). Such approaches include those that contend that the physical base of the body is shaped by social practices (Shilling 2003). For instance, if we again think back to the treatments that Toni was offered for her thyroid abnormality, the impacts of social practices are clearly visible. The surgeon wanted to remove her thyroid gland and the endocrinologist wanted to monitor it through blood chemistry. These differing approaches express the professional practices and expertise that define the medical sub-disciplines of these two specialists and, more broadly, how medical knowledge is organised.

The work of the anthropologist Mary Douglas has also helped to define this broad social constructionist approach. She argues that the social body mediates the perception of the physical body and vice versa. That is:

The social body constrains the way the physical body is perceived. The physical experience of the body, always modified by the social categories through which it is known, sustains a particular view of society. There is a continual exchange of meanings between the two kinds of bodily experience so that each reinforces the categories of the other. (1966: xiii)

These approaches identify the symbiotic relationship between the physical and social bodies. Specifically, they support the claim that biomedicine is an ethnomedicine - as its object, 'the body' is revealed to be the result of a particular relationship between biology and society. Therefore, returning to Lock's concerns, we should ask: how and why do certain representations of the body become dominant at specific times? What are the politics of these representations? And how do these representations impact on the practices associated with health and illness?

In the *Birth of the Clinic* (1976), Foucault provides a way of thinking about these questions by mapping the changes in the structure of medical knowledge. As Foucault demonstrates, the practice of autopsies from the 18th century onwards provided direct visual access to disease (Sullivan 2003: 1596). Opening up the body and finding physical disease in tissues meant that the physician was able to collect evidence of disease that was

independent from the patient's experience of it (Armstrong 1984). Therefore, the symptoms reported by individual patients became clues to a 'disease puzzle' that the doctor could solve using their specialist expertise. Subsequently, the power and the authority of the medical profession was established through these specialist practices as they alone had the knowledge and expertise to translate symptoms into diagnosis and, finally, treatments and cures. The consolidation of such techniques by the emerging and increasingly powerful medical profession also contributed to the development of a range of governmental technologies that exercised power through intervening in the fields of health and reproduction.

Foucault's concept of 'biopower' describes 'the explosion of numerous and diverse techniques for achieving the subjugation of bodies and the control of populations' (Foucault 1981: 140). Institutions such as hospitals, schools and prisons came to produce certain knowledges that in turn shaped the ways in which the bodies of the population were regulated, trained and maintained. The recording of mortality, morbidity rates and literacy rates, for example, monitored the population and provided a mechanism through which its health could be intervened upon by the state. For instance, Nikolas Rose (2001) identifies two such biopolitical strategies that emerged throughout Western Europe and the United States during the 19th and 20th centuries: the maximisation of a healthy population via the machinery of the State (for example, the compulsory medical inspection of school children and health visitors for new mothers) and the formation of the Eugenics movement. The Eugenics movement attempted to reduce the burden of disease and 'bad stock' on the nation by guiding the reproductive futures of individual citizens. It endorsed practices ranging from the promotion of 'good breeding' to enforced sterilization. Both of these strategies attempted to shape healthy bodies via biomedical practices, which in turn became a measure of the health and strength of the nation in comparison with other national populations. However, as Rose goes on to demonstrate, this way of knowing and governing the bodies of a population has been dislodged by the re-formulation of styles of governance and technical developments within biomedical knowledge in the late twentieth century. That is, although the health of the nation is still a central concern, the rationale for this interest is no longer framed in terms of a struggle between vigorous national populations. Within late modern Western societies, a nation's health is now cast in economic terms in correlation with the neo-liberal

paradigm of governance that is dominant within these locations (Barry et al. 1996). The 'body as a project' (Shilling 2003) and the maintenance of health through consumer choice means that individual citizens are responsible for maintaining their health through various lifestyle choices and health-promoting practices such as healthy eating, exercise and stress management. This contemporary formulation of governmental technologies can be clearly demonstrated in terms of current health policy initiatives within the United Kingdom.

In 2001 former banker Derek Wanless published a report commissioned by HM Treasury entitled *Securing Our Future Health: Taking a Long-Term View* (2001). The report was designed to assess the long-term resource requirements for the National Health Service (NHS) and concluded that, in order to deliver high quality health care, there must not only be an increase in resource provision, but also concurrent systemic reforms that would ensure these additional resources are used effectively. The aim of these reforms is to create what Wanless terms a 'fully engaged scenario' 2022. As Wanless claims:

Individuals are ultimately responsible for their own and their children's health and it is the aggregate actions of individuals which will ultimately be responsible for whether or not such an optimistic scenario as 'fully engaged' unfolds. People need to be supported more actively to make better decisions about their own health and welfare because there are widespread, systematic failures that influence the decisions that individuals currently make. (Wanless 2004: 4)

As described above, the 'fully engaged scenario' consists of high levels of public engagement with regard to personal health, resulting in an increase in life expectancy, an improvement in health status and confidence in the system due to the delivery of high quality care. According to Wanless, the key to realising this scenario is an increase in public health provision and specifically, the uptake of high rates of technology, particularly pertaining to disease prevention.

In 2003, Wanless received a further commission from the then Prime Minister Tony Blair, the Chancellor Gordon Brown and the Secretary of State for Health, Alan Milburn, to provide an update of the challenges envisaged in order to implement the 'fully engaged scenario'. Subsequently, in February 2004, *Securing Good Health for the Whole*

Population (Wanless 2004) was published. In the conclusion to this report, a shift was highlighted from the infectious diseases of the 19th and early 20th centuries to chronic disease throughout the 20th and 21st centuries, such as coronary heart disease and cancer, that are strongly related to lifestyle factors. Therefore, the cause of disease and explanation of symptoms have, to some extent, been relocated to the patient's social world (Armstrong and Caldwell 2004). Subsequently, 'quality of life' and patient experiences of illness are now considered to be an important 'outcome measure' for healthcare. Consequently, definitions of health and illness have been extended to include patients' evaluations of health. However, such 'quality of life' measures demand that patients take on a new set of responsibilities in order to maintain good health.

For example, the Wanless report called for 'NHS users' (patients) to make 'responsible choices' about their health by avoiding risk factors such as poor diet and smoking. This change in behaviour would in turn, it surmised, eventually take pressure off NHS resources by creating a healthier and lower maintenance population for it to serve. In 2006, the NHS launched the Patient Choice Initiative in order to facilitate 'responsible choices' that would promote the 'fully engaged scenario'. This project 'is designed to give you more choice about when, where and how your healthcare needs are met' (NHS Direct 2006). This includes the introduction of GPs with special interest (GpWSI's), who can perform minor procedures traditionally carried out in hospitals, electronic booking systems for appointments at both GP and hospital level and the choice between four local hospitals if an individual needs to be admitted for a procedure. All of these initiatives are designed to offer a more flexible healthcare service, as well as to allow patients to make decisions about when and where they receive their health care. This choice agenda, promoted by the ethos and organization of the NHS, means that the body, via an emphasis on disease prevention enacted at an individual level, is increasingly becoming a recognised site of social interaction and citizenship. For instance, in January 2009 the NHS constitution was released and its aims were as follows:

The Constitution establishes the principles and values of the NHS in England. It sets out rights to which patients, public and staff are entitled, and pledges which the NHS is committed to achieve, together with responsibilities which the public, patients and staff owe to one another to ensure that the NHS operates fairly and effectively. All NHS bodies and private and third

sector providers supplying NHS services will be required by law to take account of the Constitution in their decisions and actions. (The Department of Health 2009: 2)

Therefore, the constitution not only sets out responsibilities for the state with regard to healthcare, but also, responsibilities for patients or citizens who are expected to access and use health services in particular ways. Consequently, for the body of the citizen to access health services and remain healthy a contractual arrangement based around particular principles, values, rights and responsibilities has been established.

As suggested earlier, knowledge about the body shapes, and is shaped by, the same processes through which contemporary power relationships are enacted. For instance, the eugenic body depended on consistency and predictability: the vigorous, healthy body embodied the health and future prospects of the nation state that was measured in terms of vitality, potential and fitness. In the current era, in which power is organised less through the nation state and must be more responsive to global neoliberal economies, the health of the contemporary body depends on contingency and flexibility. Subsequently, as Emily Martin describes in her assessment of contemporary America's understandings of health and immunity, bodies are no longer understood as 'sharply bound machines' but as 'blurrily bound and complex systems' (Martin 1994: 40). This new understanding of biological mechanisms as informational systems, such as the immune system and, more recently the genome, has reinforced this contingent, 'reprogrammable' understanding of the fluid, or liquid, body. Moreover, this conceptualisation of the body can be interpreted as an example of Lock's claim that the body is constituted via dialectic between nature and culture (Lock 2001: 479). For example, the identification of genetic predispositions to certain illnesses can mean one is pre-symptomatic, generating issues of responsibility around care of the self. In this sense, it is assumed that the predictive nature of genetic information is intrinsically linked to some degree of choice. One might begin side-stepping environmental factors that might trigger 'the gene' for a disease, for example by altering lifestyle through diet (Martin 1998).

In response to these developments in biomedical knowledge and the political governance of health, the anthropologist Paul Rabinow (1992) predicted that 'the new genetics' would prove a powerful force in the

reshaping of society. Unlike previous theories of the relationship between the social and the biological, such as sociobiology (Wilson 1976), where the biological was a metaphor through which social projects were cast, the provisional, informational and manoeuvrable qualities of genetic information problematise the separation between society and biology. Subsequently, as Rabinow states:

If sociobiology is culture constructed on the basis of a metaphor of nature, then in bio-sociality, nature will be modelled on culture understood as practice. Nature will be known and remade through technique and will finally become artificial, just as culture becomes natural (Rabinow 1992: 99)

As Rabinow suggests, whereas the body and disease were once perceived to be natural – and thus fixed and inevitable – technological and conceptual innovation within the life sciences, such as the advent of genomics, means that these ‘facts of life’ are, to some extent, manipulable and controllable through intervention and technique. Consequently, the fixed category of the ‘natural body’ is no longer a tenable baseline on which social organisation can be orientated: once the body – as the object of biomedicine – has been revealed to be the product of artifice as much as nature, the authority of the biomedical sciences and the medical profession – founded on a conception of the fixed ‘natural body’ – is undermined.

As a result the, ‘natural facts’ of the body and disease are revealed to be, to some extent, social constructions - products of certain accepted ways of knowing and practicing. This unification of nature and culture within the work of theorists, such as Rabinow, means that the body is increasingly recognised as a site where social relationships and identities are performed. As Nikolas Rose and Carlos Novas (2004) argue in their account of ‘Biological Citizenship’, collectives are increasingly formed around biomedical classifications. Groups base themselves on specific conditions that an individual and/or members of their family suffer from in order to campaign for better treatment and access to services, to combat social stigma and to create networks through which fellow members of their community can be supported. The formation of such communities increases individuals’ knowledge about the specific shared conditions – both within the group itself and the wider community its campaigns address. This increased access to information regarding a condition in turn increases the responsibility of individuals to use that information in order to

avoid disease, a responsibility that derives from the control that one is now perceived to have over one's body through various self-employed healthcare strategies.

However, the agency that is often ascribed to the biosocial body has to be tempered and interrogated across a range of contexts. As Sahra Gibbon and Carlos Novas observe in the editors' introduction to their recent collection *Biosocialities, Genetics and the Social Sciences*:

Although Rabinow's much referenced concept of biosociality has provided a reflective starting point for this collection, the chapters collated here bring a much needed diversity to an understanding of the way that different biosocialities are brought to bear on a range of comparative arenas. Here the question of novelty, as well as ideas of contingency are not only subject to renewed critical scrutiny but are themselves brought into productive interface with what Sunder Rajan (this volume) usefully terms the multiple 'over determinations' associated with these developments. Indeed an important feature of this book is the way that a number of it's contributors shed light on the spaces, practices and persons which they suggest a notion of biosociality has 'failed to account for (Bharadwaj, this volume). For others, the multi-layered complexity and inherent ambiguity of the notion of biosociality has and continues to provide a useful entry point for examining these developments, which invites and encourages innovation in method, concept and critical analysis (Gibbon and Novas 2008: 14).

As Gibbon and Novas claim, the concept of biosociality has enabled us to think about 'how the social and the biological are being (co)-configured in relation to developments within the life sciences' (Gibbon and Novas 2008: 15). However, it is also clear that these reformulations of the social and the biological are multiple; hence the parameters of the concept should be consistently questioned and situated across an array of contexts, a project that this study is a contribution to. For instance, as will be demonstrated by this study, thyroid disease in the United Kingdom is organised and experienced around the technical deficiency of current diagnostic and treatment strategies – thus characterising a specific biosocial community perusing and enacting a particular kind of biosociality.

In order for the thesis to engage with ways in which the social and the biological (co)-configure each other within the context of clinical medicine, the following section will review work from within medical sociology that addresses the relationship between the social and biological aspects of

disease and illness, doctor and patient and explores how they structure the clinical encounter.

The changing nature of medical authority

Talcott Parsons, often described as the founding father of medical sociology, claimed that illness:

is a state of disturbance in the 'normal' functioning of the total human individual, including both the state of the organism as a biological system and of his personal and social adjustments. It is thus partly biological and partly socially defined (Parsons 1952: 431)

According to Parsons, the biological aspects of illness fall under the 'technical competence' (Mol 2002: 10) of physicians leaving the social aspects of illness – job functions, professional structures, doctor-patient relations – to the expertise of the sociologist. Therefore, within medical sociology disease is traditionally understood to be an organic cause for a disruption in the normal functioning of the body – an entity that can be seen and worked upon by the expertise of the medical profession. In contrast, illness is traditionally cast as a subjective condition. 'Illness' thus includes the social explanations introduced in order to account for personal interpretations of disease and the public dimensions of sickness. However, the various theoretical and technical innovations described above make it clear this distinction between disease and illness is no longer tenable. That is, the assumption that disease is simply an asocial disturbance of biology has become discredited by the recognition that the biology is the product of a dialect with the social. In the following chapter this problematization of asocial notions of biology and disease will be demonstrated and its consequences for the doctor-patient relationship will be considered.

In a study of the transmission of clinical methods through the practice of bedside teaching, Paul Atkinson recounts the following scene of a dialogue between a surgeon and his students concerning fluid loss in post-operative patients. After the surgeon asked students for sites of fluid loss, the following exchange ensued:

Student A: Urine.
Dr.: Yes. (writes on blackboard) How much?

St A: A litre and a half.
 Dr.: (writes on blackboard)
 St. B: Skin, sweating?
 Dr.: How much?
 St. B: Up to two litres.
 Dr.: In the tropics, yes
 (student laughter)
 St. C: A litre.
 Dr.: (writes on blackboard 900ml) Any other sites of
 fluid loss?
 St. D: The site of the operation?
 Dr.: Under NORMAL circumstances.
 St. B: Vomiting.
 Dr.: Under NORMAL circumstances.
 St. E: Stool.
 Dr.: In the stools. How much in the stools?
 St. E: About a hundred mil.
 Dr.: A hundred to two hundred mils. (writes on
 blackboard)
 (Atkinson 1988: 182)

Atkinson identifies two significant features of this sequence. Firstly, the surgeon adds up the responses from the students, and treats them as complete, when he calculates the final amount of normal fluid loss. Secondly, the list is constructed on the basis of what is considered to be 'normal' in the circumstances. For Atkinson, this process demonstrates that within medical practice disease is not only socially accomplished via the collaborative work of teachers and students, but also rooted in the idea that 'disease resides in the unequivocal deviation from universal and biological normality' (Atkinson 1988: 183). That is, as the philosopher of disease Georges Canguilhem (1978) recognised, the concept of biological 'normality', such as that described above, is a result of how normality is defined within the epistemological culture of modern medicine. Canguilhem claims that, biological normality is defined via limits derived from population-based data, suggesting that pathology is therefore an excess or deficit of a particular variable. (Horton 1995: 317). Therefore, the privileging of these numerical methodologies strengthens the authority of biology as a scientific endeavour, but also means that qualitative judgements about health are marginalised. As Canguilhem concludes, such definitions of normality provide us with limited understandings of health, where the functioning of the organism as a whole is surpassed by a focus on these limited definitions of normality of specific pathologies.

For instance, and as is explored further in this thesis, the focus on the negative feedback loop between the thyroid gland and the pituitary gland through the practice of thyroid function blood tests is problematic for two reasons. Firstly, often individuals who are found to have abnormal blood test results do not feel unwell, subsequently blood test results, particularly those that fall on the borderline of thyroid normality/abnormality, cause distress and result in the diagnosis of a disease that has no discernable symptomatic manifestation for the patient and is therefore difficult to manage. Secondly, this focus on strict definitions of 'normal' thyroid function is also problematic for patients who display symptoms but whose blood tests results fall within the normal or borderline range. The strict adherence to blood test results in order to diagnose thyroid disease means that such patients are often left in a no-mans land where they are symptomatically ill, and slightly biochemically abnormal, but cannot secure treatment because their individual manifestation of disease is not 'abnormal' enough.

If we think about thyroid function blood tests as artefacts of the numerical methodological culture of contemporary medicine, it is possible to conceive of them as ethnomedicine – a set of practices specific to a socio-cultural system that defines and impacts upon diseases in a particular way. As Bryan Turner notes in his sociological analysis of modern medicine:

Disease is not a fact, but a relationship and the relationship is the product of classificatory processes: a disease pattern is a class, or niche in a framework. This framework is a means of approaching or organizing crude experience, that is, for dealing with every day events in the most satisfactory way (Turner 1996: 200)

As discussed above, medicine, the body, and disease are inextricable from the power-knowledges, disciplines and epistemic formations that generate and reproduce them (Foucault: 1973). The objects of medicine are discursively shaped through language and practice, systems of classification, and hierarchies of diagnostic symptoms keyed to specific techniques of measurement and calibration. The suggestion that disease is simply the result of an organic cause that can be objectively evaluated by science and its methods is, from this perspective, naive. Specifically, as this thesis will seek to identify, not only are the diagnostic categories discursive they are also highly uncertain and are often shaped during the course of

clinical practice generating a range of, often competing, diagnostic categories and identify practices.

In her comparative study of the menopause in Japan and the United States, the anthropologist Margaret Lock (1988) notes that increasing life-expectancy is of interest to many governments due to the effects of this trend for healthcare systems in terms of resource allocation and expertise. The menopause, or the end of menstruation, can be seen as a signifier of ageing that concretizes this concern. As Lock demonstrates, much of the work and data collection that has occurred to define the menopause is based on the self-reporting of a small number of North American women who describe symptoms of emotional and physical distress during this time of life. Moreover, many of these women have had hysterectomies, a highly invasive procedure that explicitly medicalises the menopause. As a consequence, the symptoms and diagnostic categories of the menopause are biased towards a narrow and atypical sample. The resulting categorisation of menopause has become the basis for the treatment of the end of menstruation in North American and European clinical medicine. HRT (Hormone Replacement Therapy) is commonly prescribed to reverse the effects of these 'oestrogen starved' bodies, such as heart disease, osteoporosis and Alzheimer's disease. However, as Lock also points out, not only are the consequences of these ageing female bodies highly contested, but the compliance rate for HRT is low - at approximately 15% for all women who are passing through this time of life. As this comparative study reveals, not only are the iatrogenic effects of HRT generally ignored inside of this context, but also these symptoms are not always present outside this reified epidemiological population.

In Japan, by contrast, the end of menstruation, or *kōnenki*, is not commonly marked by the experience of 'classical' menopausal symptoms, such as hot flushes or night sweats. More commonly shoulder stiffness, headaches and dizziness are reported. Moreover, the cause of the disease is strongly associated with factors connected to the role of middle-aged women in Japanese society, rather than the physiological changes associated with the end of menstruation. In fact the rise of the nuclear family, the decline of the extended family, increased wealth and subsequent leisure time, are all factors which are thought to contribute to the 'lack of a role' for middle-aged women. Thus the menopause becomes a syndrome whereby the

vulnerability of the ageing body and its propensity to stress is magnified by the social role, or lack thereof, of middle-aged Japanese women. Consequently, menopausal syndrome is construed as a luxury disease suffered by women who have too much time on their hands due to the disappearance of the extended family, whom they would have traditionally attended to.

Furthermore, the physicians who see these symptoms in the Japanese context do not attribute the lack of Euro-American symptoms to an error in the accounts given by Japanese women, but to a biological difference, due to factors such as environment, diet or genetics. This results in a subjective experience of the end of menstruation in Japanese women that is different to those commonly experienced in other contexts. As Lock concludes, what this comparative account of the menopause demonstrates is not only the construction of bodies and diseases by culture, but also the construction of culture by bodies. Whilst the end of menstruation is defined by the North American medical paradigm as a problem due to the ageing process, damaging the 'normal optimal pre-menopausal body', it is also qualitatively constructed, as demonstrated in the Japanese account, by the experience of the body in other locations. In order to account for this dialect between nature and culture, Lock has created the concept of 'local biologies' that refers:

To the way in which embodied experience of physical sensations, including those of well being, health, illness and so on, is in part informed by the material body itself contingent on evolutionary, environmental and individual variables. (Lock 2001: 483)

Therefore, as with Rabinow's concept of 'biosociality', 'local biologies' is an analytical concept that offers a specific model of the relationship between nature and culture, and specifically disease and illness. Consequently, following on from this re-framing of the relationship between nature and culture, this study will identify how the specific regulatory structures, diagnostic and treatment technologies and doctor-patient relations within the NHS shape the boundaries and experiences of thyroid disease.

As described previously traditionally medical sociology has tended to understand the realm of illness to be a social response to disease, which was considered the domain of science. As Annemarie Mol notes, however,

'when sociologists realised the power that a strong alliance with physical reality grants doctors' (Mol 2002: 9) they began to question the sanctity of disease as a realm purely of science. This change in the focus of medical sociology – from illness to disease/illness – can be mapped out via the fortunes of Talcott Parsons' concept of the sick role (1952). For Parsons, a structural functionalist, all social phenomena is either a stabilizing influence on society or a threat to it, or to what he more specifically defines as the 'Social System'. In the case of sickness, Parsons asserts that being sick is realized as a specific role, which consists of four main constituent parts. Firstly, the sick individual is granted temporary relief from certain social duties, such as work, via the validation from other sanctioned roles, such as the doctor. This validation, therefore, constitutes the second part of the sick role, which is the necessity of the sick person to seek professional medical help. In turn, this relationship should be entered into an understanding that it is asymmetrical, in that the patient will act on the advice of the doctor and comply with all treatments that the medical profession prescribe for recovery. Finally, if all of the previous conditions of the sick role are met, the fourth criteria can be achieved, in that the sick role is meant to be temporary and will only last as long as the medical treatment prescribed – which it is assumed will be successful and will result in a return to work and a 'normal' social role.

Whilst it has been noted that Parsons meant the sick role to be applicable only to western protestant societies (Shilling Chris 2002), the fact remains that the sick role is a highly limited concept through which to assess health and disease, especially within a contemporary biomedical context. That is, not only does the sick role depend on a highly normative account of social relationships and organization, but also it cannot account for contemporary models of disease (e.g. chronic, pre-symptomatic) or professional and lay interactions (e.g. expert patient movements, complementary and alternative therapies). These changes in the understanding of disease and the congruent configuration of doctor-patient roles are well demonstrated in Michele Crossley's (1998) study of members of a support group who were living long term with a HIV-positive diagnosis. The National Long Term Survivors Group (NTLSG) is a patient support group whose members approach their disease through a discourse of empowerment. This can be defined as an 'insider view' of illness' that is related to the modern philosophy of self-help groups. This approach questions expert knowledge

and privileges individual experience of the management and treatment for disease. This way of inhabiting disease categories, therefore, is tied to broader epistemological shifts within biomedicine. The predictive and flexible nature of emerging biological innovations, and resultant forms of social organization, challenge the authority of the medical profession and leave it open to interpretations and dispute by lay individuals and groups.

Improved treatments mean that there is an extensive gap between diagnosis and the onset of symptoms of HIV. This in turn challenges the first proviso of the sick role – the temporary relief from work. As Crossley's interviews demonstrate, most of the participants of the study were exempt from social responsibility, such as paid employment, and had chosen to give up work as a pre-emptive move, a preventative measure that would remove the pressure of work, thereby potentially increasing the amount of time they could experience pre-symptomatic health. This subversion of the sick role is linked to the rise of 'quality of life' as an important 'outcome measure' for medical intervention. As is the case with HIV, preventing premature death is no longer the one and only concern of medicine. The eradication of acute disease and the rise of chronic disease and the focus on improving life, rather than preventing death, has become a central concern of the profession. As Armstrong (1984) points out, subjective patient meanings are now examined alongside pathology in a clinician's search for the cause, impact and solution for illness. In addition, developments within bioethics (respecting the autonomy of the patient) and the lifestyle choices associated with the management of chronic disease locate the authority to judge quality of life within the expertise of the patients (Sullivan 2003). As a result, the relinquishing of social duties, such as work, is under the control of patients, as they are the best judges of what will improve their quality of life. Thus, whilst the temporary nature of the sick role does not fit with the uncertain timescale of pre-symptomatic HIV, the exemption from social responsibilities, such as work, and the obligation to seek medical help are still present. Although, the terms of exemption are now broader and are related to the expertise of the patients as well as the medical profession. As the study goes on to demonstrate, the dependence on the medical profession is not necessarily harmonious. In fact the empowerment approach adopted by groups such as NLTSG directly opposes the authority of the medical profession.

Many members of the NLTSG expressed scepticism at the authority of the medical profession. As Crossley notes, haemophiliacs who had contracted HIV from contaminated blood were unsurprisingly critical of the medical profession. However, beyond this group, and more common to the rest of the interview cohort, was a general cynicism due to the uncertain nature of the disease progression and its treatment. For instance, the measurement of CD4/T4 counts⁶ was always highly contested by many patients as it did not seem to correlate with their own assessment of their health. As one patient in Crossley's study explains:

...Theoretically my T4 cell count is low but that seems to have no basis in real life, in the real world...they say that if your T4 cell count is below a level, then you are ill, you know the Americans set up the control definitions of AIDS as a T4 cell count below 300. Mine has been around 200 for years and nothing happens...I don't think it makes a great deal of difference...it's a normal thing...(Crossley 1998: 516)

This scepticism was also manifest in the patients' opinions of how the medical staff conducted themselves. Patients often reported feeling that the full extent of their disease, and the treatments they were expected to comply with, were not adequately revealed to them. They further complained that their knowledge of their disease was ignored by physicians in the course of treatment. Often patients made sense of their disease and state of health by responding to 'gut feelings' and 'listening to their body', via an idiom of positive thinking, whereas, for physicians, such individual and subjective testimony was at odds with the necessity for objective reliance on established protocol. Similarly, the social roles and rules HIV-positive individuals were expected to adhere to in their daily lives were also ignored or rejected by medical professionals. For instance, heterosexual haemophiliacs tended to affirm their right to have children and homosexual men refused to relinquish their right to sexual freedom. As Harry, an HIV-positive patient, describes the imperative for him to reclaim his identity in Crossley's study:

I remember one day I was at Open Door, I said, 'I must take back my sexuality' and I borrowed someone's leather jacket and went up to the Heath and stood by a tree and I would not leave that tree until I had got fucked rotten, to put it bluntly. I had to take it back, take the power back to me, not let the

⁶ CD4/T4 lymphocytes are used as a marker of disease progression

doctors or anybody else, there are plenty of people who say, you mustn't, you drop it all now (...there was a lot of guilt associated and fear, 'you don't do this and you don't do that')...(Crossley 1998: 522)

Crossley concludes that certain aspects of the sick role are still useful as a way of making sense of the experience of sickness. Although disdain seemed to be the most common 'insider view' with regard to patient opinions of the medical profession, the view tended to over-exaggerate the independence of patients from the medical profession. As Crossley notes, there is a disproportionate amount of health service provision dedicated to HIV/AIDS within the United Kingdom. The continued health of HIV-positive individuals is wholly dependant on continued innovation within the field to keep them alive. Moreover, it is also noted that the way in which the medical profession is demonised could be attributed to the construction of a HIV identity that can resolve 'personal worries, fears and vulnerabilities...by mentally passing them onto some other person or group external to the self' (Crossley 1998: 528). By casting medical professionals as 'non-empowered' others, HIV-positive individuals build an empowered self in order to manage their terminal disease.

Finally, whilst the desire to applaud the positive and empowered ownership of the HIV diagnosis by such patient movements is compelling, as Crossley concludes, that approach is only available to those who have certain material and cultural resources to draw upon. This individual-empowered approach to HIV, and disease more generally, has a massive implication for the wider 'social body'. As Crossley notes, with reference to the work of Nancy Scheper Hughes (1994), there is a clash between the 'rights of the individual' and the social ethic required for HIV prevention measures. In other words, what are the broader implications of Harry's actions on the Heath, beyond his individual need to empower himself with regard to his HIV-positive status?

In conclusion, as Lock's comparative work on menopause in Japan and the United States demonstrates, disease and illness (biology and society) are socially accomplished categories. Rather than disease being defined as 'a fact' it is understood to exist in a dialectical relationship with illness, and with the wider environment, including factors that affect both individuals and populations. From such a vantage point it becomes possible to understand how representations of disease are tied, for example, to the dominant

hegemonies of the time and place in which they are manifest. Similarly, as Crossley's study of the members of the NLTSG demonstrates, an empowered approach to a HIV-positive diagnosis hinges on substantial resource provision in order to stay alive. So whilst it is undeniable that empowered, flexible, contextualized and contingent definitions of disease and illness are apparent, it is important to remember that the material conditions of these entities are imperative to their particular manifestation. Therefore, in assessing the construction and politics of health and medicine, it is necessary to interrogate the changing practices that constitute them.

Together, these studies underscore the importance of some of the wider points emphasised throughout this chapter, including the complex co-determination of bodies, illness, knowledge, and care. As studies charting the biosocial dimensions of illness become more numerous, and more diverse, it becomes possible also to attend to the distinctions raised by Gibbon and Novas (2008) concerning the limits of the biosocial model, and the need for ongoing innovation in understandings of what Rose describes as the 'somatic ethics' (2006: 6) of approaches to disease. That is, the ways in which the new possibilities of the body bought about by biomedical innovation, coupled with devolution of the responsibility of the management of human health from the state to the individual, results in us relating to ourselves through our corporeality. As Rose claims the way in which '...the corporeal existence and vitality of the self has become the privileged site of experiments with the self' (2006: 26).

Practicing Medical Knowledge

The recognition that corporeality is a key site of subjectivity results in disease becoming a site where social-scientific work can be performed, and, subsequently, where the politics and contingencies of the traditionally asocial sphere of scientific knowledge can be assessed. This interpretation of biomedicine as a realm open to interrogation is particularly useful when addressing the rich and highly productive gap between the value-free claims of clinical science and the application of such knowledge in clinical practice (as this study of thyroid disease aims to do). In the following section, the conceptual separation between 'knowing' disease via clinical science and 'doing' disease via clinical practice is extended through a

consideration of the modes of body knowledge techniques articulated in the work of Annemarie Mol (Mol 2002). Latterly, the empirical field of thyroid conditions will be introduced as a location where such broad theoretical questions can be addressed. Firstly, however, a discussion of work that looks at the relationship between clinical scientific knowledge, and how such knowledge is applied in practice, will be conducted.

In their study of Familial Hyperlipidaemia (FH), a condition that interrupts the normal metabolism of blood fats, Helen Lambert and Hilary Rose (1996) introduce the categorisation 'peculiarly scientific' to describe the absent symptomatology of a disease that can only be 'seen' post mortem. That is, although FH can cause premature death from cardiac arrest/coronary heart disease (CHD), no obvious symptoms of disease are present before the onset of these terminal events. FH consequently has no symptoms in the daily lives of its sufferers and is only brought into being by laboratory indicators – blood tests that measure raised lipid levels. FH can be thought of as a 'disembodied' disease, leading Lambert and Rose to ask: 'How...do people make sense of knowledge from the medical science which does not look to, or speak to about the body?' (1996b: 65)

It is only with the advent of effective therapeutic responses that the genetically transmitted inability to metabolize fats has become a site of medical intervention in the case of FH. The success of medications to lower lipid levels, associated dietary measures, as well as identification of the aetiology of the disease, means that the condition can be seen within a biomedical framework and therefore successfully treated. However, as Lambert and Rose are quick to point out, even this seemingly new ability to classify FH as 'a disease' is relatively partial, due to its genetic and clinical heterogeneity. In contemporary medical practice, a differential diagnosis of the specific type of FH is rarely sought, and treatment consists of monitoring the lipid profile over time and implementing dietary and therapeutic responses, as and when they are necessary. In this sense the practices of treating FH, rather than how it is perceived as a genetically rooted disease, are what constitute FH within the clinical environment. In addition, this active process of knowing disease through practices also reflects how such a disembodied and pre-symptomatic condition is embodied by patients. Through in-depth interviews with FH patients, it became clear to Lambert and Rose that the science of FH was understood

by patients as an involved process, where individuals applied their own knowledge, personal and family medical histories to cope with and manage the condition. For instance, for most FH sufferers the disease is understood in terms of its effects on physical health, rather than its metabolic characteristics or genetic determinants. Moreover, in the daily lives of individuals who suffer from FH, general dietary practices, encouraged by various health policy initiatives to reduce the occurrence of CHD in the general population, are used. Often informal networks of family members, who all share the disorder, provide forums where treatments and test results can be compared and new information sought. Again, this affirms the importance of existing social networks and relationships in the process of managing this disembodied disorder.

What Lambert and Rose's investigation into how such a 'disembodied' and 'peculiarly scientific' disease is understood demonstrates is the remaking of scientific knowledge through clinical practice. That is, although clinical science reifies and defines what 'a disease' is, in the realm of clinical practice these categories are not so straightforward. In practice, other concerns take over and practical strategies are deployed in order to treat disease. In many ways, the disconnection between the science and practice of FH is indicative of the character of contemporary medical knowledge. Scientific innovations may enable the identification of FH at a molecular, chemical and cellular level, but this in itself does not directly translate into how FH is experienced as an illness through symptoms. FH is highly tentative: it is treated before it becomes manifest symptomatically. Strictly speaking FH is not treated at all, but rather managed through the introduction of individual behavioural changes (e.g. diet) alongside pharmaceutical interventions, such as cholesterol lowering drugs.

As discussed above, the predictive nature of such disease information suggests that the boundaries of the biological body and the certainty of biomedical knowledge have to some extent been compromised. For example, within social studies of biomedicine there is an extensive body of literature that examines a number of syndromes that have no clear pathological basis and subsequently 'are pervaded by medical, social and political uncertainty' (Dumit 2006: 577), such as myalgic encephalopathy (M.E) (Cooper 1997), chronic fatigue syndrome (CFS) (Hayden and Sachs 1998), repetitive strain injury (RSI) (Arksey 1998), and Gulf War syndrome

(Cohn et al. 2008). Consequently, as the medical sociologist Sarah Nettleton (2006) states, such studies enhance and inform broader theoretical concerns with regard to ways of living in the postmodern era. As the sociologist Zygmunt Bauman (1991) points out, in the postmodern era uncertainty is no longer a temporary nuisance but a permanent and irreducible state. Fluidity, flux and uncertainty are inherent characteristics of the process of globalization and, therefore, traditional and stable forms of authority and expertise have been reduced so that attempts to control such uncertainty, result in further uncertainty and disorder. As Renee Fox's (1980) work on medical uncertainty demonstrates the more advanced medical technologies become the less they are able to deal with clinical uncertainties – the boundaries of life are pushed and manipulated to such an extent that further fear and threats to life are created. Therefore, the risk information that typifies contemporary medical knowledge and practices alters the authority of medical knowledge and the medical profession. For instance, in the case of FH, predictive and uncertain disease information can result in clinical decisions that are often complex and necessitate shared decision making between doctors and their patients. This, to some extent, subverts the traditional 'asymmetrical' relationship between doctors and patients identified by Talcott Parsons, and subsequently threatens the authority of the medical profession.

Stabilizing illness

Following the work of fellow sociologist Eliot Freidson (1972), David Armstrong (2007) claims that clinical autonomy has remained the defining characteristic of the medical profession's authority despite the many challenges to its traditional sources of affirmation. As Freidson claims:

The profession bases its claim for its position on the possession of a skill so esoteric or complex that nonmembers of the profession cannot perform the work safely or satisfactorily and cannot even evaluate the work properly. On this basis, nonmembers are excluded from practice and evaluation. Given the exclusion and its implied basic concession of autonomy, I would argue that in spite of any formal administrative framework imposed by the profession, autonomy in controlling its technique allows it to temper many elements of that framework beyond both the intent and even recognition of its planners and chief executives. This is particularly the case for medicine, where dangerous consequences can follow upon improper

work, and where the claim of emergency and of possible dangerous consequences is a potent protective device. (1972: 45)

In the last few decades, however, others have argued that medical authority has declined in line with the more general changing nature of technologies of governance described previously. The economic rationalization of health services, and the emergent casting of patients as consumers has been claimed to make doctors accountable to fiscal imperatives and patient desires rather than their professional judgement and traditional expertise. Both of these factors, it is assumed, have contributed to the deprofessionalisation (Haug 1973) and proletarianisation of the profession (McKinlay and Stoeckle 1988). From this perspective, doctors have been seen to take on the characteristics of employees rather than professional, self-governing elite. For example, in the United Kingdom health policy initiatives, such as 'Patient Choice', that attempt to empower patients to make decisions about their health and healthcare have dislodged the authority of the profession. Shared decision-making between doctors and patients is now considered to be integral to a successful contemporary clinical consultation (May et al. 2006). Within the parameters of such an initiative, the traditional asymmetrical relationships between the 'expert doctor' and the 'lay patient' are no longer straightforward nor even desirable. However, as Freidson recognised, this story of the decline of medical authority is not so straightforward, nor is it reflected in the earning power of the profession and their continued self governance through institutions such as the British Medical Association (BMA) in the United Kingdom. Freidson suggests that, far from losing control, the medical profession, particularly the academic elite, have been deploying strategies to maintain professional authority. This has taken the form of gaining control of their individual members through the formalisation of professional practice. The most powerful and recognisable form of this professional formalisation is the evidence-based medicine (EBM) movement. This term, first coined by a group of clinical epidemiologists at McMaster University in Canada, is defined by D.L. Sackett as:

...the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients...integrating clinical expertise with the best evidence in making decisions about the care of individual patients (Sackett DL 1996).

EBM establishes scientific research, rather than an individual clinician's experience, as the basis for medical decision making. The traditional view that, by virtue of professional status, physicians can make decisions as to what treatment is necessary is questioned. It is recognised that clinical decisions can be 'highly capricious affairs, well in need of some support' (Berg 1998: 226), a situation that it is thought can be remedied by the practice of EBM. The production of 'transferable, evaluatable and scientific' (Berg 1998: 227) evidence by the clinical elite, and the establishment of tools such as treatment algorithms and clinical practice guidelines, attempts to redefine and re-establish the profession's authority. That is, they become the producers and assessors of a specialist evidence-based culture of knowledge production, defining the terms of good clinical practice and subsequently asserting the authority of the profession. EBM claims authority by developing professional standards for individual clinicians to follow. It is assumed this will mean that better diagnostic and treatment decisions are made. As Marc Berg notes in his review of the development of post-war medical practice:

...the flawed physicians mind is increasingly seen as the primary source of medical practice's problems. More and more, the escalating costs of medical care, the public's dissatisfaction with medical practice and the suboptimal quality of care are being transformed into problems which result from the individual physicians mental incapacities. (1995: 226)

This disciplining of 'rogue physicians' through the practice of EBM fulfils two aims. Firstly, it re-establishes the professional authority of the medical profession, albeit the clinical academic elite, by producing specialist reified knowledges and practices that the profession as a whole can adhere to. Secondly, its rhetoric of objectivity, through the application of reliable scientific evidence in clinical decision-making, attempts to guard against capricious clinical decision making. However, as has been discussed previously, such rhetoric of objectivity can be considered culturally specific. That is, EBM extends the rhetoric of objectivity by positing a value-free, experimentally validated and independent body of knowledge of both clinical and economic benefit and, further, posits this value as self-evident.

In 2004, the sociologists Eric Mykhalovsky and Lorna Weir stated in their critique of EBM that:

Evidence-based medicine (EBM), the project of reshaping biomedical practice by creating an organizing presence for clinical research within medical decision making, has taken the health care world by storm... EBM has grown into one of the most important and successful initiatives to recompose contemporary biomedical reasoning and practice. The evidence-based movement, as some have described it, has been met with remarkable enthusiasm on the part of the elites in academic medicine. EBM has been formally incorporated into editorial policies, has spawned new journals and approaches to reporting biomedical research, and is now routinely taught throughout medical schools in North America, the UK and parts of Western Europe...(Mykhalovskiy and Weir 2004: 1059)

However, they go on to claim, the social sciences have been slow to interrogate the EBM movement, which has come to dominate medical practice. At the time of the article's publication, social scientists had only really engaged with the movement using two traditional and 'empirically thin' approaches – political economy and humanism. Sociologists using the political economic (Armstrong and Armstrong 1996, Leys 2001, Light 2001, Navarro 2002) approach have analysed EBM as a naive endorsement of the free market principles of capitalist social organization. Ideologically, EBM buttresses the authority of the medical profession by reifying the scientific nature of medical knowledge and devaluing more traditional forms of authority predicated on clinical experience. The privileging of epidemiologically-derived statistical calculations as evidence has redefined medical outcomes as numerical, thus tying them closely to fiscal imperatives and administrative control. EBM's rationalising ethos is also challenged within more humanist analyses (Frankford 1994, Belkin 1997, Tanenbaum 1996) of the movement, for example for diminishing the doctor-patient relationship. From this perspective, EBM's over-emphasis on quantifiable, repeatable and scientific evidence transforms patients into standardised units and clinicians into data managers and technicians. Mykhalovsky and Weir ask 'how the formal rationality of EBM connects with clinical reasoning' and in doing so seek to 'move towards a more complex understanding of EBM's relationship to clinical practice'. (2004: 1064). Similarly, Timmerman's and Berg's (1997) account of oncology and a Cardio Pulmonary Resuscitation (CPR) protocol demonstrate how these semi-standardized practices have to be significantly adapted to suit specific local contexts and practices. Far from becoming cookbook medicine, clinical practice is being increasingly understood as a meaning-making activity where the formal knowledge of EBM is adapted to local uses by individual clinicians (Timmermans and Mauck 2005) .

This recognition that the contextual nature of evidence is applied within the clinical encounter is also supported by David Armstrong's account of how G.P.s in the United Kingdom introduce new anti-depressants to their prescribing repertoire (2002). As Armstrong demonstrates, the population-based evidence used to justify and recommend the new anti-depressant drugs is only one of many factors GPs take into account when prescribing treatment. Often they prescribe on the basis of the circumstances of the individual patient. For instance, they may prescribe drugs to an individual on the basis that side effects such as drowsiness would actually help them with their symptoms of sleep disturbance. As Armstrong concludes, the complex arrangements of social and scientific factors in the clinical decision-making process demonstrate a tension between evidence-based and patient-centred medicine. The 'formalised accounts' of what is the most efficacious anti-depressant drug are defined on trial evidence derived from populations of patients, but this may be far removed from the clinical decisions and prescribing protocols used by G.P.s, which are often based on the individual needs of the patient identified over time through doctor-patient interactions and relationships. Such studies suggest that not only is there a conflict between 'experience near' and 'experience far' clinical evidence, but also that it is tied to the quality of the relationship between patient and doctor – something that not only is not measured by EBM criteria, but indeed cannot be.. From the perspective of more qualitative studies, the character of the clinical encounter may be a better indicator of successful treatment than strictly objective indicators based on standardised models of patients, doctors and disease. Likewise, the stereotype of EBM as the 'heartless application of scientific knowledge' (Mykhalovskiy and Weir 2004: 1064) can be reappraised as a complex set of relationships between social and scientific orders.

This relationship is also apparent in the production of clinical evidence and decision-making tools before they are even circulated for use in clinical practice. In the anthropologist Tiago Moreira's ethnographic study of a research unit that develops systematic reviews of healthcare, the production of such evaluative devices are approached as meaning-making activities. As Moreira demonstrates, the systematic review is also a translation device, converting one form of data into another:

[K]nowledge making in systematic reviewing is structured upon parallel attempts a) to disentangle data from the milieus in which they are commonly found (databases, texts, other research centres, and b) to re-qualify that data through comparison across a variety of 'platforms' (tables, graphs, equations, and political controversies). (Moreira 2007: 180)

The evidence collected from the vast amount of published data on any given health intervention during the course of a systematic review is transformed by the practices of the reviewers, while at the same time reinforcing a new standard of neutrality. Evidence is evaluated, extracted and represented in forms that fit in with the requirements of evidence-based methodologies, as well as the practical requirements of clinical practice guidelines, which require objective criteria for comparison across vast aggregate data sets.

Since a systematic review entails an analysis of all the published data on a single research question, the aim of a review is to appraise, select and synthesize all high quality research evidence relevant to that question. The review is intended to provide the best evidence on which to base evidence-based tools, which in turn form the base for more rigorous, transparent, standardised, and often cost-efficient clinical practice guidelines (CPGs). According to the Institute of Medicine, a health policy group created by the National Academy of Sciences in the United States, Clinical practice guidelines are 'systematically developed statements to assist practitioner and patient make decisions about appropriate healthcare for specific clinical circumstances' (Institute of Medicine, Committee on Clinical Practice et al. 1992). Whereas CPGs were once based on the expert opinion of senior figures in the medical and scientific fields, they are now based on systematic reviews of published data which adhere to the current tenets of evidence-based methodology. Published meta-analytical data is placed at the top of a hierarchy starting with meta-reviews of randomized controlled trials (RCT's)⁷ and finishing with the results of expert committee reports and opinions. The privileging of meta-analysis in RCT's over expert opinion neatly summarises the core identity of the EBM movement, namely that it is a fundamentally scientific endeavour aimed at improving clinical

⁷ According to health service researchers Sibbald and Roland (1998) 'Randomised controlled trials are the most rigorous way of determining whether a cause-effect relation exists between treatment and outcome and for assessing the cost effectiveness of a treatment'

guidelines through evidence that is based on population-derived, quantitative analysis and that has been peer reviewed.

However, the technocratic hyper-rationalisation of EBM meta-review methodology has led some commentators to conclude that the resulting guidelines are 'evidence biased' rather than 'evidence based' (Moreira et al. 2006) and that the aim of EBM to remove clinical experience and opinion from diagnostic decisions does not enhance but reduces the quality of medical care, as good doctoring is considered to be an artful, as much as a scientific, practice. This charge that EBM has paradoxically inaugurated an age of 'evidence biased' rather than 'evidence based' medicine recapitulates older discussions about what constitutes good medical practice and professional legitimacy. Is it based on the individual physician's clinical expertise – their 'personal and private magic' (Gordon 1988: 257) – or should good professional practice be based on the ability of the physician to use clinical scientific knowledge to make better patient care decisions?

As Moreira's qualitative ethnographic study suggests, the production of guidelines is a key component of the neutrality-driven western scientific 'culture of no culture' described by Sharon Traweek (1992). The authority of systematic reviews is derived from their putative neutrality, based on their presumed ability to mine evidence from the vast, complex, contradictory mass of published data and reconstitute it as a more objective meta-conclusion. For instance, as Moreira demonstrates, 'at the core of systematic reviewing is the practice of neutralising the powers incorporated in the texts by their authors'. (2007: 186). Research papers are 'made docile' (2007: 181) through practices of data abstraction which reviewers actively seek and construct the information they require. For example, the methods section of a paper is initially assessed in order to ascertain the quality of the study, measured by its evidence-based robustness, before the results are eligible for inclusion in the meta-review, from which many 'inappropriate' studies are thus excluded. Research is also classified by the trial name, the protocol of the trial, the trial aims, the characteristics of patients included in the trial, the methodological quality, the comparability of trial subjects, the health measurements of subjects at the start of the trial and, finally, numerical end points of the health of the trial subjects. All such details are entered into a 'data abstracting template sheet', a table that is

used to standardise the classification of research according to a fixed and limited set of criteria. The abstraction template thus functions to identify and promote consistencies within and across trials, facilitating a construction of the best neutralised evidence. In other words, the abstraction template consolidates what the review procedures define as meaningful within the published evidence. As Moreira concludes, this process fundamentally reconstitutes the original research:

The reviewer picks out this information and actively transforms it into data. Through this process, the paper loses its material appearance of a well ordered sequential set of arguments, turning it into a figuration of interrelated, graphically linked marks and comments. In a more superficial way, they rewrite the paper; and such rewriting is an integral part of the work of recalculation that is done while abstracting (2007: 189)

However, the process does not end there. As Moreira demonstrates, the disentanglement of the published data is followed by a process of re-entanglement in order to link the technical devices of systematic reviewing (e.g. analysis of the statistical validity of a given published study) with the social and professional practices that they are anticipated to regulate. As Moreira details, this 'politico-technical' entanglement is evident in the ways in which the laboratory-derived data collected by reviewers is circulated outside of the laboratory context, for example, in the form of meetings that develop CPGs.

In CPG meetings, the results of systematic reviews undergo a further transformation as they are 'tabulated, summarised and discussed by multidisciplinary groups' (Moreira 2007: 192). As the participants of such meetings attempt to produce recommendations for practice from the data collected by systematic reviews, they may debate the validity of the meta-review process. As Moreira demonstrates, the process of translation from meta-data into CPG entails taking into account how the results of systematic reviews 'might interfere with the distributions of power and accountability within healthcare institutions'. (Moreira 2007: 192). For example, in one GPG meeting a consultant, GP and methodologist debated the methodologist's decision to exclude a study from the meta-analysis. Speaking from his experience, the consultant attempts to address the disproportionate influence of a particular study on the opinions of his peers:

I think and I have said why we think that the trial does not show specific benefits for [drug y]. In one sense it's easier to kind of put the trial to one side. The problem though is that that trial has just had one of the most successful marketing campaigns ever and most people in primary and secondary care [believe there are benefits beyond these of other drugs], and it's got in the [national tabloid] because [they] have been extremely clever with their marketing and PR. (Moreira 2007: 192)

The consultant questions whether the widespread misconception about the efficacy of the drug should be addressed in the guidelines. That is, regardless of whether the drug is more or less efficacious than others, the fact that it has such a dominant presence means that a tension emerges between the results of the meta-analysis and the perception of the drug amongst clinical professionals and the general public. For the consultant, this issue should be acknowledged in the guidelines, however the methodologist feels that such an approach would over-represent the drug, which is only relevant 'down the line in [their] recommendations' (Moreira 2007: 192). In addition, and as one of the GPs present notes, this type of approach may be met with disagreement by the drug company who sponsored the trial. As Moreira concludes, the public acceptance of the guidelines ultimately depends on how they align with a variety of actors – the systematic reviewers, clinicians, the public and the sponsors of the clinical trial (see Moreira 2005) for a detailed ethnographic account of CPG committees).

EBM is thus far more complex than the application of the best available scientific evidence to clinical decision-making processes. It can be understood, as Armstrong claims, as a strategy to re-establish the authority of the medical profession, but also as a revolution in medical practice that ensures safer and more efficacious interventions. It can also be understood as the 'heartless application' of scientific knowledge which, in turn, facilitates the commercialisation of medical care (Mykhalovskiy and Weir 2004). As Weir and Mykhalovsky quite rightly point out, any investigation into EBM must interrogate the relationship of such social and scientific orders across the localities in which they are manifest. Thinking back to the previous sections then, EBM can be defined as a form of 'regulation at a distance' (Rose 2006: 3) that typifies the neo-liberal condition. It can also be seen as an attempt to grapple with and regulate the results of the concurrent, dissolution and fluidity of the boundaries of health disease and illness, the decline in medical authority and the emergence of somatic

ethics. This complex set of relationships is one that is indicative of the management of treatment of thyroid disease within the NHS. That is, as will be described, thyroid disease is constituted by various and often contentious attempts by patients, clinicians, professional bodies and bureaucratic systems, that attempt to define and to attend to it. Therefore, thyroid disease is an ideal location in which to contribute to theoretical concepts, in particular biosociality, that have attempted to re-conceptualise the situated and fluctuating relationship between nature and culture, body and society and disease and illness in the twenty first century.

Treating the thyroid

In July 2006, the UK Guidelines for the *Use of Thyroid Function Tests* were published in order to aid primary care physicians, specialist physicians, endocrinologists, clinical biochemists and patients in understanding and establishing a standardised approach to the diagnosis and treatment of thyroid conditions. The guidelines were authored by a drafting committee including members of the British Thyroid Association (the BTA, the professional association of endocrinologists who treat thyroid disease within secondary care), the Association for Clinical Biochemistry (the ACB, the professional association of clinical biochemists who develop and conduct thyroid function tests), and the British Thyroid Foundation (the BTF, a patient organisation that provides support for sufferers of thyroid disease and is affiliated via endorsement to the BTA). The purpose of the guidelines is to:

[E]ncourage a greater understanding of thyroid function testing amongst all stakeholders with a view to the widespread adoption of harmonized good practice in the diagnosis and management of patients with thyroid disorders. (Association for Clinical Biochemistry et al. 2005)

As identified in the introduction to the guidelines and evidenced by a substantial body of published literature, all three stakeholders are revealed to be uncertain about a number of aspects of the testing of thyroid function as a result of the complex and often contradictory relationship between the results of thyroid function tests and the clinical symptoms of thyroid disease. According to the authors of the guidelines "the need for a national guideline for something as common as thyroid function testing is self

evident' (Association for Clinical Biochemistry et al. 2005: 13) in order to provide standard protocols that will resolve such confusion. Evidence-based methods are sought in order to clear up the indeterminacy of thyroid disease and produce a clear set of standardised guidelines based on the findings of a systematic meta-analysis of the best available published evidence. Subsequently, as claimed in the introduction, as the guidelines are evidence-based they meet the requirements of all three stakeholders. That is, the evidence collected in order to produce these guidelines is so methodologically robust it can capture, and do justice to, the complexities of treating thyroid disease in clinical practice across varied professional and patient contexts.

However, in a pattern that proves to be an intractable feature of this area of current medical practice, not only in the UK but elsewhere, the uncertainties of the various stakeholders mediate the current practices that surround the clinical presentation of thyroid disease and the biochemical laboratory-based test used to diagnose diseases of the thyroid. As the guidelines point out, the huge amount of information available to patients about thyroid disease from the internet, as well as various patient support groups and doctors, is considered to be confusing and at times inconsistent. Patients often find it hard to understand that the diagnosis of thyroid disease is not always clear and can vary from patient to patient. For clinicians, the non-specific nature of thyroid disease symptoms demands definitive evidence, in the form of biochemical assays, in order to make a firm diagnosis. However, the current variability of testing techniques across laboratories is seen to jeopardise the process of making a definitive diagnosis. Finally, due to the rapidly rising number of thyroid function tests being requested, the laboratories where the tests are carried out need to ensure that the clinical grounds for tests are significantly justified. Therefore, it is assumed that the problems currently relating to the diagnosis and treatment of thyroid conditions are due to a lack of consensus on current diagnostic and treatment practices between the stakeholders. However, as will become clear throughout the course of the thesis, the problematic aspects of thyroid disease extend far beyond normative discussions about how the best available evidence is applied in clinical practice. Thyroid disease is not simply the sum of the evidence used to define and treat it, nor is it reducible to what would count as evidence at all.

In chapters four and five this 'counter-empirical' relationship between the knowledge about thyroid disease and the practices of a thyroid outpatients clinic in central London is initially described in terms of what counts as 'evidence'. By introducing thyroid disease in this way the empirical focus of the thesis is identified in part as what can count as an empirical fact. As we shall see, evidence for thyroid disease is ordered by various stake holders in order to establish particular orders of thyroid biology that represent a range of needs, rights and responsibilities with regard to health and health services delivery. In turn, this disease and the debates among its many stakeholders can be seen to extend further the longstanding debate within the social sciences concerning processes of medicalisation, health as a regime of biopower, and the gendered body as a site of both agency and resistance. Before this discussion is embarked upon the following chapter will outline the methodological approaches employed by the project.

Chapter 3

Methodology: Understanding Thyroid Disease

Er no they're never really normal they're either too high or too low or, but yeah, I think it's a very hard thing to regulate and I think that it's a personal thing you've got to regulate it yourself, so what I do is I take my 25 and if sometimes I don't, I'm...I think to myself oh you know I might need to take a bit more so I might, I might take 50 for a couple of days and I do it, sort of regulate it myself.

Lorraine – Graves Disease Sufferer, Post Radioiodine Treatment

According to consultant endocrinologist Anthony Weetman in the journal *Clinical Endocrinology* entitled *Whose Thyroid Hormone Replacement is it Anyway?* (Weetman 2006), there is nothing more straightforward than the treatment of hypothyroidism. As his article goes on to claim, perceptions and practices such as those articulated by Lorraine in the above quote are the result of two factors: 'postmodern' medical practices and the lack of consensus amongst endocrinologists concerning the treatment of thyroid conditions. For Weetman, 'postmodern medicine' is typified by the 'derogation of objective facts' and the 'replacement of scientific certainty with the view that reality can have multiple meanings' (2006: 231). Consequently, it has become common for patients to develop a distrust in doctors and the belief that 'malevolent science' perpetuates, and sometimes even causes, disease and illness. As a result, Weetman claims that patients turn to 'folk models of disease' (2006: 231) and the adoption of various alternative lifestyle choices, that can be accessed through the wealth of health information available on the Internet. For instance, Weetman cites Marcia Agnell's (1996) discussion of the supposed health risks of silicone breast implants with regards to connective tissue disease. In particular, she draws attention to how women who claimed the connection between breast implants and connective tissue disease attempted to prove their case by presenting themselves, and not scientific research, 'as the evidence'. As Lorraine can confirm, subjective beliefs about the nature of disease are highly visible in the contemporary approaches to health services delivery (Armstrong 1984, Sullivan 2003), and they shape the biomedical practices that are traditionally assumed to be the province of medical experts. Lorraine's model of self-administration

of thyroxine is a personal response to, and manipulation of, an evidence-based treatment that blurs the boundaries of her biochemically⁸ measured and defined disease. A pressing issue therefore for an investigation into thyroid disease, is how to track and assess the dialectical relationship between the imagined separate domains of the 'science of' and the 'cultural responses to' thyroid disease.

This problem of the relationship between the science and culture, or the 'actual' and 'imagined', is also one which dominates anthropological theory and methodology. In particular the traditional assumption that writing up field work is, as James Clifford and George E. Marcus claim '...reduced to method: keeping good field notes, making accurate maps, writing up results' (1986: 2). That is, the assumption that writing and making anthropological texts on a specific culture are neutral acts that do not need to be scrutinised in terms of the politics of the representations they produce, is no longer tenable. Therefore, as they declare in the introduction to their seminal edited volume *Writing Cultures: The Poetics and Politics of Ethnography* (1986), such an ideology has crumbled and as a result the essays collected in the volume they see culture 'as composed of seriously contested codes and representations [that] assume that the poetic and the political are inseparable, that science is in, and not above, historical and linguistic processes' (Clifford et al 1986: 2). Consequently the ethnographic techniques used to represent the phenomena of thyroid disease will not be defined as a neutral 'writing up' of the clinic. Following Clifford's definition of culture, the representation of thyroid disease in this study is conceived as a practice that is inside of, and not above, broader cultural practices, conventions, trends, traditions and knowledge systems.

Initially this project was entitled 'Ethnography of ambiguity: thyroid conditions, the promise of genetics and the lived experience of chronic illness'. As this title suggests, the study intended to focus on the implications of the well-established familial and vague predisposition to

⁸ Thyroid disease occurs when the gland malfunctions and fails to produce too little or too much thyroid hormone. The level of thyroid hormone is measured via a blood test and this biochemical measurement translates into the level of health of the thyroid. Treatment of a sick thyroid takes the form of either topping up or suppressing the production of the gland, Lorraine is altering her dose according to how she feels, rather than in accordance with the biochemical measures of her disease.

thyroid disease with regards to how patients managed and understood their condition, in particular at the point of reproductive choice and genetic counselling. At the time when this study was first being planned my personal experience of having a thyroid condition was influenced by my interest in the emerging field of qualitative social studies of biomedicine, many of which focused on 'the new genetics' (Kevles and Hood 1992, Weir et al. 1994, Hubbard and Wald 1997). Having been advised that this was an area that could potentially garner financial support from a research council, I wrote my proposal with this focus in mind – specifically with regards to how vague genetic information might impact on the diagnostic categories of thyroid disease. However, it soon became clear that this focus was problematic in terms of the prevailing research culture at the time. As a referee commented in a failed application for an ESRC/MRC PhD studentship:

good idea to study genetic susceptibility to a common chronic illness. However, there is a danger that studying patient experiences of common susceptibilities will become an industry in the way monologic disease has been.

As the referee suggests at the time my application was submitted, social scientific studies of genetic conditions were commonplace and had become 'an industry'. This industry therefore added to what the anthropologist Paul Rabinow termed the 'hyperbolic discursive tidal wave of hope' (Rabinow 1999: 11), where the idea that genetic code would provide a 'blueprint of life' was not only increasingly unrealistic, (Lock 2005) but led to fears of genetic reductionism and determinism (Lippman 1992). The decision not to make 'an industry' out of the vaguely genetic, specifically the ways in which this idiom relates to other forms of disease, information and practices, was a cautious response to Rabinow's 'hyperbolic discursive tidal wave of hope'. Although this proposed research was attempting to think about genetic information in context, specifically as a partial and highly indeterminate entity, it was still considered by the referees to contribute to the 'industry' of social studies of genetic disease information. This example demonstrates that knowledge about our genes is not simply a matter of uncovering biological facts, it is also intrinsically linked to the politics of how and why such research is conducted and how it shapes healthcare and identity practices. However, in spite of failing to secure funding, I persevered with this research trajectory on the vaguely genetic, which

proved to be a fruitful starting point for this study. Specifically, it enabled me to recognise that many kinds of disease information and evidence are partial, indeterminate and worked out through practices during the course of care.

The ways in which knowledge about disease is transformed and shaped by practice became even more apparent when I entered the thyroid clinic during the period of data collection. As will be described in the following chapter, within the day-to-day business of the NHS thyroid clinic the well-established link between genetic inheritance and the onset of thyroid disease (Chistiakov 2005) is barely discussed, and plays almost no role whatsoever in diagnostic and management strategies. However far from being disappointing, this process of realisation that occurred during the early stages of the research actually helped reveal an alternative focus for the study. The contradiction between the 'science' of thyroid disease, exemplified by this partial genetic susceptibility, and the distorted application of this science within clinical practice, was just one of many instances where 'knowing' thyroid disease clashed with, ignored or was irrelevant to the practice of diagnosing, treating and suffering from thyroid disease. It soon became clear on entering the clinic that not only were other issues more relevant to an investigation into thyroid disease than its 'vaguely genetic' character, but also that such a study had to be approached in such a way that it created a linkage map between the traditionally separate spheres of objective scientific fact and subjective ethical value (Konrad 2005). To that end, after the first few instances of participant observation, the research questions that were designed to explore the specific issue of genetic predisposition to thyroid disease were abandoned. What ensued was a process in which the practices of the clinic, performed by both the healthcare professionals and patients, were observed, became familiar and were latterly mapped onto (often discordantly) the ways in which thyroid disease is presented in clinical scientific literature (the content of which will be discussed in the following chapters). In the following sections the process of gaining access to the field, data collection and data analysis is described.

Gaining access to the field

In December 2004 I approached a Senior Consultant Endocrinologist employed by a central-London hospital with a substantial research reputation in the field of thyroid disease. He is also a member of the development group for the clinical practice guidelines on the *Use of thyroid function tests* (Association for Clinical Biochemistry et al. 2005) and a senior member of the British Thyroid Association (BTA). Following his response, a meeting was arranged in January 2005, to which he was an hour late. This gave me some idea of the time pressures that I would face conducting research in such a busy environment. During our short meeting the Consultant was very helpful and agreed to grant me access to his clinic to do what he termed 'some type of psychological study' of patients with thyroid conditions. This short introduction to the gatekeeper of the field site was followed by a long and complicated process of gaining ethical approval to conduct research within the NHS through the Central Office of Research Ethics (COREC). No observation within the clinic could occur without this approval and it was eventually granted in January 2006, four months after the initial application was submitted in September 2005

Renamed the National Research Ethics Service (NRES) in 2007 COREC, was a government office that was oversee all of the Research Ethics Committees (REC's) within the United Kingdom. Each Strategic Health Authority (SHA⁹ in the United Kingdom has a REC and under guidelines from the National Patient Safety Agency and consist of a number of members who;

are specially trained in research ethics and often have the sort of experience which will be useful in scrutinising the ethical aspects of a research proposal. These include: patients; members of the public; nurses; GPs; hospital doctors; statisticians; pharmacists and academics, as well as people with specific ethical expertise gained through a legal, philosophical or theological background. (National Patient Safety Agency 2007a)

The first REC was established in 1966 and such committees have been present within individual hospitals (in the form of Local Research Ethics

⁹ Strategic Health Authorities manage the NHS locally and are a key link between the Department of Health and the NHS

Committees LRECs) and health authorities since that time. Their implementation and ethos is a direct response the World Medical Association (WMA) declaration made in 1964 entitled 'Ethical Principles for Medical Research Involving Human Subjects' (1964). Briefly, this declaration states that all experimental procedures involving human subjects should be formulated to a clear experimental protocol that has been passed via an ethical review process. This ethical review process must conform to the laws and regulations of the country in which the experiment is taking place and, the ethical review committee must be independent from the investigator, sponsor or any other undue influence. All adverse events must be reported to the committee as must all detail regarding institutional affiliation, project funding and any incentives for test subjects. Within the United Kingdom the primary function of REC's is to 'protect the rights, safety, dignity and well-being of all actual or potential participants' (National Patient Safety Agency 2007a).

More recently with the development of COREC in 2004 and latterly NRES in 2007, REC's have been transformed substantially. Until COREC introduced an online application form in 2004, all paperwork followed the varied formats set by local REC's. This standardisation of the application procedure is part of a move to make ethical approval procedures robust and accountable. As Dr Frank Wells, the co-chair of the European Forum of Good Clinical Practice, said;

This means that US bodies, such as the Food and Drug Administration, will review UK research in a favorable light as it will be assured that its ethical review will have been conducted to the highest standard (National Patient Safety Agency 2007b)

As I soon found out, by completing this process myself and talking to others who had also filled out the COREC form, these measures to make the ethical approval process better seemed to be universally despised within the field of clinical research! The process implemented by COREC was deemed time consuming, expensive and as much about resource allocation as than about ethics. As David S. Wald notes in his editorial in the British Medical Journal, part D of the 68 page form was essentially a record of indemnity to be signed off by the Head of Finance, which in his case 'required two drafts, nine phone calls, four faxes, 20 minutes of online internet activity, and 7.5 hours of staff time. There were no questions of ethics' (2004: 283). Not only was the COREC form seen as unnecessary

and obstructive, it was regarded as an affront to the ethical 'know how' that is embedded in the practice of clinical research and practice. The sentiment seemed to be that the independent ethical review of any research that involved human subjects was not an issue - the problem was the complexity of the process that had been developed to accomplish it. The medical value of the research, the assurance of minimal risk to participants and the provision of adequate informed consent were seen as the most important, obvious and already accomplished aspects of medical research culture. Such measures were considered to be embedded in a system of unspoken ethical beliefs and procedures that were intrinsic to good medical practice and the identity of the profession.

The belief that the COREC procedure was a means of making medical practice accountable enabled me to identify yet another area of empirical and theoretical interest. The tension between making medical practice and research better through standardization and accountability and the suspicion that it was a ruse to rationalize resources was also a tension commonly observed through the diagnostic and treatment practices used in the thyroid clinic and throughout the NHS more generally. Not only did the COREC process mean that I was granted access to the clinic, it meant that I was to some extent able to experience the pressures that clinicians face in their everyday working lives. Consequently, I developed a better understanding of the landscape of clinical practice and was able to interact with the Consultant who ran the thyroid clinic as if I were a colleague with whom I could share my frustration of the ethical approval process with. Subsequently, my fear that I would be unable to effectively decipher the specialist language and practices of the Consultant were slightly lessened by the COREC process. That is, it enabled me to learn the language of medical authority and provided me with the means with which to establish a good working relationship with the Consultant.

However, as I wasn't actually treating patients this ability to empathise with the Consultant was only ever partial. When I clicked on the qualitative research and single site¹⁰ options on the online COREC application form it was automatically reduced from 65 to 20 pages (plus supporting

¹⁰ Ethical approval has to be sought from the SHA REC and the LREC of each site where the research will be carried out. Luckily in the case of this research project only the SHA REC and the LREC of the hospital where the clinic is situated had to be applied to.

documentation). This was due to the fact that I would not be directly intervening with diagnosis and treatment, administering new drugs or using any additional hospital resources during the course of the project. In spite of feeling that I was conducting some form of 'health services research', the form still seemed to jar somewhat with the ethnographic approach of the research project, and it soon became a case of making the research fit the form, rather than providing an accurate description of how I intended to approach the project. Having to fill 20 pages with a thorough description 'of what I will do' is wholly antithetical to the purposefully 'see how you go' approach traditionally employed by ethnographic methods. As Sarah Franklin and Celia Roberts note in the methods chapter of their ethnography of PGD (Preimplantation Genetic Diagnosis), the haphazard open ended and exploratory nature of participant observation and ethnographic work in general can appear meandering, random, amateur and 'dilettante-ish' (Franklin and Roberts 2006: 81). This was especially the case in the context of a form and set of procedures that demand accuracy and accountability. For instance, the change in the initial focus of my project/research from genetic predispositions to the accuracy of thyroid function blood tests, has rather straightforwardly been absorbed into the project as a necessary alteration. That is, the data- rather than hypothesis-led approach of ethnographic methods means that changes, such as the one described, are an intrinsic and to some extent, a conventional part of the research process. In addition, methods such as participant observation, where the researcher is expected to immerse themselves in the field, making them a 'human instrument' (Powdermaker 1966) of the data collection process is an anathema in the context of clinical research. That is, in the context of a field governed by evidence-based methodology, using a researcher's subjectivity as the medium through which data is collected is considered to invalidate the integrity of the research.

In spite of this disparity between the formality of the COREC form and the relative informality of the chosen research methods in this context, the project was granted ethical approval from both the SHA REC and the hospital's LREC with relative ease. In fact, during application review with the ethical review committee, the only comment offered was regarding the measures I had taken to ensure my own safety when interviewing patients in their own homes, when one of the panel asked, what would I do if a patient came at me with a butter knife? I responded that there was very

little I could do to prevent this occurrence, but that as 'an anthropologist' I had received training in personal safety within the field. Interestingly this is another example of where the formality of the COREC process enabled me to reduce my anxiety about conducting research within a clinical environment. As demonstrated above it was necessary during the COREC process to formally identify my authority and skill as a qualitative researcher, which in turn justified my presence within the clinic as a 'specialist' who worked within a particular discipline.

Overall, apart from this concern for my personal safety and a request to change the wording on the posters I produced to advertise the study in the clinic, the agreement was given relatively quickly after I submitted the paperwork. Although the COREC ethical approval procedure was on the whole inapplicable to qualitative research, the key ethical criteria set out by the British Sociological Association (2002) were met by the process. Briefly, this includes the promise of anonymity for all participants; the availability of all transcripts of interviews for approval and the destruction of all audiotapes of the interviews after the study has been completed.

Navigating the field and data collection

The data collection process for this study consisted of observing a Monday afternoon outpatients thyroid clinic over a period of 12 months. I interviewed patients and the Consultant who ran the clinic, and conducted a review of all relevant literature relating to thyroid disease (peer-reviewed published articles and patient support group literature) and health services delivery within the NHS. I was also provided with copies of all letters that were sent to the patients who attended the clinic after their consultation. These letters were written by the Consultant and summarised their appointment including: a clinical diagnosis, a summary of treatment prescribed, and an outline of any necessary future treatments. Along with the field notes produced during the participant observation, the interview transcripts and the patient and professional literature described above, these letters formed the project data archive. In the following section these methods and types of data will be described and reflected upon in terms of the specificities of the fieldwork site.

Participant observation is the primary technique of ethnographic research. However, how one goes about this process is much less clear, as access to and participation in the field are so context dependant. In theory the practice of a good participant observation lies in the ability of the researcher to become what E.E. Evans Pritchard calls a 'doubly marginal' person (1937), referring to the ability of the researcher to sit between their own culture and the one which they are studying. However this process is something that is almost impossible to comprehend in the abstract, until a researcher has been in and gone through the process of becoming immersed in his or her chosen field. What became clear about this process is that access, and as a consequence ethnography, essentially relies on the process of establishing relationships with subjects in the field. That is, relationality itself, the exploration of its character in a particular context, is a guiding and potentially fruitful aspect that helps to structure the approach to and decipher the ethnographic location.

For example, during the process of negotiating access to and observing the clinic, I had very little contact with anyone other than the Consultant. On his instruction his secretary forwarded me electronic copies of the clinic letters after they had been typed, however, he primarily administered my research access and the day-to-day running of the clinic in general. This included such tasks as re-scheduling patient appointments and chasing up blood test results with colleagues in the hospital laboratory, with whom he had developed a good rapport. Although the Consultant seemed frustrated by the amount of administrative work he had to perform in order to ensure the smooth running of the department/practice, it became clear that this was a particular characteristic of running a thyroid clinic. The lack of support he received from other members of staff, both clinical and administrative, drew my attention to the status of thyroid disease in the discipline of endocrinology more generally. It became clear that although thyroid disease was a speciality of the Consultant, it was also an aside to the bulk of his everyday work where he attended to more serious and clinically interesting diseases such as, diabetes, Addison's disease, Cushing's disease and hyperparathyroidism. Therefore, the nature of my relationship to the Consultant, his relationship with his colleagues and thyroid disease more generally, provided an invaluable insight into the field. To be precise, it enabled me to conceptualise how the specific aspects of thyroid disease, in particular its status as a common chronic disease that is apparently

'simple' and 'straightforward' to treat, fitted into the politics of healthcare provision within the NHS and the structuring and imperatives of clinical research.

In the case of this project, as described above, the process of gaining access to the field, although long winded was relatively straightforward. However as mentioned previously, on entering the clinic the original focus of the project seemed incompatible with what was being observed. What I wanted to see was not happening and the clinical and professional literature I had read during the previous preparatory year did not seem to translate into the clinic in the way I had expected. For instance, there was often no mention of the aetiology of the disease, not all the patients had all the 'classic' symptoms, not all of them received the same treatment and not all of them felt well or even unwell at the times they were 'supposed to' during the course of the disease. It soon became apparent that I had to stop trying to write my field notes and think through what was occurring in the clinic via my preparatory reading and preconceived theoretical notions. By the third visit to the clinic I stopped writing in my notebook; I just sat there and watched in order to re-learn thyroid disease within clinical practice. After a couple of weeks patterns began to emerge, this included the complexities between how the disease is supposed to behave and how it does behave from patient to patient. I began to record commonalities, such as how the thyroid was examined and how the evidence that led to a diagnosis was ordered. Soon these similarities and disparities were transformed from a problem to the critical focus of the project. The novelty and anxiety of being a non-medical professional also wore off. I soon realized that a consultation room within a large teaching hospital was always punctuated by the comings and goings of various people: nurses, and medical students who were also observing the thyroid clinic. As one of many who sat at the back of the consultation room, I soon became comfortable and fluent in milieu of the clinic – the status of 'doubly marginal' to some extent had been reached.

Due to this new-found understanding into the life of the clinic, the anxiety of how to approach patients also subsided. Although in the COREC application I had stipulated that I would approach patients via a patient information sheet (see appendix 1), when entering the clinic, the logistics of presenting them with this document became an issue. The speed of the

clinic was not conducive to handing patient information sheets before, after or during each individual consultation, and besides, during the first couple of weeks, my assumptions about how much I knew about the disease had been problematised, making me nervous about talking to patients about their condition. However, the confidence that had been gained from the few short weeks of re-learning, plus the Consultant's increasing wonderment about what I was actually doing there (just sitting and watching?) soon dismissed this anxiety. He suggested that I was sent a copy all of the clinical letters sent to the patients after their consultation. That way I could select those who I wanted to interview in my own time. Moreover, as mentioned briefly above, this collection of clinic letters has also proven to be an invaluable archive in and of itself. I now have approximately 250 clinic letters that refer to the first and follow up appointments of approximately 150 patients. These letters not only provided me with the contact details of individuals and a means of approaching them for interviews, but also, became a resource where another representation of thyroid disease could be observed. Often these letters took the form of a patient history that detailed how the patients had come to be referred to the clinic by their GP, the symptoms that they had reported, the signs that the Consultant had observed and consequent diagnosis and treatment strategies. These reconstructions of the thyroid disease through the clinic letter also enabled me to observe how diagnostic endpoints were reached and how the evidence used to diagnose thyroid disease was handled and evaluated by clinicians and related to the accounts provided by patients. On a more practical level the letters helped me prepare for interviews with patients recruited from the clinic as they detailed their history of thyroid disease.

The shift in focus of the project from the vaguely genetic aetiology of the disease, to the tension between the pre-clinical disease model and the clinical process and practices of treating it, became an organising principle with regards to the selection of participants for interview. Often in consultations where the patients seemed dissatisfied with the diagnosis and advice the Consultant offered he would often refer them to me in order to discuss this matter further. Specifically, he acknowledged that the dissatisfaction of such patients was an issue, but one that he could do nothing about as it was something that fell outside of the scope of his expertise i.e. these problems could not be accounted for through current

evidence based diagnostic techniques and clinical practice guidelines. However, it also has to be noted that the identification of such dissatisfied patients was often highly gendered and the identification of such biomedically unsanctioned dissatisfaction was most explicitly performed in consultations with women. 'I can't help you' and 'why don't you talk to Megan who is doing a study on this sort of thing' underlined this gendering as it suggested that there was an alternative a therapeutic strategy where women could talk about their feelings, personal interpretations of symptoms and illness experience. That is, this strategy was an attempt to manage a subjective and emotional category of illness that was considered to be secondary and residual to the 'actual' manifestation of thyroid disease. In addition to the selection of the sample through direction from the Consultant, many patients also self-selected due to their dissatisfaction with the treatment they had received. Often it was an opportunity for them to communicate the tangibility of the symptoms that they attributed to thyroid disease, a connection that remained unexplained during clinical their consultations. The politics of the selection process of participants for interview again informed and helped to build up a picture of the range of professional and patient experiences that constitute the characteristics of thyroid disease within the contemporary NHS. These factors in addition to the higher prevalence of thyroid disease in women and the low response rate from men resulted in 19 interviews being conducted with women between the ages of 24 and 62 and one was conducted with a 45 year old man.

Overall the format of the interviews was fairly consistent. After I sent the letter and information sheet to patients they contacted me to arrange a meeting at their home or in a café, which was usually the preferred option because other family members at home could, as one interviewee put it, 'get in the way'. The only exception to this was when I interviewed one participant in the meeting room at his office because he tended to work long hours and it was the only time he was able to see me. All interviews were recorded on a digital recorder and then transcribed, these transcripts were then sent to the interviewees for approval. None of them wanted any passages removed for the transcripts and none of the sample withdrew from the study after seeing the transcripts of their interviews. Again because of the initial focus of the research, the patient information sheets sent to prospective participants described the study as one that was

investigating 'how genetic predispositions to autoimmune thyroid conditions influence their treatment and management.' However, as had been the case in the clinic, this focus was soon superseded by other issues the patients felt were central to their experience of the disease. The style of in-depth unstructured interviews that was used with the sample, meant that more relevant focal points came to the fore. I started the interview by asking patients to tell me their story of thyroid disease - when it had first been diagnosed, their symptoms and treatments, how they understood where it had come from etc. Perhaps unsurprisingly, those who had taken the time to be interviewed had much to say with regards to their experience of the disease and often had strong opinions about the causes and politics linked to it.

As mentioned previously my understanding of the clinical literature changed consistently throughout the course of the data collection process. For example, at first I was sure that my focus on the vaguely genetic was 'correct' as there was an established research community looking into genetic triggers for thyroid disease. However, the literature review became a constant process which evolved in response to the course of the fieldwork. Moreover, the on-going process of literature review became one that consisted of disregarding particular strands of published research as well as including relevant sources. Therefore a data archive that consisted of particular published texts, field-notes, patient support group websites etc., was collated in response to the twists and turns of being in the field. For example, three books were recommended by a patient support group website, the Consultant, and a couple of patients, - *Thyroid disease: the facts* by R.I.S Bayliss and W.M.G. Tunbridge (1998), *Understanding thyroid disorders* by Dr Anthony Toft (1995) and *Fast facts: Thyroid disorders* by Gilbert H. Daniels and Colin M. Dayan (2006). These books became my handbooks for understanding thyroid disease as they were for those who suggested I looked at them.

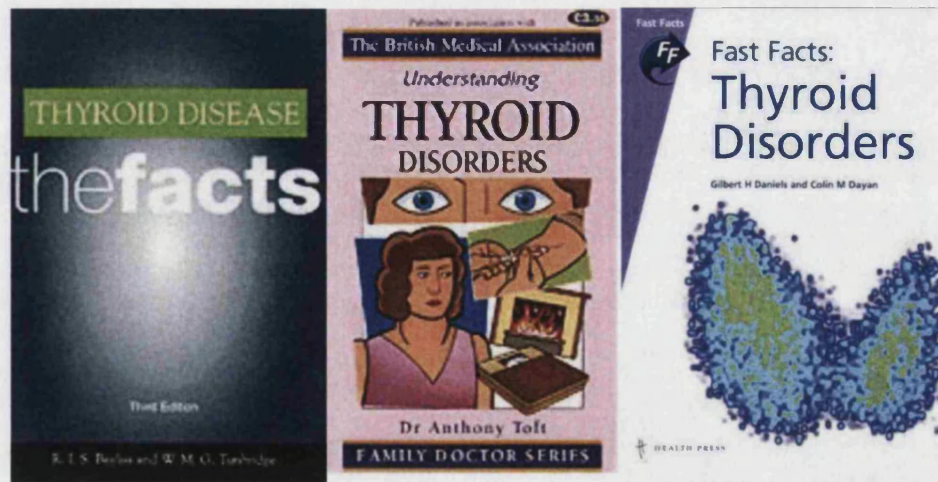


Figure 2: Cover of *Thyroid disease: the facts*
 Figure 3: Cover of *Understanding thyroid disorders*
 Figure 4: Cover of *Fast facts: Thyroid disorders*

Other patients who were dissatisfied with their treatment and disagreed with the current guidelines of treating thyroid disease also suggested an array of material that supported their point of view. In particular a number of such patients suggested I looked at Dr Barry Durrant-Peatfield's book, *Your Thyroid and How to Keep it Healthy* (2006), telling me that it contained as one informant put it 'everything I needed to know about thyroid disease'. Consequently, the amalgamation of the data archive was not only useful because it provided a quantity of data that could be analysed. It was also useful because its meandering and sometimes confusing course contributed to my grasp of the ambiguity of thyroid disease, a characteristic that has become a key theme of this study, and that has been used to contribute to broader debates about the boundaries of health disease and illness in western healthcare contexts.

Handling and structuring data

With reference to the theoretical issue raised at the beginning of this chapter, the analysis and write up of the data collected in the field has not been approached in a 'matter of fact' style. Within anthropology the assumption that a researcher can travel to a distant part of the world, find 'a culture' and after three years of field work come back with a monograph that neatly captures it, now seems impossible and ridiculous. Not only has the distance between us (moderns) and them (primitives) shrunk due to modernization (Eriksen 2001), but the politics of such a dichotomy has, rightfully, been called into question. The geographical and epistemological

boundaries of culture have been blurred, and the produced nature of ethnographic accounts has been brought to the fore. Furthermore, as discussed, the assertion that ethnography can claim to be a science is problematic due to the subjective nature of the data collection process. The implications of the problem of the definition of culture, however, have opened up new sites in which ethnographic research can take place. The recognition that the distinction between the self and other is problematic and that cultures are not whole and subsequently amorphous and complex, means that the questions that one can ask about a culture and society are relevant both at home and abroad. This is particularly true for social studies of science where the traditional 'culture of no culture' can be intimately explored via qualitative methods. Therefore, whilst there has been recognition that ethnographic writing is problematic, this has meant that more theoretically informed and rigorous approaches have been developed (Marcus 1995, Strathern and Association of Social Anthropologists of the Commonwealth. Conference 1995, Marcus and Fischer 1999).

For instance, in the case of this study it could be seen as problematic that I am both a researcher and sufferer of a thyroid condition. My position as both researcher and to some extent subject could be seen to jeopardise my ability to be 'doubly marginal', as I may be too much of a 'native' of thyroid disease to be able to conduct a proper data collection process and analysis. However, this problematic concept of the researcher who has overstepped the boundary between self and other is, to some extent, an obsolete epistemological issue. Underlying this problem is the assumption that the researcher can through the correct methodological approach be an objective recorder of ethnographic data. However, as has been discussed this assumption is highly problematic because of the recognition of the politics of objectivity with regards to the inherent production embedded in the act of writing an ethnographic account. Therefore, rather than dismissing ethnographic accounts as pure works of fiction we should, as readers and writers of ethnographic accounts, as Geertz suggests 'learn to read with a more percipient eye' (1988: 24). In consequence, when approaching the field I always kept in mind the factors that would shape the account of thyroid conditions I would produce. The complexity of the data I collected aided me in being mindful of the forces that would generate the account. For instance, having hypothyroidism myself when interviewing patients about their symptoms, I often found it relatively easy to understand

and even empathise with them when talking about the slow onset of thyroid disease and the often long list of vague and dissipated symptoms. However, this was not the case all of the time, sometimes other sufferers experiences clashed with or were unrecognisable to mine. Therefore, the recognition that I had to be mindful of the account I produced as both a sufferer and ethnographer producing work after the 'post modern turn', in combination with the complexity and 'situatedness' of the data collected, helped me overcome this issue.

The inability to produce a clean and straightforward ethnographic account of an isolated culture is embedded in the substantive focus of this thesis, specifically, the ways in which the meaning and value of evidence is grappled with by thyroid patients and clinicians. Form and content, therefore, have fundamentally influenced each other with regards to the data collected and the representation of the data in the analysis. For instance, the disparity between the representation of thyroid disease in the clinical literature and the performance of thyroid disease in the clinic has been transformed from a narrative that doesn't quite fit together, into an account that can evaluate this tension as part of the epistemological tradition of biomedicine, where rational science and irrational culture are separated and cast as opposing forces. Subsequently, the first step in the data analysis was a comparison of the formal accounts of thyroid disease (clinical guidelines, journal articles and text books) and the practices and alternative accounts of disease that were demonstrated in the data collected from participant observation in the clinic, interviews with patients and the consultant who ran the clinic. This process of comparing these data sources not only provided critical focus for the research, but also allowed for a complex understanding of thyroid disease to emerge.

After the period of data collection had finished, after the field notes made during clinical observation and interview transcripts were re-read thoroughly, I identified a number of key and recurring themes . Due to the relatively small sample size, the data was coded manually and all themes were colour coded across the transcripts and field notes so they could be easily identified for analysis. Once this had been done, the literature on thyroid disease that had been read during the preparatory stages of the research project was re-analysed in light of the data that had been collected through the period of fieldwork. Additional literature that was

referred to by the Consultant and patients during clinical observation and interviews was also investigated/consulted. The Consultant often bought up for example 'the Colorado disease prevalence study' (Canaris et al. 2000), which is explored in detail in chapter five, and as mentioned previously the book *Your Thyroid and How to Keep it Healthy* (Durrant-Peatfield 2006) (also see chapter five) was recommended to me by a patient during an interview. By revisiting the formal clinical literature in light of the data collected from the clinic a more thorough understanding of the field was achieved. This deeper understanding of the field of thyroid literature means that the choices I made with regards to what literature would be included in the analysis were empirically informed. However, this is not to say that literature that did not seem relevant to the practices of the clinic was simply cast to one side. In fact the recognition that was made during the analytical process about the extent to which formal knowledge about thyroid disease clashed with the everyday practices of the thyroid clinic has become a central finding of the thesis.

Finding a focus

Throughout the process of conducting a preliminary literature review, negotiating access to the field, data collection and analysis, a common theme emerged. There is a gap between what is known and what is done with regard to the diagnosis, management and treatment of thyroid disease. For example, if we return to Lorraine's method of taking thyroxin, this form of 'self regulation' is contrary to what her doctor prescribed and what clinical practice guidelines suggest. Between planning the research, submitting an application for ethical approval, and conducting participant observation the focus of the research changed. After the practices of the clinic, the Consultant and his patients were observed, the initial focus of the research (the impact of genetic pre-dispositions to thyroid disease on management of the disease) was absent and therefore seemed inappropriate as a focus of study. Moreover, when applying for ethical approval it became clear that I would have present my intended research methods in such a way that they fulfilled the criteria of contemporary styles of clinical ethics, even though this jarred somewhat with my disciplinary training and the actual approach that was eventually used.

Whilst this gap between 'knowing' and 'doing' thyroid disease was initially disconcerting, it eventually helped to identify and structure the critical focus of the thesis. That is, the disconnection between knowing and doing thyroid disease in particular, the evidence-based approach to diagnosing it and patient experiences of it, came sharply into view. How could these different accounts of thyroid disease co-exist in the clinic? What was the impact of these obvious disconnections on the actual conditions of thyroid health? In particular, how does the qualitative embodied evidence provided by patients and the quantitative epidemiological evidence provided by thyroid function blood tests relate? What is good evidence and why? How is this disconnection overcome if at all? Returning to sentiments presented by Weetman, who and how is thyroid disease defined when the experiences of patients and the expertise of the medical profession often confound each other to such a degree?

In the following chapter an entrance into the phenomena of a sick thyroid is provided. An account of thyroid disease will be presented that is a bricolage of data sources (participant observation, interviews, clinical and patient support group literature and NHS policy documentation), and subsequently the key issues that will provide the basis for the following substantive chapters will be identified. What will become clear from this analysis is my struggle to come to terms with the data – specifically the often competing and contradictory accounts of thyroid biology and pathology it represented – has actually become the focus around which my analysis is structured. That is, the ambiguity and frustration that typify much of the data collection process and the data collected are embedded in the theoretical arguments that are made by this thesis. Consequently, 'the culture' of thyroid disease is revealed to be constituted by Clifford and Marcus' 'contested codes and representations' that are concurrently, poetic, political, scientific, historical and linguistic processes. Specifically, it is the relationships between these disparate knowledges and practices of thyroid disease that seem to constitute it within the context of the contemporary NHS in the United Kingdom.

Chapter 4

Ailing Butterflies: Diagnosing and Normalising Abnormal Thyroid Function

In this chapter I provide an account of thyroid conditions based on a collation and analysis of a range of thyroid literature including clinical texts (journal articles, books, conference/consultation transcripts, clinical practice guidelines etc.), patient support group materials (websites, newsletters, information sheets), participant observation in a NHS thyroid clinic and interviews with patients who attended the clinic. From an analysis of this 'thyroid archive' a common theme emerges with regard to the tension between pre-clinical knowing thyroid disease and the clinical practices of treating it. As will be argued throughout the thesis the formal models of thyroid physiology and pathology are constantly negotiated, with varying degrees of success, by clinicians and patients during the course of the diagnosis and treatment. In particular the evidence used to diagnose and treat thyroid disease (symptoms and blood tests) become entities through which patients and clinicians justify particular, and sometimes oppositional, disease management and treatment strategies. This chapter will describe how thyroid disease comes to be known and is performed through formal clinical knowledge, clinical practices and patient experiences. Towards the end of the chapter the relationships and tensions between these realms are highlighted, thus introducing the intellectual focus of the thesis – how the 'natural categories' of thyroid disease are constituted, maintained, but also contested by both clinicians and patients in order to fulfil a range of particular needs, rights, responsibilities and desires.

As described in the literature review, within medical sociology and anthropology disease is understood to be situated and performed within broader social relationships via the production of specialist and professional knowledge and practices (Good 1994, Lock and Gordon 1988, Illich 1975, Freidson 1988, Armstrong 1983). Countless qualitative studies of health and disease (Kaufman 2006, Berg 1998, Crossley Michele 1998, Franklin 1997, Lock 1988, Martin 1987, Martin 1994, Mol 2002, Rapp 1999b, Gibbon 2006, Lambert and Rose 1996a) and social studies of biomedicine

(Rabinow 1996, Oudshoorn 1990, Canguilhem 1978, Foucault 1976, Haraway, Rose 2004, Roberts 2002) have interpreted bodies, pathologies and subsequent interventions as political constellations, cultural artefacts and social relationships, as well as biological entities. As the ethnographer and philosopher, 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, Mol's method of analysing a disease as an assemblage of objects and practices across a number of contexts is one that has been able to do justice to the data collected by this study of thyroid conditions. As will become clear throughout the thesis there are a number of different, contradictory and contentious versions or orders of thyroid biology and pathology favoured by various patient and professional groups, all of which appear across a number of textual sources (clinical practice guidelines, patient group websites etc.) and practices (prescription of medications, interpretations of evidence, dosage compliance etc.). Mol's approach (which will be discussed in more detail in chapter six) is able to track these various objects and practices that assemble and crosscut pathologies of the thyroid gland and in doing so account for both the biological and social dimensions of disease and illness.

This chapter provides a descriptive introduction to the field of thyroid disease and then moves on to demonstrate, through the specific example of the definition of a goitre¹¹, the significant tensions between how symptoms are categorised in formal clinical literature and how they are manifest within the clinic. In addition to providing a description of thyroid disease this chapter will also address a critical issue that will provide the basis for discussion in the remaining chapters of the thesis, namely the relationship between established biochemical measures of thyroid dysfunction and its 'classic' clinical symptoms. The following section will introduce the ethnographic location of the research - the NHS out-patients thyroid clinic, interviews with patients who attended the clinic and clinical and patient support group literature.

¹¹ A goitre is the enlargement of the thyroid gland that can be both a symptom of thyroid disease, but also a harmless and common abnormality.

Entering the clinic

The primary informant of the study was a Consultant Endocrinologist who, single-handedly, ran the Thyroid Clinic. He also ran a Thyroid Cancer Clinic that was not included as a site of the study. Their General Practitioner referred all patients to the Consultant and all were expected to have had a Thyroid Function Blood Test (TFT) before the clinic, so the Consultant could assess how the gland was functioning.

The hospital where the clinic is held is situated in central London. It has 900 beds, employs approximately 4,900 people, has a major accident and emergency service, and houses all branches of surgery and medicine. An associated medical school conducts medical research, much of which is, the hospital website claims, 'of an international standard'. This huge, austere hospital is situated at the bottom of a hill, which may account for the fog that always seems to collect around it. It is also always damp and cold and has at least two sides covered in scaffolding at any one time. Inside it is typically an NHS hospital built in the late 1970s - clean, but slightly faded.

The Thyroid Clinic takes place on the last three Monday afternoons each month (the first Monday of the month is reserved for the Thyroid Cancer Clinic). Between the hours of 1.45 and 6pm the Consultant sees an average of 16 to 25 patients. Each appointment, although scheduled for ten minutes, can last up to thirty and occasionally longer. The Thyroid Clinic is situated on the first floor of the hospital and is part of a larger outpatient section that covers the lower ground, ground and first floors. For patients, access to the Monday afternoon Thyroid Clinic is relatively easy, as this busy outpatients area is well sign-posted. The outpatient facilities are shared with other clinics, each of which exists within a specific time slot and does not begin until the designated receptionist and consultant have arrived. Consequently, each receptionist is responsible for, and arrives with, the particular notes of those patients who are scheduled to attend the clinic that day. When the Thyroid Clinic is prepared, all patients, both new and returning, report to reception. After reporting, patients then wait in seats that line the long corridor close to the consulting room. The location in which the Thyroid Clinic takes place is more temporal than physical, as the space is part of the 'ENT Paediatric Clinic'. All consultation rooms have

ample provisions for children, with a large play area, a basket of toys and a child height-chart on the wall. This location bears no relationship to the adult patients who attend the Thyroid Clinic. Moreover, the temporary or transitional location of the Clinic itself is notable as it can only function when the receptionist and Consultant are present. Any delays to their arrival will disrupt the afternoon schedule – a common occurrence due to the seemingly impossible schedule of the Consultant.

The Clinic serves patients at various stages of diagnosis, treatment and management for their thyroid condition:

- Patients with disease of the thyroid, as well as 'abnormal' but non-toxic (healthy, normal functioning) swellings of the thyroid gland.
- First-time patients as well as follow-up appointments. (Most patients had been referred through their GP. Sometimes this was for particular confirmation of a diagnosis or delivery of specific treatment).
- Patients who have requested to see a consultant for a second opinion or further information on their condition.
- Patients who are receiving on-going treatment and monitoring for more complex and/or recurring thyroid disease.
- Patients who have other diseases and conditions that might make the treatment for thyroid disease more complex and vice versa.

After each consultation, patients were sent a copy of the letter that the Consultant sent to their GPs and other clinicians who the patients might have been seeing and/or who would aid in any treatment required for their disease¹². The practice of copying all correspondence between clinicians to patients concerning their case was implemented in 2004 in response to guidelines suggested in the NHS plan (2004). The rationale for this is:

¹² For example, Wendy, a female patient in her fifties, was going to the thyroid clinic because she had been rendered hypothyroid by the lithium she was taking for her bi-polar disorder. Therefore, copies of her clinic letter were sent to her GP and psychiatrist so they were kept informed about her condition.

The NHS has an obligation to involve patients in decisions about their health care and communicate with them. Copying letters is an effective way of keeping patients up-to-date with their diagnosis and treatment and demonstrates a commitment to good communications and valuing patients.(Department of Health 2003)

However, this policy aim of involving patients was often not met by this practice of copying patients into clinic letters. The data collected during the interviews suggested that often these letters were not the most effective way of communicating with the patient, as they often did not correlate with what they thought or remembered had occurred in the consultation. This was due to a number of reasons including the use of clinical terms that they had not heard of before and errors in the information, such as their dosage of medication, or the list of additional conditions they had (or did not have). These details were always listed at the top of the letter along with other details, such as date of birth and suspected or confirmed diagnosis (See appendix 2 for an example of a clinic letter). In addition, it is important to note that these letters were not written with the patient as respondent. They were essentially communications between the consultant and his colleagues (GPs or other consultants the patient was seeing). Therefore, the extent to which patients used these letters as a way of 'keeping up to date' with their diagnosis and treatment is often questionable. Frequently these letters were not used by the patients to piece together their account and understanding of their thyroid disease. Other information sources, such as their memories of specific comments that the consultant made, or particular events where certain symptoms developed, seemed to inform their understandings and accounts far more than these letters. Often, at the end of interviews, the patients would suddenly remember they had been sent these clinic letters and ask if I would like to see them, prefixing the offer with, 'I don't know how useful they will be though'. Therefore, the letters initiated to assist patients often do the reverse. As described above, they often emphasise the disconnection between medical 'facts' and patient realities.

In addition to the clinic letters, if further treatment was required, the Consultant would also enclose patient information sheets for various therapies being considered by the patients (see appendix 3 and 4 for an information leaflet the Consultant would send patients who had been diagnosed with hyperthyroidism and hypothyroidism). These seemed to be

read by the patients as they would often refer to them at their follow up consultations. However, acknowledgement of this reading of the information was often followed by a 'but'. Frequently, they still wanted more discussion with the Clinician in the clinical context and seemed to favour this interaction far more than any other form of information gathering that happened outside of 'clinic time'. Moreover, the interaction during clinic time became the most effective way of understanding the conditions, for both the patients and myself as researcher. The Consultant's practice and focus on treating the cases presented to him, one by one, person by person, became a very useful way of filtering the information about the conditions gathered from a bewildering array of websites, books and articles. Not only did the irrelevant information get ignored, but also, the common and repeated rules and phrases, with regards to explaining and treating the conditions, became recognisable. Therefore, this situated 'disease talk' was able to translate and condense vast amounts of information about thyroid disease and apply it to the individual case of the particular patient.

In preparation for observing the clinic, I read extensively about thyroid conditions. The more I read, the less the thyroid conditions were recognisable as 'traditional biomedical diseases' with clear-cut causes, categories of symptoms, and regimes of treatment,. Starting with patient support group websites (and their subsequent information packs) and moving on to the more technical clinical literature, I began to track and decipher recurring words such as 'subclinical hypothyroidism', 'euthyroid' and 'postpartum thyroiditis'. I became familiar with, and could identify, not only a basic aetiology of these diseases, but also complications, times of elevated risk for specific groups of patients and contested areas of knowledge between specialists. Yet a consolidated understanding of what thyroid conditions are continued to fall beyond my grasp. Some of this lack I can now identify as the result of reading about a condition as a social scientist in a manner that would be considered counter-intuitive by, for example, an endocrinologist or clinical biochemist. A lack of basic understanding of the biological systems and mechanisms in which the disease was located, such as autoimmunity or the endocrine system, meant that frequently I had to pause to look up words and define concepts. However, this anxiety was also a result of the highly non-specific nature of the symptoms of thyroid disease and the difficulty in connecting them to

objective measurements of disease such as the results of diagnostic blood tests. The anxiety caused by such a disjointed account of thyroid disease remained until I entered the clinic, and realised this discordance was a key characteristic of the condition. As the clinical narratives of the many observed consultations eventually became more familiar, it became clear that in the everyday practice of treating thyroid conditions only particular literatures within the field were useful or relevant in the therapeutic realm.

For instance, and as previously described in chapter three, the genetic predisposition to thyroid disease is both well acknowledged, but also, peculiarly tangential in relation to the diagnostic practices commonly used in the clinic. Research that has attempted to develop an understanding of the complicated genetic aetiology of predispositions to autoimmune thyroid conditions (Hall and Stanbury 1967, Chistiakov 2005, Tomer et al. 1997, Tomer et al. 1999, Tomer et al. 2001) may be of interest to, and understood by, a consultant physician in a NHS outpatient clinic, but is not necessarily used in the process of developing a diagnosis or treatment. On the 'frequently-asked questions' page of the British Thyroid Foundation website the following information is provided with regards to the question of whether thyroid conditions are 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)

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 when explaining 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. First, it does not offer a superior diagnostic tool to

the standard thyroid function blood test and second, knowledge about the disease process at a molecular level does not change or influence the available treatments.

Moreover, familial dispositions to thyroid disease were conceptualised by patients in yet another way. Often the vagueness of the hereditary nature of the predisposition to thyroid disease was made sense of by embedding it 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 of 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 multiplicity of ways in which knowledge about thyroid disease was practiced and understood was also reflected in the ways it was embodied across the patient population. For example, some patients complained of particular symptoms more than others, some patients complained of symptoms even when the evidence provided by blood tests suggested that disease was not present and vice versa. The realisation of the concurrent interrelation, lack of awareness and ignorance between literatures on, and experiences of, the same group of conditions of the thyroid gland proved to be crucial in grasping, then selecting what would be included and excluded when writing an account of the disease. Moreover, the location, patients and professionals with whom I was working fundamentally structured the form of this account. The everyday milieu of the clinic, the point at which clinical trials and best practice guidelines, for example, are brought into practice and mingle with the bodies of thyroid patients in the course of treatment, all structured this process of learning and selecting what information and knowledge was relevant and when. A process which, as described above, is also shared with the Consultant and patients through

the disease talk they perform in the clinic, where information about thyroid disease was selected and filtered and made it relevant to individual cases. Consequently, this account of thyroid conditions is probably very different from the one which would have been written if I had chosen to work with the molecular biologist hoping to understand the genetic aetiology of the diseases or in a laboratory with a clinical biochemist who analyses blood samples in order to diagnose thyroid failure. Therefore, this process of learning about thyroid disease confirms Mol's point that diseases are assemblages of situated knowledges and practices that shape the boundaries and experiences of particular pathologies.

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 1998b: 1).It averages approximately 20 grams in weight (Toft 1995: 1).

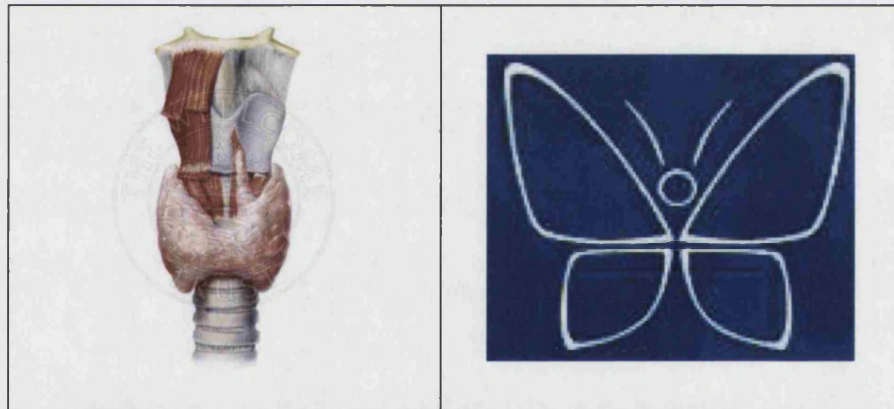


Figure 5: The thyroid gland (Wellcome Collection)

Figure 6: Logo of the British Thyroid Association (BTA)

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 in order to facilitate a number of different functions and processes in cells around the body. As the British Thyroid Foundation Website explains in its 'What is the Thyroid Gland?' section:

The thyroid gland is an endocrine gland. This means that it is a gland that manufactures certain hormones, which are chemical substances secreted into the blood-stream and act as

messengers to affect cells and tissues in distant parts of your body. (The British Thyroid Foundation 2006)

The endocrine system, its glands and the hormones they secrete are commonly conceptualised as a communication system in which messenger chemicals or hormones 'regulate' and 'tell cells what to do' to ensure that various processes and conditions are maintained in a state of 'normal function' within the body. This idiom of hormones as the messengers of a regulation system found in the clinical and support group literature is also emphasised in the consultant's account of the thyroid gland to patients during their first appointment: 'so your thyroid gland is told by your pituitary gland to produce thyroid hormones...' The thyroid gland produces thyroid hormone thyroxine (T4) and, in much smaller quantities, triiodothyronine (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 there is not enough, the body cells work too slowly. According to Bayliss and Tunbridge in their book *Thyroid disease: the facts*:

For example, the growth and development, both physical and mental, of a baby depend upon the presence of the correct amount of thyroid hormones. We see this in the animal world too. Without thyroxine a tadpole will not change into a frog, and without thyroxine in the correct amount, a newborn baby will not grow properly, nor will his or her brain develop properly. (Bayliss and Tunbridge 1998b: 3)

In other words, in the absence of the 'correct' amount of thyroid hormones, bodies do not grow or function sufficiently. The specific 'conversation' that the thyroid gland has is orientated around its relationship to the pituitary gland. The pituitary gland, described as a 'pea sized structure, hanging from the under surface of the brain just behind the eyes, and enclosed in a bony depression at the base of the skull' (Bayliss and Tunbridge 1998a: 1), sends a hormone called thyroid stimulating hormone (TSH or Thyrotrophin) to the thyroid gland which 'tells' it to make thyroid hormones (T4 and T3). The process of thyroid hormone production consists of the cells of the gland combining iodine and the amino acid tyrosine to make T3 and T4. Via a process commonly described as negative feedback¹³ (Daniels and Dyan

¹³ 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.

2006: 10), the amount of TSH regulates the level of production of thyroid hormones ensuring that there is always the right amount of 'fuel' for metabolism to take place. This mechanism of thyroid function dependant on the relationship between the pituitary and thyroid gland is called the thyroid pituitary axis.

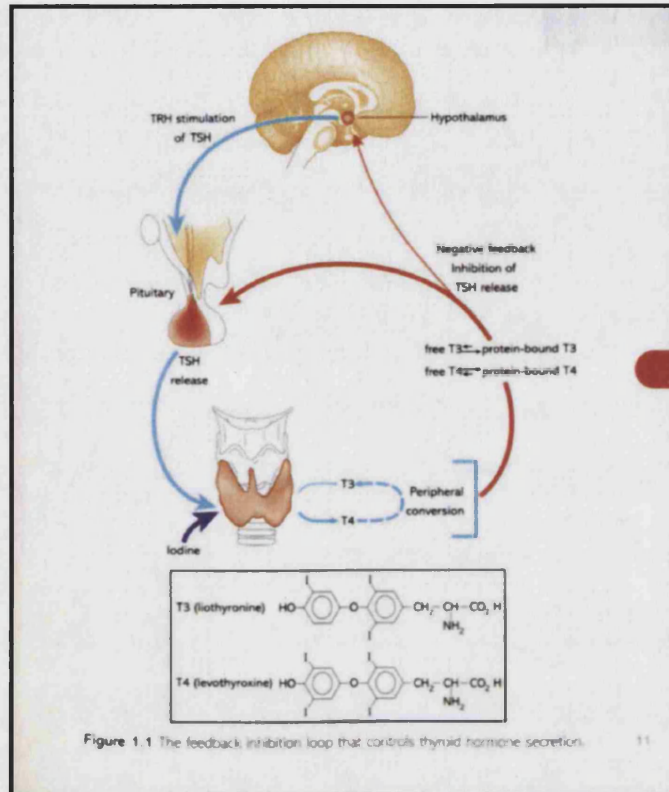


Figure 7: Diagram representing the feedback mechanism between the hypothalamus, pituitary gland and the thyroid gland (Daniels and Dyan 2006: 11).

Again, in keeping with the regulation idiom that describes the endocrine system as a whole, the relationship between the pituitary gland, TSH, thyroid gland and thyroid hormones is often described within the patient support literature as being 'like a thermostat' (Bayliss and Tunbridge 1998:5), as the following extract from the British Thyroid Foundation website demonstrates, the pituitary gland is the 'thermostat' for the metabolism, or 'central heating':

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)

T3 and T4 are released into the blood-stream and are transported throughout the body where they then perform their role of regulating metabolism. When this system is functioning normally and producing thyroid hormones at an acceptable level, thyroid function is defined as being Euthyroid. However, as the following diagram from *Fast Fact: Thyroid Disorders* (2006) by professor of medicine Gilbert H. Daniels and clinical researcher Colin M. Dayan demonstrates, the thyroid gland fails when this mechanism fails to control the level of production of the thyroid gland.

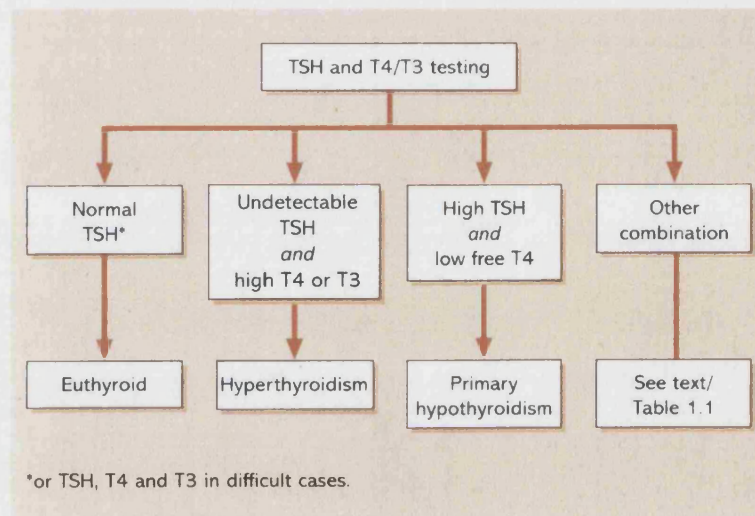


Figure 8: Guide to interpreting the results of thyroid function blood tests (Dayan and Daniels 2006: 13)

As the diagram depicts, when the level of TSH in the blood is low or undetectable and the production of thyroid hormones is high it means that the thyroid gland is producing too much thyroid hormone. That is, in spite of the pituitary gland's attempt to stem the production of thyroid hormones by reducing the amount of TSH it releases, the thyroid gland is unable to respond and continues to overproduce T4 and T3. As the diagram illustrates, this state of increased thyroid function is called hyperthyroidism. In contrast, when the level of TSH is high and the production of thyroid hormones is low, thyroid under activity occurs. Again this state is a result of the failure of the feedback mechanism. That is, in spite of the pituitary gland's effort to encourage the thyroid gland to produce more T4 and T3 by releasing more TSH it fails - a state that is termed hypothyroidism.

Consequently, thyroid disease can be defined as the disturbance of this feedback mechanism resulting in various states of thyroid malfunction.

Such pathological states of thyroid function are identified by the presentation of particular signs and symptoms (that will be detailed shortly), but primarily through a thyroid function blood test (TFT) that measures the level of TSH and thyroid hormones in the blood. Once the levels of these hormones in a patient's blood have been established they are then compared to reference ranges that represent the 'normal' levels of these hormones in a reference population of healthy individuals. As Dr Geoff Beckett, a Reader in Clinical Biochemistry at the University of Edinburgh, explains in an issue of the British Thyroid Foundation newsletter:

When interpreting blood tests, doctors need to know how a patient's results compare with the range of values found in a group of selected individuals who are considered healthy. The 'healthy individuals' are known as the 'reference population'...Once the results of the blood tests in the reference population are known, the 'reference range' is defined as the range of values found in the mid 95% of the reference population. Thus a reference range, by definition excludes the values for the test found in the 2.5% of normal subjects with the highest values and the 2.5% of normal subjects with the lowest values. This means that 5% (1 person in 20) of the healthy ('normal' population will have a test result that is either slightly above or below the reference range. It also means that if your doctor does 20 different test on your blood, there is a very good chance indeed that one of these blood tests will be 'slightly' outside of the reference range (i.e. appear to be abnormal) even though you may be perfectly healthy. Note the emphasis on the word 'slightly'. A result that is very much higher or lower than the reference range is highly likely to indicate that illness is the cause of the abnormal result. Since some 'normal' individuals are excluded from the reference range the use of the term 'normal range' is considered to be misleading and inappropriate: it is thus no longer used. (Beckett 2003: 5)

Consequently, for people whose TSH, T4 and T3 are 'very much higher or lower' than the reference range (i.e. beyond the 2.5% found at either end of the reference range) thyroid dysfunction is present. Currently, according to clinical practice guidelines the reference ranges used to measure thyroid function are as follows (Association for Clinical Biochemistry et al. 2005: 56):

Thyroid stimulating hormone (TSH)	0.4 – 4.5 mU/L
Free T4 (FT4)	9.0 – 25 pmol/L

Free T3 (FT3)	3.5 – 7.8 nmol/L
Total T4(TT4)	60 – 160 nmol/L
Total T3 (TT3)	1.2 – 2.6 nmol/L

However, in addition to these clearly defined states of thyroid function there are also subclinical states that suggest that thyroid dysfunction is: mild, too mild to treat at the present time and 'may occur in the future'. These subclinical states of thyroid function occur when the TSH is either too high or too low, but the production of thyroid hormones remains within the reference range. As the Consultant described in the clinic these subclinical states can be described as the thyroid pituitary axis working at 'full pelt' to maintain the normal function of the gland. Currently, the guidelines state that subclinical hypothyroidism should only be treated if the level of TSH is greater than 10, consequently TSH that is between 4.5 and 10 mU/L although abnormal is considered asymptomatic. Decisions to treat subclinical hyperthyroidism, defined as a TSH below 0.4 mU/L, FT4 above 25 and FT3 above 7.8 pmol/L should be referred to a specialist who should investigate the underlying cause of the mild dysfunction and decide on treatment accordingly. In the case of both subclinical hypo and hyperthyroidism where treatment is deferred it is suggested that thyroid function should be tested regularly for life or until it becomes overt. However, as will be described throughout the thesis these borderlands of thyroid disease are fraught with disagreement and uncertainty. Specifically a number of patients and sympathetic clinicians believe that even the mildest of these subclinical states do cause symptoms. Consequently thyroid function tests and the reference ranges used to interpret the results are considered by these groups to be inaccurate. In the following sections the various pathological states of thyroid function will be described through symptomatic and biochemical categories and treatments for them will be detailed. As these descriptions unfold, it will become clear that the rules of thyroid disease are often ambiguous, and when situated in relation to the experiences of patients and the practices of the clinic, become partially incoherent, confusing and even frustrating.

Disease of the thyroid

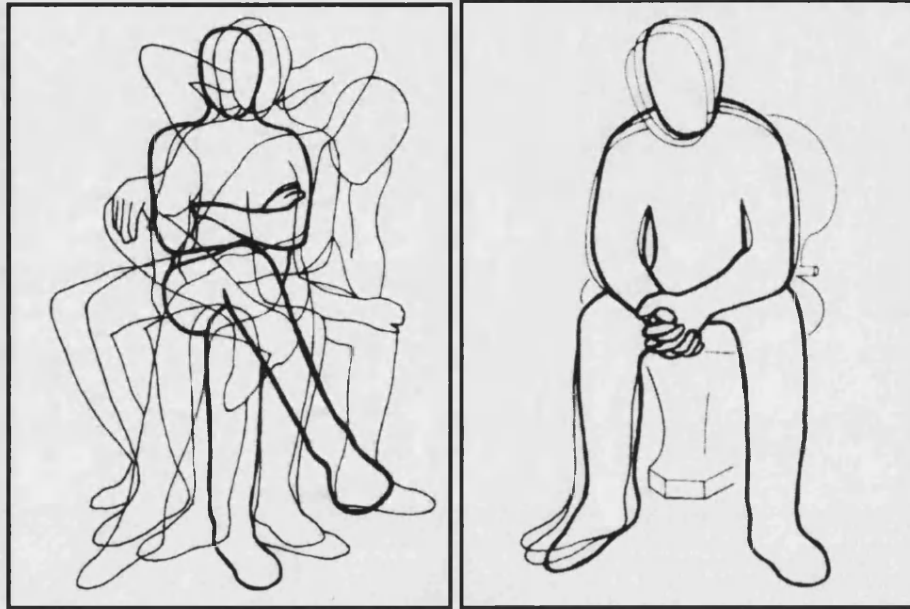


Figure 9: Illustration depicting the physical states of the body when an individual is hyperthyroid (Bayliss and Tunbridge 1988: 42)

Figure 10: Illustration depicting the physical states of the body when an individual is hypothyroid (Bayliss and Tunbridge 1988: 93).

As described in the previous section disturbance of thyroid function falls into two functional categories - hyperthyroidism or hypothyroidism. The disturbance of thyroid function is attributable to a number of factors, all of which differ in significance with regards to diagnosis and treatment. Therefore, whilst diseases of the thyroid have a number of causes, in the clinic they are generally considered to be of secondary importance, as treatment is organised around the restoration of the feedback mechanism between the pituitary and the thyroid gland, and not the underlying cause of dysfunction. These states of dysfunction will now be described through published clinical literature, patient support group information, interviews with patients and observations from the thyroid clinic.

Hypothyroidism

Hypothyroidism is described as 'an insidious condition with significant morbidity' (Association for Clinical Biochemistry et al 2005 : 11) because of the wide range of non-specific symptoms that it causes. These include a goitre (swelling of the thyroid gland that is not necessarily a symptom of

dysfunction), weight gain, brittle nails, the loss of the outer third of the eyebrow, constipation and lethargy. (see appendix 5 for a full list of symptoms from the patient organisation the British Thyroid Foundation). However, as clinical experts Daniels and Dayan point out their text book entitled *Fast facts: thyroid disorders*, the attribution of these clinical symptoms alone to thyroid disease is problematic:

Both euthyroid and hypothyroid individuals may complain of fatigue, weight gain, dry skin, mental torpor, depression or constipation. Muscle cramps, joint pains, menorrhagia or infertility may be unrecognized clues to the presence of hypothyroidism. Marked obesity, or relentless weight gain, is seldom caused by hypothyroidism. The clinical diagnosis of hypothyroidism is often unreliable. (Dayan et al. 2002: 74)

In sum, they conclude that the non-specificity of thyroid type symptoms, in particular those suffered by women such as menorrhagia (heavy periods) or infertility, mean that clinical diagnoses based on symptoms alone is unreliable. Moreover, often when patients were describing the onset of their disease they were quite explicit about how non-specific and slow moving it had been. Their accounts described a lengthy process over months and even years during which they developed feeling of not being 'quite right', Therefore a key characteristic of thyroid disease is its highly ambiguous symptomatology. As Alexis, a hypothyroid woman in her early thirties, recounts;

And remember thinking, oh I just seem to be putting on weight for no apparent reason and I was quite tired, and so I went to St Lucia and came back and I just thought oh I must be really jetlagged all the time. My friend kept ringing me about 7 o'clock at night but I was already asleep. And then she kept saying to me what is wrong with you, and I says what do you mean, and she said well nobody goes to bed at 7 o'clock you know there's something wrong with you. And I was like well I guess I'm just jetlagged you know, and I think you know, I'd put on quite a bit of weight even though I wasn't really eating anything. And I was tired all the time and I kept thinking oh I wonder what it is.

The non-specific aspects of the disease are, therefore, not only reflected in the clinical symptoms of the disease but also in the ways in which these symptoms become manifest in the everyday lives of the sufferers. Moreover, (as Alex demonstrates) they are often hard to identify as symptoms because they can also be easily attributable to other factors. For example, she describes how she initially thought her lethargy was caused by jet lag.

The causes for the disturbance in thyroid function resulting in under activity are typically described in text books as the following:

- Iodine Deficiency – a deficiency in iodine in the diet which means that the thyroid gland has no iodine to convert into thyroid hormones. Described as 'rare in the USA and uncommon in Western Europe' due to public health measure such as fortifying mains water supplies with iodine where necessary. (Daniels and Dyan 2006: 69) This cause of hypothyroidism was not observed in the clinic.
- Iatrogenic Hypothyroidism – Resulting from a surgical removal on the gland due to excessive enlargement or radioiodine treatment that is also used to stem over-activity, which will be discussed in more detail shortly (Toft 1995: 13). Hypothyroidism can also be caused as a side effect of Lithium, used in the treatment of Bi-polar disorder and Amiodarone (Toft 1995: 26), a drug used to regulate the heartbeat.
- Chronic Autoimmune Thyroiditis – Most commonly called Hashimoto's Thyroiditis after the Japanese clinician who first described the auto-immune attack on the gland (Dr. Hakaru Hashimoto) and the most common cause for hypothyroidism in iodine sufficient regions. The immune system attacks the gland as if it were foreign, subsequently destroying it (Bayliss and Tunbridge 1998: 83)
- Postpartum Thyroiditis – A variant of Hashimotos Thyroiditis which is triggered by pregnancy and diagnosed if the disease occurs up to one year after pregnancy. After the first occurrence of postpartum thyroiditis there is a 50-75% risk it will reoccur in subsequent pregnancies. It is often transient, however, in some cases it is persistent, even up to two years after pregnancy (Daniels and Dyan 2006: 101).
- Thyroid conditions also vary in prevalence according to iodine levels in different geographic regions. Therefore, in iodine-replete

populations such as Western Europe and the USA, where public health measures have been used to correct iodine deficiency in diet, autoimmune thyroiditis is the most common cause of hypothyroidism. Thyroid conditions in iodine replete populations, are therefore, described as affecting 2% of the population and as being ten times more common in women than men (Daniels and Dyan 2006: 70).

However, as suggested previously, within the clinic the cause of the disease is not the primary location of intervention. As the extract below outlines, with reference to Hashimoto's Thyroiditis, the cause of the disease is not a factor that can be addressed in treatment:

We have no safe reliable way of modifying the faulty immunological system that mistakenly believes your thyroid cells are 'foreign'. Thus the basic cause of Hashimoto's thyroiditis is untreatable, although the symptoms can be totally alleviated. (Bayliss and Tunbridge 1998: 86)

As this passage suggests, the treatment of hypothyroidism is relatively simple entailing the administration of synthetic T4 replacement called thyroxine, which is a generic preparation and is therefore relatively cheap. T3, the other hormone that the thyroid gland produces, is not usually offered as part of this replacement therapy as the general consensus amongst thyroid specialists is that it offers no additional benefit since it is a short acting and unstable hormone (Clyde et al. 2003) (Also appendix 6 for see the BTA's executive committees statement on Armour Thyroid (USP) and combined thyroxine/ tri-iodine as Thyroid Hormone replacement, an issue that will be discussed in more detail in chapters five and six). This treatment regime can be administered at GP level and functions alongside biannual TFTs that can monitor any changes in thyroid function and subsequently adjust dosage as appropriate. However, many of the patients did not experience the treatment of thyroid conditions as a straightforward procedure conducted by their GP. As Laura reflects:

...I mean you don't want to have to take medication if you don't have to do you? Especially if you've got to take it for the rest of your life. No I'm not happy about it...

Often the consultant would stress that having to take thyroxine was not like taking medication for the rest of one's life. Rather it is a replacement

therapy that merely 'tops up' what the thyroid is no longer able to produce. This disparity between the attitude of the clinician and the patients, with regards to the straightforwardness or seriousness of the treatment for hypothyroidism, is linked to how it is conceptualised – either as a one off medical event or a disease that would have to be managed for life. For the clinician it was simply a case of restoring the feedback mechanism by topping up hormone production; for the patient it was about taking a pill for the rest of one's life. Therefore, the context in which the disease is perceived is fundamental to determining its severity. For patients the disease extends into the future; for the clinician it is dealt with in the time that it takes to diagnose then correct the levels of TSH and thyroid hormones in the blood. As will be described in chapter five, the clashing temporalities of thyroid disease presented by some patients and clinicians can have a significant impact on the success of and satisfaction with established treatment practices.

This discrepancy in the seriousness of hypothyroidism can also be found in terms of the expertise that is deemed necessary for its treatment. 'The Consensus Statement for Good Practice and Audit Measures in the Management of Hypothyroidism and Hyperthyroidism' (Vanderpump et al. 1996) states that hypothyroidism can be sufficiently treated at GP level. Referral to a specialist is supposed to depend on the presenting signs and symptoms, the expertise of the doctor making the referral, access to a specialist, local circumstances and patient preference. All of the hypothyroid patients who were referred to see the Consultant were usually there for one of two reasons: either their GP. was not sure how to treat and diagnose the condition and wanted reassurance from a specialist or the patient had asked to see a consultant as a preference. In cases of patient preference, the Consultant's views were sought because the patients wanted reassurance or a second opinion. Often these reassurances were motivated by what some patients felt to be the discrepancy between their self-reporting symptoms and the results of their TFTs. Usually these claims were countered by the consultant with the seemingly irrefutable evidence of a 'normal' blood test result, which is regarded as a definitive marker of the absence of disease due to the unreliable nature of the many wide-ranging and non-specific symptoms. It is this ambiguity and disagreement between the types of evidence (symptomatic and biochemical) that will become the critical focus of the essay and attempt to address the central research

question - how and why the 'natural categories' of thyroid disease (thyroid physiology and pathology) are constituted, maintained but also, contested by both clinicians and patients.

Hyperthyroidism

Hyperthyroidism, is described as having significant short term and long term morbidity and occurs in 0.5% of the U.S. and U.K. population (Daniels and Dyan 2006:26). Hyperthyroidism describes the presence of disease due to excessive production of thyroid hormones by the gland. This results in metabolism working too quickly causing disease. Again, the symptoms of hyperthyroidism are described as insidious and are non-specific, wide-ranging (see appendix 7 for a full list of symptoms from the patient organisation the British Thyroid Foundation). And again patients felt these symptoms manifest as just 'not feeling quite right' over months and even years. Many patients described how they realised their bodies and their minds were working 'overtime', leaving them exhausted and irritable. However, they also added that they found it impossible to 'stop' – sit down, relax, stop thinking and even sleep. For example within the clinical literature these ambiguous symptoms are described as follows:

Tiredness is often an early symptom, to be followed by weight loss palpitations or increased awareness of your heartbeat, nervousness and particularly irritability so that you have 'a short fuse' and increased sweating. Looseness of the bowels is not uncommon and sometimes diarrhoea maybe a prominent symptom...You may feel hot and be uncomfortable in hot weather. You may complain that the central heating is set too high or throw off the bedclothes at night, yet your partner complains that it is not that hot. Your skin may itch but there is no rash...(Bayliss and Tunbridge. 1998: 41)

Causes for disturbance to the function of the thyroid gland resulting in Hyperthyroidism include the following:

- Toxic Nodular Disease – A condition where clumps of over-active cells develop and render the production of the gland excessive. This can be in the form of one or many toxic or 'hot nodules'. Generally toxic nodular disease this accounts for 20% of all cases of hyperthyroidism seen in clinics. Solitary toxic nodules can also occur, where a single hot nodule of overactive cells cause

hyperthyroidism, this accounts for 5% of hyperthyroidism. (Daniels and Dyan 2006: 119-123)

- HCG - HCG (human chorionic gonadotrophin) is an important pregnancy hormone that is secreted at the moment of conception. Sometimes this hormone can be overproduced and therefore stimulate the thyroid gland in a state of over-activity. As this condition is transient (i.e. only occurs during the early stages of pregnancy), it usually requires no treatment (Daniels and Dyan 2006: 96). Often called gestational hyperthyroidism, this was not observed in the clinic.
- Iodine Induced Thyrotoxicosis – Another iatrogenic cause for thyroid dysfunction. Pharmacological doses of iodine, found in various medications can cause excessive production in the gland (Daniels and Dyan 2006: 29) .
- Graves Disease – The most common cause for hyperthyroidism is Graves Disease - an autoimmune condition like Hashimoto's disease, where the gland is stimulated by antibodies of the immune system into over-production. This disease made up the majority of hyperthyroid patients seen in the clinic.

As with Hypothyroidism, the most common cause of Hyperthyroidism is an auto-immune condition called Graves Disease, which is named after the Irish physician, Robert Graves, who first described the symptoms in detail over 200 years ago (Toft 1995: 5). Also, as with Hashimoto's Thyroiditis, Graves Disease is an auto-immune disorder that is considered to be familial. However, in contrast, the thyroid 'stimulating' antibodies encourages the gland to over-produce thyroid hormones, instead of under produce as is the case with hypothyroidism. There is evidence to suggest that the onset of Graves Disease can be partially attributed to traumatic life events, commonly 1 to 2 years before the onset of the disease (Paunkovic et al. 1998)¹⁴ and smoking. Again it is more common in women than men,

¹⁴ This study demonstrated that Graves disease increased significantly during the Bosnian War (1992 – 1995) suggesting that Graves disease could often be triggered by extreme stress

but, although, the proportion of male sufferers is higher in comparison to hypothyroidism.

In addition to the disturbance of thyroid function, a possible complication of Graves Disease is a related eye condition that is also caused by the auto-immune disease mechanism. In most cases of hyperthyroidism, there are some optical symptoms. A recognised symptom of the disease is lid retraction, where the upper eyelids are pulled upwards exposing the whites of the eyes resulting in a 'staring quality, like that of an actress when she wishes to convey an expression of anxiety or terror' (Bayliss and Tunbridge 1998: 63). In addition, lid-lag can occur where the lids are slow to follow the movements of the eye.

However, for patients with Graves Disease, the most common cause of Hyperthyroidism, there is, in addition, more specific eye involvement. A secondary, yet often related auto-immune attack may occur, which targets the muscle tissues that move the eyes. This attack can happen independently of Graves Disease or often months or years after, or even before its onset. However, it is more common as a related occurrence of Graves Disease, and is thought to be the result of a cross reaction between the thyroid antibodies and similar antibodies found in the tissue around the eyes (Daniels 2006: 39). Approximately 10-20% of patients with Graves will develop eye disease, 5% of which will be severe cases. The condition is more common in men and smokers when compared with the Graves Disease population generally.

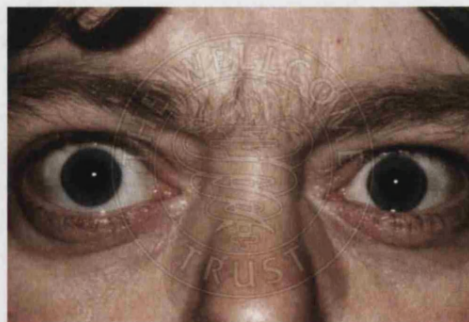


Figure 11: A photograph of the eyes of a patient with Graves disease (Wellcome Collection)

In cases of auto-immune eye disease, the eyes are pushed forward due to the inflammation of the muscles, becoming puffy and watery due to impaired drainage. They feel gritty and sore and are more prone to infection as they are less protected by the swollen eyelids. Their movement becomes laboured due to the impairment of the muscles. This muscle

impairment results in double vision and the increased pressure in this area, due to the inflammation, which in some cases can result in restriction of the optic nerve that may subsequently mean that eyesight is threatened. Many patients with these symptoms also felt that the disease was particularly upsetting due to the extreme change to their appearance. As Diane, a Grave's disease patient in her early fifties with extensive eye involvement, explains, she 'feels like a monster' and 'feels like people are looking at her all the time'.

Treatment for Hyperthyroidism is more complex and must be overseen by a consultant in a hospital. Once diagnosed, the first stage of treatment is the administration of a drug, most commonly Carbimazole, to suppress the activity of the gland. The dosage of this therapy is imperative; if it is too high it will cause hypothyroidism and if too low it will not relieve symptoms. In order to reach this balance, the dosage of Carbimazole is titrated against TFTs (every 4 to 6 weeks) until they eventually enter the normal range and a maintenance level has been reached. When this level has been reached, TFTs on this dose will be performed every three months. However, this treatment is not permanent and will not stop the disease for good, something that the consultant made very clear to the patients after they had reached a stable maintenance dose. Consequently, in order to overcome the disease for good a 'definitive treatment' that could halt the over-activity of the gland permanently was offered. Traditionally this consisted of the removal of the gland through surgery (thyroidectomy), but this is now considered to be an out of date and unnecessarily invasive method. Currently the preferred treatment is Radioiodine Therapy (RI) that halts Graves Disease, the activity of 'single hot thyroid nodules' and 'multi-nodular toxic goitres' for good. Briefly, RI is administered through the hospital's Nuclear Medicine department and consists of the patient swallowing a radioactive capsule from which radioactive iodine will be taken up by the thyroid gland. This, in turn, destroys the tissue of the gland; either minimizing its production of thyroid hormones or stopping it functioning all together. In some cases, if the hyperthyroidism is severe, more than one dose of RI will be required.

Patients who were undergoing this treatment were required to stop taking their Carbimazole a week before their RI treatment. In addition, patients with Thyroid Eye Disease (TED) must take a steroid to protect their eyes, as the

antibodies found in their gland are also found in their eyes, and could, therefore, destroy tissue in that area. The treatment itself is administered during an out-patients appointment in the department of Nuclear Medicine – a branch of medicine and medical imaging that uses radioactive substances in diagnosis and therapy. After this treatment has been administered that patient can return home, but they must stay away from children under 12 and generally avoid contact with large groups for up to two weeks. This part of the treatment, unsurprisingly, was described by many of the patients as incredibly upsetting and difficult, especially for those who had young children or grandchildren who they had to avoid. One woman who underwent the treatment described having the RI pill, which was contained in a sealed glass tube, passed across a table by a radiologist who quickly exited the room before she swallowed it. Another woman recounted that on leaving the hospital after swallowing the RI pill how her husband jogged in front of her in order to maintain a safe distance!

Post RI patients require life-long follow up to identify development of Hypothyroidism which is the inevitable consequence of 'knocking the thyroid out', as the consultant described it. Once post-RI patients become hypothyroid they will then be placed on a life-long regimen of thyroxine. Not surprisingly, many patients found the suggestion of this treatment quite shocking. Firstly, because of the danger associated with being fed a radioactive capsule, and secondly, because of the outcome of becoming hypothyroid and having to take thyroxine for life. In the clinic, I listened to many occasions when the Consultant attempted to convince patients with Hyperthyroidism taking Carbimazole to opt for RI, often using the argument that Carbimazole was not a definitive treatment and would not get rid of the disease and any subsequent degeneration. In terms of being rendered hypothyroid by the RI, the Consultant often used the argument that the patient's body could have attacked the thyroid, either making it Hyperthyroid or Hypothyroid. He would then argue that if the immune system had destroyed, and not stimulated, the gland they would be taking thyroxine anyway. Again, many post-RI patients on thyroxine complained of the persistence of symptoms, in spite of their medication.

Through this description of thyroid disease it has become clear that there is often a disparity between the formal explanations of and experiences and practices of thyroid disease. For instance, as has been described, the

biochemical model used as the basis for the diagnosis and treatment of thyroid disease often does not relate to the slow onset of the many vague clinical symptoms of thyroid disease. Subsequently, as has also been described, many patients found it difficult to relate to and accept the diagnoses and treatments that were offered on the basis of this rigid biochemical model. For example, correction of hypothyroidism and hyperthyroidism by 'simply restoring' the feedback mechanism between the pituitary and the thyroid gland was often questioned by patients. For many, the invasiveness of such treatments (RI and/ or thyroxine for life) seemed to be ignored by the straightforward approach of clinicians who focused their treatments around the restoration of a euthyroid state of the thyroid pituitary axis. Therefore, as claimed at the beginning of the chapter, in the process of diagnosing and treating thyroid disease, the basic model of thyroid biology and pathology seem partial and unable to account for the complexity of the disease, specifically the relationship between the thyroid pituitary axis and the non-specific clinical symptoms attributed to thyroid disease. In the following section of this chapter the contingent nature of 'the facts' of thyroid disease will be discussed with regards to a classic clinical symptom – a goitre. Through this discussion it will be further demonstrated that in the clinic there is a constant slippage between ideal models of thyroid disease and the practices used to attend to it as well as patient experiences of it.

Normal abnormality

As well as diseases that interrupted 'normal' thyroid function, a substantial number of patients presented abnormalities of the gland where no disease was present. Typically, these patients presented thyroid enlargement or lumps in the thyroid gland called goitres. As the textbook explanation below confirms, goitres are highly ambiguous entities:

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 an increased 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)

Generally, patients who presented no symptoms other than a swollen and lumpy thyroid were not considered to be suffering from any underlying clinical disease and subsequently required no treatment after they had been investigated to rule out 'anything sinister' (i.e. thyroid cancer), as the consultant tended to phrase it.

The examinations of all patients with a goitre, for whatever reason, followed a simple protocol. Firstly a physical examination of the gland would occur. For this the Consultant stood behind the sitting patient and reached round to the front of the lower part of the neck, locating the gland. The Consultant would then gently feel and press the gland to determine the form and size of the swelling or the number and size of any individual nodules. The consultant would then ask the patient to swallow, in order to further ascertain the size and positioning of the goitre. Sometimes he would comment on how the patient had a 'nice slim neck' making the goitre easy to see. However, most of the time this physical examination 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 GP who had first noticed it.

Palpation occurred in all the first consultations with patients and any follow-up appointments where the swelling was suspicious or was not clearly a side effect of thyroid dysfunction (hyperthyroidism or hypothyroidism) in order to monitor changes in a suspicious lump. During one consultation, where two medical students were present, the consultant encouraged them to take turns in 'seeing' the lump through this examination protocol. Telling them to ignore the books, where it was suggested to stand in front of the patients, the consultant advised them to make sure they always asked the patients to swallow and asked them questions, such as, 'is the swelling smooth or nodular?' or 'how many lumps can you identify on the left and right side and which, if any, is the biggest?'

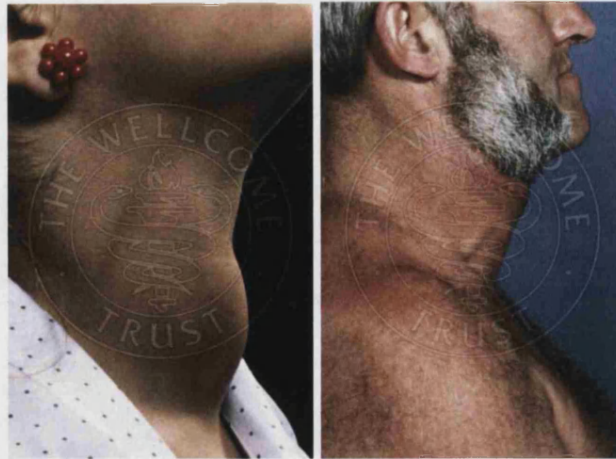


Figure 12: A simple non-toxic diffuse goitre (Wellcome Collection)
 Figure 13: A multinodular goitre (Wellcome Collection)

Often, in individuals from non-iodine replete countries, thyroid swelling occurs due to an attempt by the gland to increase its manufacturing capacity of thyroid hormones. This process is explained in the following case history taken from 'Understanding Thyroid Disorders' by Dr Anthony Toft a Consultant Physician and Endocrinologist and former President of the British Thyroid Association (BTA):

Ahmed was born in a village in the high mountains of northern Pakistan where he spent most of his childhood. At the age of 20 he came to London to study engineering when, at a routine medical examination, he was noticed to have a goitre. He felt well and his thyroid tests were normal. The cause of the goitre was attributed to an iodine deficiency when Ahmed told the doctor that most of the people in his village also had a goitre. His diet had contained enough iodine to prevent the development of Hypothyroidism, but his goitre is likely to remain, even though he has decided to live the rest of his life in a part of the world where there is an adequate amount of iodine in his diet (1995: 4)

In the clinic being observed by this study, Ahmed appeared in many guises, as this case history was applicable to a number of patients both male and female who were not of British origin. There was a middle-aged woman from Eritrea, a thirty year old man from Ethiopia and an elderly lady from Poland. In fact, the Clinician often explained the swelling to these patients as something that is common for people from certain parts of the world where the iodine in the water was low, adding that it was also significantly more common for those who lived in these environments until 'around the age of twelve'.

Within medical literature, iodine deficiency is attributed to the consumption of produce in locations where the soil has low iodine content such as mountainous areas, hence the origin of Toft's archetypal Ahmed in the 'mountains of northern Pakistan'. Areas in which the diet contains anti-thyroid or goitre-producing substances (goitrogens¹⁵) also have a high prevalence of goitres in the population. In areas such as these, goitres are found in more than 20% of the population and are therefore considered endemic – a 'natural' characteristic of the people of the region. Where goitres are endemic there is also usually the addition of one major socio-economic causal factor - iodine deficiency and subsequent thyroid swelling which is the result of malnutrition. In fact, it is believed that 500 million of the world's population have goitres, the majority of whom live in developing countries. This tends not to be thought of as a causal factor in the U.K. as it is considered to be an iodine-replete community, where malnutrition is uncommon. When iodine deficiency is discussed epidemiologically within various literatures, with regard to Western Europe, it is described as mild and specific to lowland areas and/or those situated far from the ocean. For instance, in the U.K. a goitre is traditionally known as 'Derbyshire Neck' (Bayliss and Tunbridge 1998: 115) as it used to be a common feature of the population in the county due to the low local iodine levels, before corrective public health measures were taken to fortify salt and bread with iodine. Thus, in contemporary literature directed at the investigation of the goitre in iodine replete communities another explanation is offered. Where no disease is present, the development of a goitre is attributed to unknown yet 'complex reasons' (Toft 1995: 35) - a common abnormality that happens to a substantial proportion of the population. The 'normality' of a goitre is something that much of the literature is quick to point out. For example;

It isn't normally a problem appearance wise - quite the opposite as far as some people are concerned. For example, the great seventeenth and eighteenth century artists often added a goitre to the female figure to enhance her beauty! (Toft 1995: 36)

The correct term for a goitre that is the result of a uniform swelling of the gland is a Simple Non-Toxic Diffuse Goitre. Simple implies that the gland is

¹⁵ Goitrogens are foods such as those of the brassica variety (broccoli, kale, cabbage), or even milk from cattle fed on such foods, that can suppress the function of the thyroid gland by interfering with the synthesis of thyroid hormones. As Bayliss and Tunbridge (1998) note goitres are common in some parts of Africa where cassava (a woody shrub and goitrogen) root is a staple food. It is also a staple in parts of South America, India and Indonesia.

uniformly enlarged, not hard and benign, Non-toxic implies that it shows no signs of a disease causing autoimmune process (Graves Disease or Hashimoto's Thyroiditis) and Goitre simply means enlargement. As consultant endocrinologists Bayliss and Tunbridge state in their book *Thyroid Disease: The Facts*, a 'Simple Non-toxic Diffuse Goitre' is a 'time honoured clinical description of a certain type of goitre' yet there is 'nothing simple about a simple non-toxic goitre!' (Bayliss 1998: 111) i.e. where no disease or environmental factors are present a goitre is an unexplained and complex phenomena of the thyroid gland.

Therefore, in a clinical context, whilst goitres are defined as an abnormality of 'normal thyroid physiology', without the accompaniment of symptoms or abnormal thyroid function blood tests, they tend to be left alone. The only exception to this process of diagnosing a goitre as a 'safe abnormality' is if it enlarges too much. Sometimes this benign process can result in the need for treatment, as goitres can become so large they put pressure on the windpipe, or become involved with surrounding nerves or the vocal cords, resulting in a 'hoarse voice'. In cases such as this, the thyroid is surgically removed (a thyroidectomy) resulting in the necessity of thyroxine therapy for life in order for the patient to substitute thyroid manufacture lost by the elimination of the gland. However, this level of enlargement is rare and tends to be reserved for extreme cases where, as was the case with one woman who had visited the clinic, there was 'retrosternal extension [growth down behind the breast bone] and severe tracheal compression' (extract from clinic letter to the general surgeon who would perform the total thyroidectomy).



Figure 14: An x-ray of a retrosternal extension. (Wellcome Collection)

Over time, Simple Non-toxic Diffuse Goitres may become smaller, or larger, or more irregular and eventually develop nodules and become a multinodular goitre. Often, the Consultant would tell patients with this presentation that this sort of thing can 'just happen', the thyroid gland 'is

like breast tissue, it can change and get lumpy', the thyroid can simply swell in this manner 'out of the blue'. This nodular pattern of enlargement, as well as being a symptom of the aforementioned endemic and unexplained thyroid swelling, is also symptomatic of 'hot nodules' which, as described earlier, cause hyperthyroidism. Because of the stronger association between disease and multi-nodular goitres, hot nodules tend to fall under a regime of long-term surveillance rather than a one-off diagnosis of harmless enlargement of the gland.

When these types of swellings appeared in the Clinic, the patients were usually sent to undergo an ultrasound scan to ascertain the number and size of the nodules. In cases where there was a large, fast growing or unequal distribution of nodules between the lobes of the thyroid, a Fine Needle Aspiration (FNA) test was performed. This test consists of a needle being passed into the nodules in order to remove cells for analysis, and ascertain the cytology of the nodule - benign or malignant. The likelihood of thyroid cancer was explained by the Consultant as one-in-twenty and throughout the literature is defined as extremely rare:

Compared with the incidence of malignant disease elsewhere in the body, thyroid cancer is responsible for less than 0.5 per cent of all deaths from cancer. Thus thyroid cancer is rare (Bayliss and Tunbridge 1998: 127).

Moreover, as the following extract demonstrates, much of the literature is concerned with drawing attention to the rarity of thyroid cancer rather than, for example, encouraging self examination for it:

Although people who develop thyroid nodules often worry that the lump may be cancer, this rarely turns out to be the case. (Toft 1995: 43)

Often, FNA tests came back inconclusive and were performed again, as too much blood and not enough cells were collected, so no 'definitive cytology' could be decided upon. On these occasions, the Consultant would refer patients to a Radiologist at a neighbouring hospital (but belonging to the same Trust) whose expertise with this type of test was highly regarded, especially if the nodules were in 'awkward places'. Much of the work done by the Consultant in these cases was to reassure the patients that the lumps were not sinister. For instance, in a follow up letter to one patient's

GP the Consultant wrote that 'For reassurance I have organised for her to have an ultrasound scan plus FNA to obtain a definitive cytology'. Many goitres are, therefore, defined not only by how they look and feel, but also by how they are not a symptom of any underlying disease; being instead a 'common' and 'benign' 'abnormality'.

Many patients who passed through the clinic had various lumps and bumps double-checked and monitored and after investigation, were reassured they posed no threat to their health. They could live with them quite normally. After their lumps had been investigated and deemed safe, these patients tended to be discharged from the clinic after two appointments. Typically, the Consultant would reassure them at the end of the process that they were more than welcome to return to the clinic if they felt their thyroids had got bigger and had begun to cause problems. However, the idea of a lump that would be left alone was often unacceptable and confusing for some patients, as they were often not quite convinced of the safety of leaving such abnormalities. They often asked the Consultant, after he had delivered his assessment, if he 'was sure' about leaving their lump or goitre alone. In addition, some patients wanted their goitres removed for cosmetic, as well as what they (and not the clinician) perceived as health, reasons. One woman, who was referred to the clinic with a thyroid swelling, wanted to discuss the option with the consultant of being given replacement thyroid hormone (thyroxine) to shrink her goitre, which she felt had been steadily getting bigger over a number of years. The consultant quickly responded saying there was no evidence that this worked at all and was an unnecessary treatment. He also added, after an examination of her neck, that she was like a number of women who tended to lay a fat pad on their neck, which in her case made her goitre look bigger than it actually was. In the follow up letter to this patient and her GP the Consultant added that there is no family history or documented thyroid dysfunction, surmising that:

In these circumstances I think it highly unlikely that any therapy will make any major cosmetic difference to her and I did not think it was warranted. I reassured her that all was well and certainly there was nothing in the clinical picture here to suggest any sinister aetiology and I have been as reassuring as possible. She will continue to have an annual check of thyroid function from now on. So the existence of a goitre can clinically mean a number of things - the presence of a number of

diseases that disturb thyroid function, as well as 'normal' and 'non-pathological' abnormality.

What is thyroid disease?

As I have argued in this chapter, 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 the result of a straightforward pathological process. In exploring these representations and analysing them here, I have deliberately pointed to their indeterminacy as a means of emphasising what I perceived as a strong common thread throughout both the literature I read and the conversations I observed and participated in; namely that to understand thyroid conditions and the experiences of people who suffer from thyroid conditions it is necessary to acknowledge, and even to prioritise, the substantial grey areas that prevent easy distinctions between normal and abnormal, diseased vs healthy, euthyroid/hyperthyroid, euthyroid/hypothyroid and knowledge/practice. As I have tried to show, the experience of the disease shared by most patients of 'not feeling quite right' due to the array of non-specific symptoms they suffer from over months, weeks and even years, means that their understanding of the disease is overwhelmingly ambiguous. Repeatedly in interviews it was impossible for the patients to decide when they first started feeling the symptoms of their thyroid dysfunction. Moreover, it was difficult to decide if these 'symptoms', even after diagnosis had been confirmed, were a result of the thyroid disease or just life in general – work, childcare, aging, stress, diet, relationships, etc. Amidst this fog of symptomatology, a key area of ambiguity that often caused confusion and sometimes even anger in the clinic, was the essential, but often opaque, relationship between the function of the thyroid pituitary axis, measured by TFTs, and the clinical signs and symptoms with which these levels were assumed to be correlated. Due to their non-specificity signs and symptoms are considered by clinicians to be unreliable forms of evidence that cannot be directly attributed to a pathological state of thyroid function. That is, the symptoms of thyroid dysfunction are often considered to inhabit the realm of illness, and are therefore considered to be social and subjective (Turner 1996: 198) indicators of disease that are not based on scientific fact - what is 'actually going on' in an individual's thyroid gland.

However, and to the dismay of patients and clinicians alike, the difficulties of diagnosing vague and incoherent thyroid symptoms actually seems to be escalated by the practical responses deployed to treat it. The transformation of vague thyroid symptoms into laboratory-based biochemical measures is so disconnected from its symptomatic presentation that its clinical diagnosis often contributes to the disjointed and awkward narrative of thyroid biology and pathology, rather than alleviating it. Although clinical texts and medical experts can accept the vagaries of the disease when elucidating its symptomatic presentation, they cannot easily translate this understanding when explaining and delivering treatment to patients, especially within the narrow and rigid biochemical idiom that defines current treatment procedures. Descriptions of disease in the clinic, in textbooks and in patient support group literature are effective in linking the often bizarre list of symptoms together to create a realistic account of what it *feels* like to have a thyroid disease. However, when the business of *treatment* needs to be attended to, this complexity is lost and symptomatic ambiguity is abandoned. Indeed, as will be discussed in the following chapters, some clinical symptoms are entirely ignored as factors relevant to the decision as to whether treatment is required or has been successful. As a consequence many patients are left distressed, confused and even angry due to a high degree of 'existential uncertainty' that such a paradigm of clinical care produces. To be precise, the technical and explanatory deficiency of TFTs to account for thyroid disease in some patients compounds and amplifies the feelings of uncertainty that are already inherent to the state of being ill.

As the sociologist Christopher Adamson notes with regards to his study and personal experience of Bowel Disease and Avascular Necrosis (1997) we most commonly and intensely experience uncertainty during periods of illness. As Adamson goes on to describe patients, in collaboration with medical professionals, perform a number of tasks related to diagnosis and treatment and in doing so confront a range of uncertainties. For instance: those associated with the disease course, diagnosis, prognosis and treatment, the competence of medical personnel and continuity of care. In the case of thyroid disease where there is a high degree of epistemological uncertainty with regards to pathological, symptomatic and diagnostic categories and practices such existential uncertainty is amplified. As a

consequence, as the presentation of data throughout this chapter has demonstrated, it is the high degree of existential and epistemological uncertainty that characterises the manifestation of thyroid disease is within the context of the contemporary NHS.

Moreover such epistemological existential and clinical uncertainty is not merely of interest because it demonstrates a disconnection between specialist knowledge of disease, embodied experiences and clinical practice. It is also significant because it indexes factors that are beyond the specific location of the thyroid clinic and the experience of being a thyroid patient. For example, I often interpreted the Consultant's retreat into an overtly mechanical mode of delivering treatment as a response to the way in which patients perceived his specialist knowledge and professional role, as well as their own status as sufferers. When a patient entered the clinic asking for particular diagnostic techniques or treatments for his or her condition that they had read about on the internet the Consultant's face would often glaze over. This response could be attributed to the patients asking for treatments and medications that were simply not available through the NHS, but also, perhaps, to his feeling that if that is what they really wanted there was 'no point in him being there'. I.e. if they wanted to indulge in treatments for which, in his opinion, there was no sound evidential base, such as dietary measures or herbal remedies, what was the point in him taking the time to treat them otherwise? Whilst this opinion could be negatively construed as a medical professional protecting his bruised ego, it was also apparent that this view was embedded in the pressures on NHS resource allocation and a prevailing ethos of professional medical care. From the point of view of resource allocation, time and efficiency are at a premium, leaving little time to discuss the many theories about the diagnosis and treatment generated by a Google search on the Internet. There is also the associated quandary of the extent to which clinicians should listen to patients and take their views and opinions on board, but also, how far to 'go along with' patients' attempts at self-diagnosis and treatment derived from often unreliable and conflicting Internet sources.

Often the Consultant's sighs of disbelief at the demands of the patients were expressed in the brief periods between patients during the tightly packed appointment schedules that were a routine feature of the over

stretched and under staffed clinic, where there was simply no time to discuss unsubstantiated and biomedically speculative theories. In addition, some patients seemed to reproach the Consultant in response to his frosty reception of their preferences for treatment, assuming that he was behaving in this way because he was too narrow-minded in the methods he employed in treatment of thyroid disorders. This clash of professional and lay biomedical models would often mask another key reason for the Consultant's dogmatism with regards to the treatment algorithm. He didn't entertain some treatments because he believed, and in some cases had seen, how these other methods could make 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 administration of thyroid replacement therapy, by either themselves or other clinicians¹⁶.

As I have suggested, the conflicts that are manifest within the experience, diagnosis and treatment of thyroid conditions, in particular the existential uncertainty wrought by current explanatory models of thyroid biology and pathology, bring into relief broader practical issues with regards to current health care policy within the U.K. The Patient Choice Initiative that encourages patients to become informed about their health and healthcare and the Evidence Based Medicine (EBM) movement that believes that clinical decisions and interventions should primarily be based on the best available published scientific literature, are both implicated in the conflicts over the diagnosis and treatment of thyroid disease. On the one hand, within the context of the NHS there is the aim to encourage patients or 'health care users' to become more interested and involved in the management of their disease. However, the biochemical model of thyroid function that is currently used to diagnose thyroid disease, often does not represent how patients embody and experience *their* thyroid disease though clinical symptoms. Therefore, on the other hand, evidence based

¹⁶ There were at least five occasions observed in the clinic where patients had been referred to the consultant because they had become hyperthyroid due to the over-prescription of thyroid replacement. Often these cases of iatrogenic hyperthyroidism were a result of private clinicians, who questioned the accuracy of TFTs, prescribing on the basis of the alleviation of symptoms, hence sometimes increasing thyroid function into the realm of hyperthyroidism. Sometimes patients actively sought to have this over prescription corrected through their GP and the Consultant as they felt unwell. However on other occasions these patients were sent to the Consultant by GPs so he could convince them to stop taking so much thyroid replacement due to threat of various long term health risks even though they found hyperthyroidism a desirable state due to the energy it gave them and weight loss it resulted in.

diagnostic and treatment strategies, embodied by TFTs, mean that such patients are often alienated from the clinical care they receive and thus cannot overcome the existential uncertainty the state of thyroid related illness brings with it.

As Rose and others have claimed, EMB and patient centred medicine are both contemporary neo-liberal technologies of governance that facilitate devolution of the management of human health from the state to, amongst other things, various professional groups and individual citizens through the process of responsabilization. In the case of thyroid disease these two governmental technologies clash, and ironically fail to govern the health of the thyroid population. That is, the technical deficit of current technologies of thyroid disease result in patients rejecting this standardised evidence based approach and seeking alternative expertise and treatments from outside of the medical establishment in order to overcome ill health. Therefore, although such patients are engaging with the improvement of their health and well-being they are doing so because of the inadequacy of health technologies.

The following chapters will discuss this conflict in more detail, exploring the disconnection between the types of evidence used to diagnose and guide treatment for thyroid disease and how they are understood and handled by various groups of patients and clinicians. Specifically, the remaining chapters of the thesis will focus on how the evidence of thyroid disease is negotiated and coordinated to various orders of thyroid biology, all of which are a response to the wants, needs, rights and responsibilities of these various groups. In particular, the thesis will focus on how the disconnection between symptom based and biochemical based orders of thyroid biology and pathology identify and provide an interesting addition to the concept of biosociality. Specifically, the ways in which the remaking of thyroid biology through practice is based, not on biomedicine's ability to control and engineer thyroid pathology, but on its inability to fully grasp and control this non-compliant and ambiguous pathology.

Chapter 5

Circulations of evidence: defining the boundaries of thyroid disease

In the previous chapter I suggested that once thyroid disease is tracked experientially – be it by a patient or a clinician -- it can no longer be understood as a straightforward set of diagnostic categories and procedures. In particular, as I observed in the clinic, the biochemical reference ranges used to ‘definitively’ diagnose thyroid dysfunction often do not translate into the symptoms that individual patients report. I further argued that the very means of attempting to reconcile a diverse and often indeterminate array of symptoms against a range of standardised test result calibrations can itself become the cause of greater suffering. In this chapter I examine the disparity between the types of ‘evidence’ used to diagnose and treat thyroid disease, and the further ramifications of this discrepancy. I conclude by suggesting some of the implications this might be seen to have for existing social scientific models of embodiment, medicalisation, somatic ethics, care of the self’ and in particular biosociality.

In this chapter, I begin with a further examination of the difficulty in attributing symptoms to an underlying pathological cause situated within the thyroid gland and the inadequacy of the thyroid function tests introduced in an attempt to resolve this issue. This is followed by an account of the uncertainty of both symptomatic and biochemical evidence used in the diagnosis of thyroid disease, emphasising the ways in which both clinicians and patients rely upon the indeterminate aspects of these sources to construct discrepant versions, or ‘orders’, of thyroid biology that correspondingly fulfil an array of rights, responsibilities, desires, and consequent management and treatment strategies. I conclude by suggesting that thyroid diseases provide a highly relevant context in which to further evaluate the analytical purchase of Rabinow’s concept of biosociality. Specifically, I examine how the community that collects around thyroid pathologies is bound together by an array of often contradictory techniques and practices that attempt to take control of the *lack of technique* that most prominently characterises current methods used to diagnose, treat and manage thyroid disease. In other words, I try to suggest in this chapter that, in the case of thyroid disease, it is the lack of control

over thyroid pathology that results in the development of various techniques that remake its biology through cultural practices. I further demonstrate that it is this lack of technique and control which generates much of the solidarity – again among both clinical and patient communities – that might be described as ‘biosocial’ in its focus on a specific disease.

The problem of symptoms

According to the medical text book DeGowin's Diagnostic Examination (2004) the word symptom:

is derived from the Greek word meaning ‘something that has befallen one.’ A symptom is usually considered to mean abnormal sensation that is perceived by the patient, in contrast to a sign that can be seen, felt, or heard by the examiner. (DeGowin et al. 2004: 25)

As DeGowan et al point out, a symptom is a subjective indication of disease provided by the patient. A sign, on the other hand, is an abnormality that is observed by a doctor during a clinical examination. The distinction between signs and symptoms is not always clear. When a doctor observes a symptom reported by a patient, the symptom is also a sign. Some symptoms, however, cannot be seen by a doctor and therefore cannot also be classed as signs. For example, a goitre is both a sign and a symptom of thyroid disease as it can be observed by both patients and doctors. In contrast, sore muscles and increased sensitivity to the cold, symptoms of hypothyroidism, can only be classed as symptoms because they can only be felt by the patient and not observed by the doctor. On the other hand, the results of thyroid function tests can only be classed as a sign because although a patient may think they have a thyroid problem through the experience of symptoms, they cannot observe this dysfunction until it is revealed through blood test results provided by hospital laboratories. Therefore, the results of thyroid function blood tests are considered to be superior evidence of thyroid disease because they are signs of disease that can be observed by the doctor independently of the subjective accounts of individual patients. Moreover, they are able to attribute illness to an underlying pathological cause, for instance the under or over production of thyroid hormones by the gland. Consequently, whilst symptoms can be useful indicators of disease, they are also considered to

be too subjective and too general and therefore can complicate and mislead the process of diagnosis. For example, as De Gowan et al also note, when a patient history is taken 'the patient's description of symptoms must be clarified and quantified' (DeGowin et al. 2004: 25). The symptoms can then be transformed into clearly defined clues that will facilitate a robust and thorough diagnostic decision:

The chief purpose of the history is to furnish clues for diagnosis. As the narrative unfolds, you should be simultaneously performing three operations (a) the accumulation of facts (obtaining the history), (b) evaluation of the facts (testing credibility of symptoms, seeking more details of time and quantity, and (c) preparation of hypotheses. Having formed a list of hypotheses, question the patient about other symptoms specific for diseases on the list, either to support or to discard the hypothesis (DeGowin et al. 2004: 25)

Therefore, within the established diagnostic protocols of western medicine, symptoms are approached with caution as they are considered to be inherently subjective and could undermine the integrity of objective and well hypothesised clinical reasoning. As Foucault demonstrates in *The Birth of the Clinic* (1973), this organisation or 'spatialisation' of symptoms, signs and pathology is key to understanding the discourse of modern medicine that emerged during the nineteenth century:

The space of configuration of the disease and the space and localization of illness in the body have been superimposed, in medical experience, for only a relatively short space of time – the period that coincides with the nineteenth century medicine and the privileges accorded to pathological anatomy. This period that marks the suzerainty of the gaze, since in the same perceptual field, following the same continuities or the same breaks, experience reads at a glance the visible lesions of the organism and the coherence of pathological forms; the illness is articulated exactly on the body, and its logical distribution is carried out at once in terms of anatomical masses. The 'glance' has simply to exercise its right of origin over truth. (Foucault and Sheridan 1973: 3)

According to Foucault, during the eighteenth century symptoms were treated as the illness. For example, stomach ache was considered to be the illness itself, rather than the effect of one. This two dimensional model of illness was replaced during the nineteenth century with the emergence of the clinic and of hospital medicine, where symptoms, signs and pathology became the framework through which disease was understood. In this 'three dimensional' framework, the symptoms presented by patients were

evaluated alongside signs that were observed by skilled clinicians, both of which indicated an underlying pathological lesion that was the disease. Consequently, the clinical examination became a process in which the body of the patient was mapped out in order to find the exact location and form of the disease-causing lesion. For example, in 1873 the first description of hypothyroidism was published by the physician Sir William Gull (1873-1874). Through a close examination of a female patient referred to as 'Miss B', Gull's account carefully recorded a number of clinical signs and symptoms such as the size of the tongue, the colour and texture of the skin and the behaviour of the patient. These clinical signs and symptoms were then placed alongside pathology reports that indicated a wasting, and even the absence, of the gland. Thus, hypothyroidism – a pathological condition of the thyroid gland – was revealed.

Therefore the triangulation of signs, symptoms and pathology in the specialist space of the clinic meant that illness was no longer seen primarily through the subjective realm of symptoms but through the clinical expertise (or what Foucault terms 'the gaze' (1973: 45) of the doctor. Throughout the nineteenth and twentieth century, the gaze of the medical profession and the subjugation of patient symptoms developed further through the expansion of an array of technologies (x-rays, blood tests, cytological tests etc.) designed to observe pathology in increasing detail (Armstrong 1995). For example, in the 1970's thyroid function tests were developed that could measure the negative feedback loop between the thyroid and pituitary glands as well as the level of hormone production of the thyroid gland. This meant that the pathological process that caused thyroid disease, as well as its results (under or over production of thyroid hormones) became available to the clinical gaze. As a consequence, the symptoms of thyroid disease are perceived to be un-reliable diagnostic evidence - unlike blood tests - because they can only *suggest* and not prove the presence of disease within the thyroid gland. However, as will become clear throughout this chapter, far from creating a better and more precise definition of thyroid disease such tests have resulted in increasing levels of uncertainty and contestation. In fact, the process of 'tidying up' thyroid disease into clearly defined biochemical reference ranges has meant that many patients feel that their symptoms and experiences are ignored.

Disciplining evidence

As the *UK Guidelines for the Use of Thyroid Function Tests* claim, 'Hypothyroidism is an insidious condition with significant morbidity and the subtle and non-specific symptoms and signs may be mistakenly attributed to other illnesses, particularly in post-partum women and the elderly' (Association for Clinical Biochemistry et al. 2005: 11). This suggests, the symptoms of thyroid disease are problematic because firstly they are incredibly general and can be indicative of a plethora of other diseases. Secondly, they are highly subjective and therefore difficult to attribute to an underlying dysfunction in the gland. For example, in the case of hypothyroidism the symptoms of low mood and lethargy could also indicate mild depression and the 'symptom' of weight gain could be a result of eating too much. In order to overcome the problem of the uncertain aspects of symptoms, a thyroid function test has been developed to definitively and objectively identify pathology within the gland. As described in chapter four, this blood test measures the negative feedback loop between the pituitary and thyroid glands, as well as measuring the amount of thyroid hormones that the thyroid gland produces. Thyroid function blood tests, then, are used to differentiate whether 'thyroid type symptoms' are in fact a result of thyroid dysfunction or some other cause.

For example, in the clinical text book *Thyroid Disease: The Facts* written by consultant endocrinologists R.I.S Bayliss and W.M.G.Tunbridge, continuing depression in patients whose blood test results demonstrate that their thyroid is healthy is explained as follows:

Persistent depression after correction of thyroid deficiency is quite common and is a cause for patients being dissatisfied with their response to thyroxin treatment. It is, of course, essential that the correct replacement is being given and most patients with thyroid deficiency feel at their best when the dosage of thyroxin raises their free thyroxin level towards the upper end of the normal range, or even a little above it. If you have received for several months the amount of thyroxin that meets these criteria but still continue to feel generally unwell, it is likely that your ill health is due to a persistent degree of clinical depression, which merits treatment in its own right. Fortunately there are many psychotropic drugs, with few side effects, that will correct this situation. (Bayliss and Tunbridge 1998a: 164)

As Bayliss and Tunbridge explain, it is common for patients who have had their thyroid function returned to a level within the accepted reference range to complain of persistent symptoms. However, without the 'definitive' proof of thyroid dysfunction, provided by blood tests, such lingering symptoms are attributed to other possible causes such as depression. In spite of this, many patients refuse to accept that their thyroid disease has been treated and that their symptoms have been eradicated. As Alex, a hypothyroid woman in her mid-thirties recounted in an interview at her home in London, the level of replacement thyroxin she had been prescribed on the basis of thyroid function tests did not return her to an acceptable level of health. When describing why her dosage 'isn't right', Alex expresses a view common among the patients interviewed that TFT results are misleading and lead to incorrect dosages that fail to alleviate symptoms:

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 they say their hair only falls out when their tablets aren't right, and they have been taking their tablets for years so erm...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...

Although Alex acknowledges that thinning hair takes time to correct itself, even after thyroid replacements are prescribed, she still feels that there is an underlying problem with her thyroid function and supports her view by relying on the authority of more experienced patients. Therefore, as demonstrated by Alex and claimed by a number of patient groups (e.g. Thyroid U.K, Thyroid Patient Advocacy, Stop The Thyroid Madness!!!), the process of diagnosing thyroid disease primarily on the basis of biochemical assays seems to ignore symptoms of disease experienced by patients after biochemical evidence suggests that normal thyroid function has been restored. As claimed by the patient support-group Stop the Thyroid Madness!!!, the idea that there is a rigid 'normal range' of thyroid function is erroneous and dubious to begin with.

...we are HELD HOSTAGE to a lab [test] called the TSH (thyroid stimulating hormone) with its erroneous and dubious

'normal' range. Patients note that when in that 'normal' range, they continue to have symptoms.(Stop the Thyroid Madness!! 2006)

This tension between the embodied evidence presented by patients and the biochemical evidence preferred by current best practice guidelines returns us to a broader conflict within western healthcare, namely that between Evidence Based Medicine (EBM) and the patient-centred medicine, as discussed in chapter two. The medical sociologist David Armstrong (2007: 77) notes that proponents of the patient-centred model believe that 'EBM denies the individuality of the patient and their particular therapeutic needs' (2007: 77). Dr G.R.B Skinner, a practicing clinician who endorses the treatment of biochemically euthyroid ('healthy') but clinically hypothyroid individuals, supports this contention:

[I]t is increasingly unusual for family practitioners, physicians and endocrinologists to diagnose and treat hypothyroidism in clinically hypothyroid patients unless free thyroxin (FT4) and/or thyroid stimulating hormone (TSH) falls outside of the 95% reference intervals. This arises from the current misconception that evidence based medicine means laboratory-based medicine wherein clinical observation-albeit of an objective nature, for example pulse rate or blood pressure –is accorded a lower evidential weight than laboratory measurements[...](Skinner et al. 2000: 115)

According to Skinner, the 'increasingly unusual' practice of diagnosing thyroid disease on the basis of clinical signs and symptoms demonstrates that EBM erodes the individual needs and experiences of patients. The reference ranges that Skinner refers to, as described in chapter four of this thesis, are the range of thyroid function values (TSH free T4) found in the mid 95% of a reference population. It is claimed by Skinner and other like-minded colleagues that such population-based reference ranges are misleading because they frequently do not account for those individual cases of thyroid disease that do not fit into such biochemical population-derived diagnostic categories. For instance, some patients may have overt hypothyroidism in spite of blood tests that suggest that their thyroid function is normal. Consequently, Skinner claims that the practice of extrapolating these reference ranges to account for an individual's thyroid function is abstract, intangible and unrealistic. This approach to thyroid disease therefore considers symptoms to be a more reliable indicator of thyroid

disease because they can be experienced by patients and observed by clinicians through clinical examination.

The criticism that clinicians such as Skinner level at thyroid function tests also reveals another aspect of the EBM debate, in which it is claimed that clinical reasoning primarily based on scientific method erodes the skill and authority of doctors. As Dr Barry Durrant-Peatfield, a retired GP and supporter of diagnosing disease on the basis of clinical symptoms, declares:

We find ourselves in the grip of a medico-technocracy. The blood test is God, and eventually takes over the diagnostic process. I deeply deplore this state of affairs, since I was taught and have never had any reason to doubt, that the best diagnostic weapons of all are the Mark I eyeball and the Mark I earhole. Look and listen. The other excellent lesson I learnt was that if the blood test doesn't bear out your diagnosis, believe in your own learning and experience (Durrant-Peatfield 2006: 70).

However, in terms of the tenets of EBM the extreme form of patient-centred medicine and experienced-based clinical reasoning, favoured by Skinner and Durrant-Peatfield, exemplifies and encourages 'ineffective and inefficient practice (Armstrong 2007: 77). For example, Skinner's belief that symptoms and not blood test results should be the target of treatment rejects the triad of symptoms, signs and pathology which, as discussed earlier, defines modern medicine. Hence, such approaches are considered by 'establishment' endocrinologists to be ineffective and inefficient for two reasons: firstly they are treating symptoms that may not be the result of thyroid disease; secondly they are prescribing medication and providing annual tests to monitor thyroid dysfunction in individuals who may not even have thyroid disease. This in turn, it is claimed by proponents of EBM, has a negative effect on the distribution of health service resources. For example, the canon of established thyroid literature places the incidence of hypothyroidism, (the number of people who fall under the biochemical reference range of thyroid function) at 1 to 2% of the general population (Flynn et al. 2004). However, according to Durrant-Peatfield, symptoms should also count as definitive evidence of disease, and the incidence of hypothyroidism in the population is therefore estimated at 30% (Durrant-Peatfield 2006: 125). Therefore treating this 30% is considered to be ineffective by supporters of blood tests because it is done on the basis of a clinical reasoning that is unable to provide a causal link between disease of

the thyroid gland and the onset of non-specific symptoms. Furthermore, such a strategy would also be inefficient because it would mean that people would be prescribed thyroid replacement therapy on the basis of clinical anecdote and not scientific facts. Consequently, this issue of clinical decision making is also related to the efficient distribution of NHS resources through which thyroid replacement therapy is prescribed to individuals where efficacy can be definitively demonstrated.

This disagreement within the medical profession with regard to how a diagnosis of thyroid disease is reached is also compounded by the campaigns of a number of patient activist groups who are highly critical of treating thyroid disease primarily on the basis of blood test results. As discussed in Chapter three of this thesis, Professor Anthony Weetman's 2006 article *Whose Thyroid Hormone Replacement is it Anyway?* (2006) suggests that changing attitudes towards the medical profession and scientific knowledge are the cause of the dissatisfaction with treatment for hypothyroidism. When discussing the reasons why some patients demand treatment for their thyroid disease in spite of blood test results that fall inside of or on the borders of the reference range, Weetman claims it to be a result of the subversion of scientific knowledge in the contemporary era. The derogation of objective facts in contemporary Western culture, he complains, corrodes conventional hierarchies of knowledge and any single sense of what is 'real'. Moreover, this corrosion of medical authority means that factors outside of the realm of biomedicine are currently guiding how patients make sense of and interact with their health, for example in terms of lifestyle and consumer choices. As Weetman claims:

[T]he rise of 'healthism'¹⁷... has been characterized by the following features: high health awareness and expectations, information seeking, self-reflection, distrust of doctors and scientists, healthy and often alternative lifestyle choices, and a tendency to explain illness in terms of folk models of invisible germ-like agents and malevolent science (2006: 232)

Therefore, according to Weetman, disagreement amongst the medical profession coupled with the rise of 'healthism', results in the corrosion of a single, scientifically rigorous definition of thyroid disease - specifically, the

¹⁷ The term healthism was first coined by the political economist Peter Crawford in his article *Healthism and the medicalization of everyday life* (1980) Healthism describes the shift in the responsibility of health from the state to the individual in late capitalist societies through practices of consumption.

claim that thyroid disease can be diagnosed on the basis of clinical symptoms alone.

In 2005, the *UK Guidelines for the Use of Thyroid Function Tests* were developed in an attempt to solve the confusion and disagreement between clinicians and patients with regard to the interpretation of thyroid function tests. The purpose of the guidelines is outlined as follows:

It is hoped that the document will provide guidance for primary care physicians, specialist physicians, endocrinologists, and clinical biochemists. The accompanying patient information sets have been especially designed to explain thyroid function testing and to summarise the main recommendations in the guidelines in everyday language. The purpose of the guidelines is to encourage a greater understanding of thyroid function testing amongst all stakeholders with a view to the widespread adoption of harmonised good practice in the diagnosis and management of patients with thyroid disorders. The guidelines are also intended to provide a basis for local and national audit and each section offers recommendations that are suitable for the audit process. (Association for Clinical Biochemistry et al. 2005: 5)

As briefly described at the end of Chapter two of this thesis, these guidelines were produced by a development group that consisted of endocrinologists, biochemists and patients, who were represented by Janice Hickey and Betty Nevens, the Director and Office Manager of the British Thyroid Foundation (BFT), respectively¹⁸. The group met on a number of occasions, and subgroups were created to take responsibility for chapters related to particular themes: hyperthyroidism, hypothyroidism, thyroid function and pregnancy, thyroid function testing in thyroid cancer and laboratory aspects of thyroid function testing. The whole group then considered each draft chapter. The guidelines do not question the validity of TFTs, rather the aim was to encourage a greater understanding of TFTs and their widespread adoption in order to develop 'harmonised good practice' (Association for Clinical Biochemistry et al. 2005: 5). This objective entailed the eventual development of a process of local and national audits

¹⁸ BFT is associated with the British Thyroid Association (BTA) a professional organisation of thyroid clinicians and scientists within the U.K. Both of these organisations agree that treatment for thyroid disease should primarily be based on thyroid biochemistry. This relationship and shared approach to diagnosing and treating thyroid disease is made explicit on the website of both of these organisations. Moreover, the BTA and BFT websites both warn about the danger of other websites that provide misleading and information that is not endorsed by 'medical research'.

intended to monitor the uptake and effectiveness of the guidelines. Hence, the assumption that the biochemical evidence provided by TFTs is superior to clinical signs and symptoms is a priori within these guidelines.

Consequently, it is unsurprising that Dr Gordon R.B. Skinner made the following response to the first draft of the guidelines on the Thyroid Patient Advocacy Groups¹⁹ website:

One of the difficulties with Guidelines in modern day medicine is that they cannot be divorced from political and regulatory influences...[It is thus crucial that the medical profession acknowledge and state in the public domain that Guidelines are there to assist practitioners but are not sticks to beat them if they fall out of line. (Thyroid Patient Advocacy UK 2005)

In this statement, Skinner is rehearsing the argument that evidence-based guidelines are not simply apolitical tools that ensure better and safer practice. He is claiming that they are tools used to regulate and rationalise medical knowledge through the collation of heavily selected trial data. Specifically, he is inferring that the production of guidelines for the use of thyroid function tests is a scheme by the established community of thyroid professionals to regulate the practice of individual clinicians such as himself. As Skinner goes on to claim:

[...]if a patient has high TSH level or a low FT4 level, then that patient is likely to be unwell and suffering from the clinical features of hypothyroidism. The mischief lies in the assumption of the reciprocal, and I am unaware of any evidence which teaches us that if you have clinical features of hypothyroidism with reasonably normal thyroid chemistry, the patient does not suffer from hypothyroidism and will not benefit from thyroid replacement (Thyroid Patient Advocacy UK 2005)

Therefore, Skinner concludes in his response that he disagrees with the guidelines because he believes, in his experience, that the exclusion of hypothyroidism through blood test results that fall inside the reference range 'results in patients being chronically under treated'. Interestingly, Skinner's solution to this predicament is the development of a 'properly

¹⁹ The Thyroid Patient Advocacy Group UK (TPAUK) is a patient support group based in the United Kingdom that believes that the current diagnosis and treatment practice(s) espoused by the established medical profession are inaccurate and "ignores and/or dismisses relevant physiology that has been known to medical science for nearly 40 years, ignores medical ethics, and ignores the human rights of patients". This claim will be addressed in greater detail later when the concept of 'orders of thyroid biology' is introduced.

controlled clinical trial' that can investigate the efficacy of treatment in euthyroid but symptomatic individuals. Consequently, it is clear that Skinner and other like-minded clinicians believe that adjusting the primacy of biochemical evidence in relation to clinical signs and symptoms will aid in the identification of thyroid disease in biochemically euthyroid but symptomatic individuals. Moreover, by suggesting that clinical trials should be conducted to investigate this claim, they are suggesting that it is possible to prove that symptoms themselves can be scientifically rigorous evidence of thyroid disease. That is, through a clinical trial it is possible to quantify the efficacy of treating patients on the basis of symptoms alone.

In sum, the deployment of standardised biochemical tests as definitive markers of disease is one measure that could offset the expensive consequences of 'anecdotal thyroid disease' and its potential drain on busy clinicians, nursing staff and the NHS. As a result, the practice of administering thyroxin exclusively to individuals who are biochemically hypothyroid, is construed by some clinicians and patient groups to be an ideologically conservative response based on a traditional 'faith' in biochemical evidence. For example as the patient group Stop the Thyroid Madness!! advise patients on their website:

you have to now make a paradigm shift in the way society and doctors have taught you to ascertain whether you are hypothyroid or not, or whether you are adequately treated or not. Namely, you have to make SYMPTOMS your primary clue, NOT lab work like the TSH. Lab work should only serve as additional information, NOT as the initial force of reality. If you continue to look at lab work as THE answer, you are no better than hundreds of thousands of doctors around the world who have kept thyroid patients SICK! (Stop the Thyroid Madness!! 2006)

At first glance, the claims of clinicians such as Skinner and patient support groups such as Stop the Thyroid Madness!!! appear to suggest an example of the misguided confusion presented by Professor Anthony Weetman (as described in chapter 3). However, there is a growing body of literature that demonstrates that the relationship between thyroid biochemistry and clinical signs and symptoms remains indeterminate.

In 2007, the Cochrane Collaboration ²⁰ produced a systematic review entitled *Thyroid replacement for subclinical hypothyroidism* (Villar et al. 2007). The purpose of the review was to assess the effects of thyroid hormone replacement on subclinical hypothyroidism, and therefore assess the effectiveness of prescribing thyroid replacement to patients whose thyroid biochemistry is on the borders of the reference and who, according to the established thyroid literature, should be asymptomatic. The overall conclusion of the review was that replacement therapy for subclinical hypothyroidism did not result in decreased cardiovascular morbidity²¹ or a significant improvement of health-related quality of life and symptoms between intervention groups. Moreover, there was some evidence that indicated that thyroid replacement treatment improves some parameters of lipid profiles and left ventricular function (the health of the lower chamber of the heart that can be impaired by high cholesterol). However, the Cochrane Collaboration review noted that, although the results of the published data suggested that treating subclinical patients was not definitively efficacious, there are a number of studies that demonstrate some benefits as well as disadvantages to this course of treatment. As the review summarises:

Thyroid substitution could prevent progression to overt hypothyroidism, improve neuropsychiatry symptoms and somatic symptoms, mood disorders, cognitive dysfunction, atypical responses to standard psychiatric therapeutic interventions, and minimize deleterious effects on cardiovascular function and lipid levels. However, there may also be adverse effects from replacement doses of thyroxin, these include nervousness, palpitations arterial fibrillation, and exacerbation of angina pectoris. Reduction in bone density of the spine, femoral neck, distal radius, and femoral trochanter can also occur. The over-treatment with thyroxin probably contributes to the development of osteoporosis in post-menopausal women, and an undetectable TSH level suggests over treatment. (Villar et al. 2007: 6)

Furthermore, as this review concludes, until better evidence is available, decisions to treat subclinical hypothyroidism should be based on 'clinical judgement and patient preferences.' (Villar et al. 2007: 12). Consequently, even with access to the most reliable and exhaustive evidence available, the authors of the Cochrane Collaboration systematic review struggle to

²⁰ Founded in 1993 and based on the vision of Archie Cochrane (1973) the father of EBM, the Cochrane Collaboration review and publish the effects of health care interventions tested in biomedical randomized controlled trials.

²¹ One of the symptoms of hypothyroidism is high cholesterol. It is thought that this may occur and result in morbidity even for individual who are mildly hypothyroid.

negotiate the competing values embedded in the biochemical and symptomatic evidence used to diagnose and treat thyroid disease. To be precise, they are unable to definitively prove that there is no benefit to treating patients whose biochemistry suggests mild thyroid failure and whose symptoms may have no sound physiological basis.

To summarise the previous section, then, it is clear that there is a tension between the blood test results and the clinically-observed symptoms that constitute evidence in the diagnosis and treatment of thyroid disease. In my analysis I point to a growing body of literature that suggests this tension is itself symptomatic of the shifting ethical, political and economic values and priorities guiding health service provision – and is in some ways epitomised by the conflict between evidence-based and patient-centred models of health care. The categorisation of thyroid disease in terms of population based biochemical reference ranges is an example of how clinical practice is becoming increasingly infused with the scientific values of EBM. TFTs offer the possibility of disciplining the non-specific symptoms of thyroid disease within a scientific, population-based and biochemically-defined disease management infrastructure, thus also appealing to the post-audit culture values of transparency, efficiency and accountability (Power 1999). However, as will be detailed further later in this chapter, attempts to reduce the indeterminacy of thyroid disease through hyper-rationalised diagnostic strategies has many disadvantages. As the proponents of patient-centred approaches to health insist, TFTs, EBM and the like require the suppression of individual patient's evidence and experience based on their everyday lives. Clinicians are also critical of the EBM paradigm, claiming it undermines their professional expertise and relies on a reductive model of illness that may even exacerbate existing pathology. Some patients and clinicians suggest EBM is itself the source of iatrogenic illness – a claim for which, ironically, an increasing amount of statistical evidence can be mobilised to support.

Consequently, this study suggests that because both the biochemical and symptomatic evidence of thyroid disease are highly indeterminate, neither can be used to discipline thyroid disease. It is for this reason that the very 'vagueness' of thyroid disease may make it a powerful model of both medicalisation and management in the context of shifting expectations and knowledges concerning health, health care, and the self-management of

patients. In other words, it is because thyroid disease provides a weak context for medicalisation that it becomes valuable as a contested site in which the boundaries of disease can be seen to belong to changing and conflicting orders of biology, medicine and science presented by both patients and clinicians. In the following section, the negotiation of these actively contested dimensions of thyroid disease will be described in terms of how they are used to discuss and justify various diagnostic and treatment strategies.

Opinionated evidence

As described above, the indeterminacy of the evidence for thyroid disease presents it as weak context for medicalisation. That is, when considered in light of a discussion of the problem of the evidence sources used to diagnose thyroid disease, it is impossible for the medical profession to discipline it into a biochemically circumscribed disease category. Consequently, physicians who practice outside of established theories of thyroid disease, and patients who are dissatisfied with their treatment, are able to use the gaps in the evidence to negotiate an order of thyroid disease that satisfies their various rights, want, needs, desires and responsibilities. Therefore, such negotiations can be described as what Margaret Lock would call a 'local biologies,' of thyroid disease - the ways in which physical sensations (well being, health, and illness) are in part informed by the material body, which is itself contingent on evolutionary, environmental and individual variables. Subsequently, the 'local biology' of thyroid disease consists of the construction of various orders of thyroid disease and biology that attempt to explain physical sensations through the shifting ethical, political and economic values and priorities guiding health service provision within the NHS (epitomised through the evidence based and patient centred models of healthcare). Specifically, this 'local biology' sees the construction of particular orders of thyroid biology and pathology that attempt to fill the explanatory gaps left by the established model of the thyroid pituitary axis and the unreliability of symptomatic evidence.

If we compare two interpretations of thyroid symptoms, it is apparent that their ambiguity can be mobilised to construct different accounts of the

disease. Samantha is a patient in her early thirties with Grave's disease. She describes the onset of her disease as follows:

So I was writing up my PhD so this would be, a long time...err lets see that was 1998 to 2005 when I started this one, so probably about 2002/2003. And what was happening confusingly at that point was that I was feeling quite, erm low in mood I think really was the kind of thing that was going on and really very tired, and yeah I think increasingly putting on weight and things like that, erm, and I was kind of because of my psychology background I was kind of interrogating myself saying could this, could I just be depressed is that what's going on here, and I thought not really it, there isn't any kind of cognitive cause for it and it's not that I feel kind of despairing or self blaming or anything I just feel kind of exhausted and a bit stuck and not moving forward and not energetic...

If you contrast this account to a list of hypothyroid symptoms listed by Diana Holmes in her book *Tears Behind Closed Doors-Failure to diagnose an underactive thyroid: the truth behind the tragedy* (Holmes 2002), the symptoms are interpreted very differently. In fact, the non-specificity of symptoms is increased and the category of the 'symptom' is expanded to incorporate additional entities.

The patient is overweight and puffy or thin and worn out**	Dry Mouth and sore throat*
Goitre	Swallowing difficulties
Exhaustion	Slow Speech
Lethargy	Slow thinking
Dizziness	Loss of appetite
Weight gain	High blood pressure**
Swollen Ankles*	Allergies*
Puffy Face and Hands	Sensitive to the sun*
Thick tongue*	Loss of balance
Brittle nails	Pains over the heart and crushing feelings in the chest*
Dry Hair	Poor memory and concentration
Hair loss over parts of the body*	Morbid thoughts
Thinning eyebrows	Insomnia**
Feeling cold	Dreams*
Overheating**	Visual disturbances*
Headaches*	Nervous and jumpy**
Migraines*	Anger and irritability**

Aching limbs and joints, also cramps Lower back pain Carpel Tunnel Syndrome* Palpitations** Deafness* Pallor Constipation Diarrhoea** Swollen stomach* Menstrual problems Shaky Hands, Shaky body, Juddering in the spine** Pins and Needles* Numbness* Decreased Sweating* Hoarse Voice	Panic attacks** Anxiety** Hallucinations Voices in the head* Guilt* Suspicious of people's motives* Persecution complex* Cry or get upset easily* Lack of confidence* Claustrophobia** Agoraphobia** Mood swings* Depression Low libido Heavy eyelids
General problems including loss of balance, florid complexion (florid hypothyroidism), fear of what is happening to your body, clenching teeth, being an 'emotional wreck'.*	
*Not in the BFT list of symptoms **Also a symptom of hyperthyroidism	

Contrasting Samantha and Diana Holmes' descriptions of symptoms reveal two issues of note. Firstly, both of the accounts describe physical and psychological sensations that are borderline symptoms, sensations that could also be interpreted as 'normal' feelings that are not necessarily the result of disease. Secondly, in spite of the fact that Samantha and Diana describe a number of the same symptoms, they are in fact talking about different diseases of the thyroid. Samantha has been diagnosed with Grave's disease and Holmes is referring to hypothyroidism. These accounts demonstrate not only the indeterminacy of the symptoms of diseases of the thyroid, but also the way in which these symptoms form the basis for a particular construction of the disease. For example, Holmes' position as patient activist impacts not only on how symptoms are represented, but also on which symptoms are attributable to thyroid disease. Some of the symptoms of thyroid disease can be understood as

responses to the politics of thyroid disease as well as forming interpretations of physical and psychological sensations. As Holmes' description of 'guilt' demonstrates:

I felt so guilty about being unwell and at one time doubted my own perceptible abilities. I found myself on the defensive most of the time and not liking myself at all. My self esteem was at its lowest ebb (Holmes 2002: 58).

It is difficult to discern whether this sense of guilt is a symptom or a personal reflection on the state of having a disease. Are these 'symptoms' of guilt and doubt a result of being placed in the category of 'a problem, time-wasting patient' who ignores professional expertise and contests their disease on the grounds of unsubstantiated biomedical theories? Is it a symptom of a social category rather than a pathological one? How can we think about the relationship between physical sensations and personal feelings and experiences?

The relationship between different forms of evidence of disease is perhaps best described by the philosopher Ian Hacking's concepts of 'making up people', (Hacking 1986) and 'looping effects' (Hacking 1995), which he describes as follows:

... 'Making up people' referred to the ways in which a new scientific classification may bring into being a new kind of person, conceived of and experienced as a way to be a person. The second, the 'looping effect', referred to the way in which a classification may interact with the people classified (Hacking 2002: 285)

If we consider thyroid disease in these terms, the scientific classification of particular levels of thyroid function (hypothyroid, subclinical hypothyroid, euthyroid, subclinical hyperthyroid and hyperthyroid) that are used to order the indeterminate phenomenological experience of thyroid disease, calls afflicted individuals into a specific type of being. These classifications that have 'made up people', are in turn informed by the people being classified. For instance, the classification of thyroid dysfunction on the basis of the discipline of clinical biochemistry means that subclinical patients who complain of symptoms do not fit into this established diagnostic system. As a result, new categories are established or become related to the phenomena, such as the somatoform disorder, described by Weetman as over-informed problem patients who have read unreliable information on

the Internet. Again this discussion demonstrates that the clinical signs and symptoms and laboratory measures used to categorise thyroid disease are by no means definitive. Consequently, the next step in a discussion of the boundaries of thyroid disease is to explore how such ambiguity is organised into particular categories. What are the politics of making sense and managing such ambiguity?

In her book *Tears Behind Closed Doors* (Holmes 2002), Diana Holmes refers to an article that appeared in the British Thyroid Foundation newsletter by Dr Peter Daggett, a Consultant Physician and Endocrinologist, entitled *The Perils and Pitfalls of Unnecessary Thyroid Replacement* (Daggett 2000). In the article, Daggett describes a hypothetical case of a patient who claims to have hypothyroidism, but who has blood test result that fall inside the reference range. Daggett begins by describing hypothyroidism as follows:

An underactive thyroid gland produces many symptoms, which make people feel unwell, and because the condition develops slowly, affected individuals have often felt ill for a long time. The stress and strains of modern life, however, commonly cause similar symptoms and when the effect of our miserable weather is added, it is not surprising that many people are convinced that their thyroid gland is abnormal, even when it is not. Picture the scene. A middle aged lady (and it is usually ladies who are affected) feels tired and cold (it is cold in January). She is gaining weight (too much of the poor diet) and her hair is dry (the wrong shampoo). Her GP suspects that she is suffering from an underactive thyroid gland, but the laboratory report nothing wrong. She is not convinced and goes for a second opinion. (Daggett 2000: 1).

Daggett then describes the patient's experience with the clinician who provides the second opinion. She is told that she has clinical symptoms of a slow pulse and low temperature, which are indicative of an underactive thyroid and she is given a natural thyroid replacement which the clinician believes to be superior to standard synthetic thyroxine. The result of this prescription is that 'the lady feels great (placebo effect)' (Daggett 2000: 2) but eventually, after a few months, becomes unwell again and is told to increase her dose. However, after a few months, she feels unwell again. The patient then goes back to her GP who discovers that she is thyrotoxic (has an overactive thyroid gland because of the excessive dose of natural extract thyroid given to her by the second clinician). Her G.P. asks her to cease taking the natural thyroid extract but she does not want to do this

and asks to see a hospital specialist, 'pity the poor endocrinologist' (Daggett 2000: 2). The endocrinologist finds that her thyroid gland is working normally but it is impossible to read her TFTs because they are being distorted by the thyroid hormones she is taking. The only option left is to treat the patient by stopping the hormone-replacement so that her endocrine system can return to normal. Daggett's hypothetical concludes:

She is unhappy. The GP is unhappy because he has been bypassed and the endocrinologist is unhappy – he has seen this sequence many times and is tired of getting into trouble for telling patients that he does not agree (Daggett 2002: 2).

Daggett's article 'feminises' the symptoms of thyroid disease, specifically psychological impairments, in order to deal with experiences of the disease that do not fit into the formal model of thyroid dysfunction that is arbitrated by biochemical reference ranges. The characterisation of hysterical middle-aged women claiming to have thyroid disease when they have simply used the 'wrong shampoo' infers that all patients who express dissatisfaction with their diagnosis and treatment are hypochondriacs and timewasters who place unrealistic expectation upon clinicians and the health service. Dissatisfied patients become cast as attention seekers who demand recognition from the medical profession in order to fulfil an unobtainable sense of well being. That is, the ordering of thyroid biology they have deployed in order to rid themselves of symptoms is deemed not only to be un-scientific, but also hysterical and self-interested. Moreover, such patient-led criticisms of the current management strategies for thyroid disease, are considered to jeopardise the professional ability of the 'poor endocrinologist' whose expertise is questioned by such troublesome patients.

Given opinions like Daggett's, the grievances of dissatisfied patients also include the bedside manner of doctors when approaching their problem. For instance, in 2006, the Society for Endocrinology newsletter received a petition raised by Diana Holmes (see appendix 8) of over 2000 signatures registering:

...a formal complaint against the clinical practice of the majority of the medical profession with regards to the diagnosis and management of hypothyroidism' including 'the over reliance on blood tests...emotional abuse and blatant disregard...over the

suffering experienced by untreated/incorrectly treated thyroid patients (Holmes 2007)

As the above patient and professional accounts of thyroid disease have demonstrated, the symptoms can be defined as a result of thyroid disease, but also, as subjective assessments of health and well-being. Consequently, experiences of thyroid disease do not fit into the biochemically established categories and thus their corresponding clinical symptoms slip from the status of 'supported and validated phenomena' and become liminal thyroid abnormalities that fall within the 'experience' of the 'individual'. As has also been demonstrated, one of the mechanisms through which this is done is the feminisation of 'bad' evidence that does not fit into the established evidence-based regime that dominates current medical practice within the NHS and western healthcare systems more generally. However, in spite of this, such strategies still fail to account for the symptoms that some patients complain of, despite TFT results, that fall within the reference range and, therefore, fail to silence the dissenting voices of dissatisfied patients and sympathetic clinicians. Having discussed how symptomatic evidence is deployed by various thyroid stakeholders, the following section will explore the socially embedded nature of the biochemical reference ranges which, it is claimed, provide more 'reliable' evidence of thyroid disease.

Evidence of what?

The Consultant who manages the thyroid clinic on which this study is based gave two explanations for the dissatisfaction of some individuals with regard to the diagnosis and treatment of their thyroid disease. According to the 'conventional academic medical' explanation, patients who fall within the reference range of normal thyroid function, but who still suffer from symptoms, are suffering from a functional somatoform disorder. A functional somatoform disorder is a psychiatric condition in which individuals complain of chronic varied physical symptoms that have no identifiable physical origin (Sharpe and Carson 2001). As discussed, this label is also used as a polite way of accusing dissatisfied patients of the charges brought against them by clinicians such as Daggett. That is, as Weetman claims in quote below, often this diagnostic label is misused and

not explained to patients properly. Consequently, it is perceived by patients as a rejection of their experience of symptoms and suffering:

Most patients with functional somatoform disorders are given explanations which are experienced as either a rejection of the reality of the symptoms or a simple collusion and acquiescence to the patient's own biomedical theory. (Weetman: 2006; 233)

Interestingly, Weetman does not believe that the way to tackle thyroid disease is through the derision of the patients and their various biomedical theories. Unlike Daggett, Weetman asserts that it is important to communicate with, and not simply ridicule such patients. In doing so, he claims that patients can be empowered to understand their problems further and 'reduce the demands they make on health services' (Weetman 2003: 233). Therefore, although both Daggett and Weetman agree that there is no thyroid disease without abnormal thyroid biochemistry, they approach patients who claim otherwise in very different ways. These different approaches are important to highlight because, as demonstrated, the dissatisfaction is not only exacerbated by the established science of thyroid disease, but also by the ways in which patients are handled by the medical profession during the clinical encounter.

The second explanation, or set of explanations, for patients claims of malingering symptoms, identified by the Consultant is a number of, as yet, unsubstantiated biomedical theories that prove that some biochemically normal/borderline patients do in fact have a biological basis for their illness. Briefly, these explanations can be listed as; Genetics, the problem of standard thyroid replacements and thyroid autoimmunity. Each of these potential explanations are described further below.

Genetics

One explanation which has been advanced suggests that genetic variation between individuals with regards to thyroid metabolism, receptor affinity or receptor (Walsh 2002b: 717) located around the deiodinase type II gene. Briefly, this theory suggests that certain organs may find it harder to break down T4 into active T3, resulting in slow metabolic function. As will be discussed below, this could have an impact on the efficacy of the standard synthetic T4-only replacements that the clinical practice guidelines currently

endorse (Association for Clinical Biochemistry et al. 2005). However, this theory is currently unsubstantiated:

...recent animal experiments indicate that only the combination of T4 and T3 replacement, and not T4 alone, ensures euthyroidism in all tissues of thyroidectomized rats. It is indeed the experience of many physicians that there exists a small subset of hypothyroid patients who, despite biochemical euthyroidism, continue to complain of tiredness, lack of energy, discrete cognitive disorders and mood disturbances...Confirmatory studies on this issue are urgently awaited. It could well be that a slow-release preparation containing both T4 and T3 might improve the quality of life, compared with T4 replacement alone, in some hypothyroid patients (Escobar-Morreale et al. 2005: 2490)

In addition, when discussing this issue with the Consultant who ran the thyroid out-patients clinic studied as part of this thesis, he suggests that these 'confirmatory studies' may not be merited, as they do not account for the 'normal' status of the pituitary gland which is ascertained through the measurement of TSH as part of standard thyroid function blood tests. As the Consultant explained to me during an interview:

...the alternative view is that it's for some reason the thyroid hormone, although it's normal in the blood is not normal in parts of their tissues, but the evidence supporting that is non-existent and is extremely speculative because the whole idea is that the pituitary gland is the most sensitive part of us, that monitors our status and so the big sort of key feature is that if the pituitary gland is making, if you've got an intact pituitary gland, as an assumption which usually is the case, um, then if the pituitary gland is happy then your bones, your brain, your muscle, your heart, they're all seeing the same amount of hormone.

Standard Thyroxine Replacement

As outlined above, there is some evidence (Escobar-Morreale HF et al. 1996) to suggest that there may be a biologic basis to the dissatisfaction that some individuals feel when taking a course of standard L-thyroxin replacement. It is claimed that this preparation is not appropriate for all patients, as they cannot successfully convert the stable T4 into active T3 in the tissues of some organs of the body. This problem of conversion is not picked up by standard thyroid function tests because they only measure the levels of TSH, T4 and T3 and not the process of hormone action. That is, according to the patient support group Thyroid UK, such individuals appear to have normal function because tests are measuring the thyroid hormones that are 'hanging around' in the blood and not what is being converted into

a form that will fuel metabolism. Subsequently, many of these dissatisfied patient groups and sympathetic clinicians claim that a combination of T4 and T3 replacement, or even natural thyroxine derived from the thyroid glands of pigs, would provide a better therapy because the treatments are more natural and mirror what a 'normal' thyroid gland would produce (Durrant -Peatfield Barry 2006: 132). However, as the Consultant reflects, this theory is untested by evidence-based research methods and, therefore, cannot be introduced to the practices of the clinic:

There have been one or two animal studies that have tried to look at the T4 and T3 levels in different tissues and have suggested that they might not be quite as straight forward as that, but any study that's tried in a double blinded randomised way to look at the beginning problems into normal people for comparing T4, T3 or whatever and it's not actually been able to consistently demonstrate that people do any better. So, in the current era of evidence based medicine, it is actually if very difficult...

Moreover, the established professional thyroid community disapproves of 'natural' preparations, because they are considered to be unstable and unsafe and they are currently un-licensed in the United Kingdom. As the British Thyroid Foundation states:

There is no current evidence of any advantages of taking natural thyroid extract over synthetic thyroxine. The disadvantages are that because it is made from pooled thyroid glands from animals, which may vary from batch to batch in their thyroxine levels, the exact dose of thyroxine cannot be estimated precisely. Therefore one dose from one batch of tablets may well create different thyroxine levels from the same dose in a second batch of tablets. As a result, endocrinologists have abandoned using this in favour of the pure form of thyroxine, which is chemically exactly the same as that which is made by the normal thyroid gland. In this sense, thyroxine treatment is as natural as possible (The British Thyroid Foundation 2006)

Subsequently, as the two extracts above demonstrate, this disagreement about what form thyroid replacement should take embodies the broader debates that surround EBM. The established thyroid community, represented by the BFT, support the prescription of synthetic thyroxine because it addresses broader concerns about standardised protocols of efficacy and safety. The alternative thyroid clinicians, such as Dr Durrant Peatfield, endorse the prescription of 'natural' thyroxine because it is

considered to be a better match to the hormones that individual patients produce.

Thyroid Autoimmunity

As described previously, the most common cause of thyroid disease is an autoimmune response that attacks the gland and hinders its ability to produce thyroid hormones normally. There is a theory that the presence of auto-antibodies in individuals might cause ill health through mechanisms that are independent of hypothyroidism, specifically depression (Pop et al. 1998). Therefore the persistent symptoms that some biochemically euthyroid individuals complain of may not stem from a somataform disorder, but from this autoimmune mechanism. However, there is little evidence to support this.

Since these factors are as yet unsubstantiated, they have little impact in the established guidelines and practices within the clinic – apart from when they are raised by dissatisfied patients who have independently researched possible explanations for continued ill health. As discussed in the previous chapter, hypothyroidism is usually treated within the primary care system. Difficult patients are sent to the Consultant for what is termed 'reassurance', meaning that 'problem patients' will hopefully stop contesting their diagnosis and treatment in the light of what the more experienced specialist advises. However, much to the bemusement of the Consultant in the clinic that was observed for this study, often this did not happen. As he reflects in the following quote, his expertise was often questioned:

Um, I think it's odd that people can say my uncle had that, or my grandmother or the cats nephew had radio-iodine there and didn't do well there so, I'm not doing it and I just sort of think if I went to QuickFit [?] and said I need, you know, and they said, look, part three of your exhaust needs doing, I don't go well, I think it's actually part two and I just think it's odd that people just don't believe me.

Therefore, although these alternative theories of thyroid disease remain unsubstantiated, they do have an impact on the clinic and the practice of the Consultant, as much of his work is concerned with convincing dissatisfied patients that there is nothing wrong with their thyroid gland. Subsequently, these alternative theories saturate the practices of the clinic and are also performed alongside the established biochemical standards

and treatments of thyroid disease. However, the lack of evidence to support them means that they are weak in terms of the evidence based discourse of contemporary health service delivery that guides the practice of the Consultant. Due to this weakness, there is currently very little work being done to develop them, therefore maintaining the current definitions of disease, specifically the dichotomous and hierarchical relationship between biochemical and symptomatic evidence.

This maintenance of the biochemical model of thyroid disease, and the lack of investment in research of alternative models of disease, is unsurprising as the potential impact of alternative models of thyroid disease would include complex and expensive diagnostic tests, for example a genetic test to ascertain an individual's thyroid receptor affinity. In addition, the development of a new form of thyroid replacement therapy would almost certainly cost exponentially more than the current generic preparation. It should be noted that failure to follow up on these research trajectories rehearses the EBM patient centred debate, where economic concerns and scientific rationality are valued at the expense of patient care. However, as the following discussion will demonstrate, this caution is also necessary due to the ambiguity of even the most 'scientific' of evidence, especially when it is situated within the everyday experience of thyroid disease.

Negotiating biochemistry

In February 2000, *The Colorado Disease Prevalence Study* (Canaris et al. 2000) was published in *Annals of Internal Medicine*. Referring to a familiar problem, it states that the prevalence and significance of thyroid dysfunction remains controversial due to the poorly delineated systemic effects of abnormal and, specifically, mild thyroid failure and the unclear relationship between symptoms and biochemical thyroid function. Thus, the paper sought '[t]o determine the prevalence of abnormal thyroid function and the relationship between (1) abnormal thyroid function and lipid levels and (2) abnormal thyroid function and symptoms using modern and sensitive thyroid tests' (Canaris et al. 2000: 256). The study recorded the biochemical thyroid function (TSH and T4), lipid profiles and responses of 25,862 individuals who completed a hypothyroid symptom questionnaire at a state health fair in Colorado during 1995. As detailed in appendix 5, high cholesterol is a symptom of hypothyroidism and there is an established

relationship between mild and subclinical hypothyroidism and raised lipid profiles. It is seen, therefore, as a risk factor that could affect cardiovascular health. Moreover, this relationship could provide a reason to treat subclinical hypothyroidism since prevention of raised lipid profiles could influence long-term coronary health.

The Colorado Disease Prevalence Study revealed a prevalence of elevated TSH (subclinical hypothyroidism) at 9.5% and decreased TSH (subclinical hyperthyroidism) at 2.2%, both of which correspond to similar studies (Flynn et al. 2004, Vanderpump et al. 1995). Of those already diagnosed with hypothyroidism, 40% had abnormal TSH levels, suggesting that clinicians need to monitor patients on thyroid replacement more frequently in order to make sure that the regimen fully corrects the inhibited production of the gland. Significantly, it was found that 'lipid levels increased in a graded fashion as thyroid function declined' (Canaris et al: 2000: 526), suggesting a long term issue of cardiovascular health. Symptoms were reported more often in hypothyroid than euthyroid individuals. Moreover, if symptoms were reported to have developed in the last year, the presence of the disease, confirmed biochemically, was more likely. The confirmation of newly developed symptoms affirms their difference to 'normal feelings' that may be confused for the many non-specific symptoms of thyroid disease. However, in spite of the link between the amount of symptoms presented and the temporal onset of such symptoms, the study concludes that the relationship between symptoms and biochemistry is 'weak' (Canaris et al: 2000: 533). The study found that although symptoms increased as thyroid function depleted, there was no clear pattern to the type of symptoms suffered and the severity of disease. The fact that sensitivities of symptoms are low means that disease cannot be ruled out even if a symptom is not reported. The possible existence of disease without perceptible symptoms compromises the validity of the symptom as evidence of disease. The poor predictive power of symptoms means that they can produce false positive results.

As this study of disease prevalence demonstrates, within this range of evidence, 'reliable', 'repeatable' and epidemiologically-defined levels of thyroid function cannot be linked to their less reliable, individual, symptomatic evidential cousins. It shows that even definitive abnormal biochemistry is not always tied to symptoms, a point that was often brought

up by the Consultant in 'reassurance' consultations with dissatisfied patients in the clinic. For instance, as he reflects:

Nearly 10 % of subjects not taking thyroid medications had a thyroid abnormality, which was probably unknown to them, and the abnormality was detected because of testing

In the clinic, the deployment of this research was used to destabilise the status of symptoms as evidence. Although in some respects this strategy also destabilises biochemistry as evidence, it also recapitulates the established hierarchy of evidence. Symptoms remain less reliable markers of disease than biochemistry and, moreover, ones that are more difficult to interpret and connect to a cause with any certainty due to their ambiguity. It would seem that current studies of symptomatic evidence acknowledge the complexity and indeterminacy of the evidence but, ultimately, opt for the treatment option that is safest in terms of the prevailing evidence-based tenets of contemporary clinical practice. As stated by a clinician and researcher from another hospital who interviewed for this study in connection to the administration of T3 and natural thyroxine:

So, in the current era of evidence based medicine, it is actually very difficult... you would not get a, you would not get a chemotherapy drug on that basis, you wouldn't start a new [unclear] breast cancer prophylactics or a new heart attack drug so if you apply the same principles that we have to, to everything else, then just because one or two people report feeling better, I'm afraid, that doesn't stand up.

Therefore, even though the biochemical evidence for thyroid disease is fraught with ambiguity, it is used selectively by both clinicians and patients and coordinated to various orders of thyroid biology that value specific diagnosis and treatment strategies. However, due to its evidence-based endorsement it maintains the position of the most reliable data with which to make diagnostic and treatment decisions. As the Consultant comments above, 'in the current era of evidence based medicine' such principles have to be adhered to.

Deploying indeterminacy through 'biologisation'

As discussed previously the symptoms of thyroid disease were first observed and carefully documented by the renowned clinical physicians William Gull (hypothyroidism), in 1874 , and Robert Graves

(hyperthyroidism), in 1835. In the last thirty to forty years this initial observational 'evidence' of disease has been transformed into population-derived, standardized reference ranges assessed in the laboratory – now considered the 'frontline test' (Association for Clinical Biochemistry 2006) in the diagnostic process. However, the assumption that the technology of the blood test has simply made professional practice better, standardised and more reliable is a gross over-simplification. The measurement of the thyroid pituitary axis, the creation of reference ranges of function and the increased sensitivity of such tests have meant that sub-clinical measures of thyroid function have come into being. These sub-clinical measures suggest mild glandular failure and/or evidence that the gland may fail in the future. Ironically, therefore, the 'hard' biochemical evidence that is supposed to make diagnosis more reliable has in fact created a new realm of uncertainty: it is not clear if these subclinical categories cause symptoms or not. Furthermore, this transformation of the disease from one understood through symptoms to one based on biochemistry has led to the subjugation of the medium through which most individuals understand and experience their disease no matter how severe it is. This disparity produces immense frustration on an individual level, as expressed by Janice, a hypothyroid woman in her forties:

Yeah, some are, they are quite good at fobbing you off, you know, because they think, yeah, well, that's in this level, therefore it's this. If it's in this level it means this. They don't think, well, actually, this... Hang on a minute. Step back. This person's showing... If perhaps you look at that, plus her symptoms, maybe it does mean that; that we're not all going suddenly into compartments. They want to quickly put you in a compartment. You're in that compartment... Yeah, the results of the blood test and they didn't seem to be very holistic in their, you know... I'm someone who strongly believes in a holistic approach to health and I'm very into sort of alternative things. You know, aromatherapies and I have Chinese massage at the moment, Indian neck massage, you know, all those sort of things as well and looking... And that, I feel, I just feel that sometimes they're not looking at the whole picture...

Moreover, this frustration is not only centred on a tension between professional, alternative and lay models of medical practice. It also affects how patients experience the symptoms of disease in relation to their biochemistry. As Janice recounts:

I've got friends who have got massively high thyroid levels erm, and they get the opp, they get opposing symptoms. They get what you get with low, and then I've got a friend who's got such low thyroid levels she's on 200 micrograms or milligrams a day, and she's a total skinny little rake....Yeah she's got racing heart, erm all the things so I don't think always its as cut and dry with thyroid.

It can be argued, therefore, that biochemically-defined thyroid disease is a version of the disease which complicates thyroid pathology and results in it becoming even more ill-defined. Conversely, however, this model of disease can also represent an individual experience of thyroid dysfunction more than sufficiently. As the Consultant who ran the thyroid clinic pointed out, many of the patients who attended his clinic were there because they were dissatisfied and it was his job as a specialist to reassure them that they were getting the correct care. He also added that the majority of patients were happy with their treatment and it was successfully treated within primary care using evidence based methods. Therefore, it is important to note that although the evidence for thyroid disease is uncertain, it does not mean that all patients are dissatisfied. The evidentiary forms that represent both of these experiences intertwine to comprise the definitions and practices of thyroid disease: laboratory tests that measure the biochemical activity of the thyroid gland, the palpation of the thyroid gland within the outpatients clinic, the symptoms that patients report, and the symptomatic detective work that leads a patient's G.P. to order a thyroid function test in the first place.

It is therefore impossible to separate 'evidence' that is scientifically substantiated from 'evidence' which is anecdotal and subjective. Firstly, because the symptoms of thyroid disease are difficult to identify as discrete entities, they cannot be separated from individual assessments of health and well being. Secondly, the disease will not always manifest itself symptomatically, even when there is irrefutable biochemical evidence – as demonstrated by the 10% of the respondents in the Colorado Disease Prevalence study who were biochemically hypothyroid but who did not complain of symptoms. As a consequence again it is clear that the technology of the thyroid function blood test, developed to make the diagnosis of thyroid disease more straightforward and reliable, has fundamentally shaped how thyroid disease has come to be defined and attended to within the clinic. This again destabilises the idea that there is a

single, pre-existing form of evidence that can be unearthed by clinicians or a specific pathological lesion in the body identified.

As the medical historian Keith Wailoo (1997) describes in his history of haematology, technologies that have been developed throughout the 20th century to assist clinicians in their work have become embedded in the identity of the disease they have been designed to attend to:

In the twentieth century, doctors have learned to think and act through their technologies. That is, two key features of twentieth century medicine have been the deployment of technologies by specialists and the ways in which technologies such as EKGs and fetal heart monitors have assigned coherent meaning to particular symptoms and bodily features, guiding the physician's mind and hand. Such technologies have played a large role in assigning and 'identity' to diseases (Wailoo 1997: 1).

For example, as Wailoo demonstrates, in the early 1980s the acquired immune deficiency syndrome (AIDS) was a fragmented collection of rare diseases (Kaposi's sarcoma, pneumonia, tuberculosis). Only after a test for human immunodeficiency virus was developed did AIDS become a 'discrete, immunologically defined entity' (Wailoo 1997 :4). This shaping or 'looping' effect on disease by diagnostic and therapeutic technology is clearly demonstrated in the case of thyroid disease. The invention of tests that, with increasing sensitivity, have focused on the negative feedback loop between the thyroid and pituitary gland has transformed the identity of thyroid disease over the last century. The collection of non-specific and idiosyncratic symptoms caused by various states of thyroid dysfunction, have been calibrated to the thyroid pituitary axis. Subsequently, they have been reigned into the narrative of thyroid biochemistry, ordering them and the overall disease into a more coherent linear form. Moreover, as well as making the disease legible and pulling it into line with the current tenets of EBM, the technology of the thyroid function blood test has also fundamentally changed its traditionally symptomatically-defined borders.

Clinicians who are sympathetic to the grievances of dissatisfied patients are critical of the increasingly biochemical definition of thyroid disease and see it as proof of the negative consequences of EBM on good clinical practice. Clinicians such as Dr Gordon R.B. Skinner and Dr Barry Durrant-Peatfield, for example, believe that reliance on thyroid biochemistry erodes the clinical skills traditionally used to translate symptoms into a clinical

diagnosis. In contrast, clinicians and patients groups (such as the BTA and its affiliated patient group the BTF) who think that the diagnosis and treatment of thyroid disease should be led by evidence-based biochemical reference ranges believe that such an approach ensures safe and efficacious practice. In addition, as Armstrong (2007) would claim, such evidence-based practice also protects the profession from threats to their expertise by commercial and state pressures. That is, biochemical reference ranges provide a firm, professionally-mediated categorisation of thyroid disease that can, to some extent, deflect and debunk lay theories of disease and subsequent demands for untested treatments. Moreover, this firm definition of thyroid disease means that established and economically viable treatments are maintained as the standard. However, this tension between these evidence based and patient centred approaches to thyroid disease, and the partial explanation of thyroid disease they generate, often provides the basis for further strategies that attempt to attend to this non-compliant pathology. As has been described a number of patient activist groups, dissatisfied with the treatment they receive, use the indeterminacy of evidence to construct an order of thyroid biology that can account for their suffering and justify additional treatment strategies. For instance, patients and sympathetic clinicians imbue symptomatic evidence with greater authority on the grounds that they are a more accurate expression of their individual biology. They also claim that thyroid extracts derived from animals are a more efficacious treatment because they are more 'natural' and therefore absorbed by their bodies more efficiently.

Thyroid disease, therefore, is not only medicalised by clinicians (through the application of the thyroid pituitary axis), but is also 'biologised' by various groups of dissatisfied patients. That is, patients make themselves objects of an alternative biology of the thyroid gland in order speak to, and then contest, the language of the thyroid clinic and hopefully secure their desired diagnosis and treatment. As Charis Cussins (1996) claims in her ethnography of an infertility clinic in the United States, such acts of objectification give patients agency and actually enable them to participate in the process of their treatment. That is, individuals objectify their bodies during various points in the therapeutic process in order to 'bring about desired changes in her [their] identity' (Cussins 1996: 600). In the case of the infertility clinic, the desired change is pregnancy and motherhood, and in the case of the thyroid clinic, the desired change would be a symptom-

free life. In so doing, the traditional humanist argument that 'selves need to be protected from technological objectification to ensure agency and authenticity' (Cussins 1996: 575) is undermined.

For example, the process of 'Objectification as Medical Operationalization', identified by Cussins, renders particular body parts visible through various technological practices in order to make them available to intervention. A pelvic examination identifies fibroids, cysts and, therefore, potential causes of infertility and subsequent treatments and procedures that can overcome them. Similarly, in the thyroid clinic the results of blood tests isolate thyroid disease to thyroid and pituitary axis. Therefore, patients in both of these locations make themselves compatible with the instruments, drugs, procedures and material surroundings of the clinic that promise to bring about the desired changes in identity. This process of forming a 'functional zone' where things of different kinds (individuals, tests, tools) can be coordinated is what Cussins calls 'ontological choreography'. Such objectifications allow the coordination of 'things' that will realise the desires of the subject (e.g. blood test results hopefully identify a cause of symptoms that can then be treated).

However, as Cussins also notes, this process does not necessarily 'guarantee a seamless and successful solution'. (Cussins 1996: 575). As evidenced in the thyroid clinic, such objectifications are only successful, and can only facilitate 'ontological choreography', if they maintain a 'metonymic relation' to the subject. That is, as soon as the instruments, drugs, procedures and material surroundings of the clinic become incompatible with, or fail to meet, the desires of the patient, the functional zone in which 'ontological choreography' takes place disintegrates. Consequently, ontological choreography is not achievable for dissatisfied thyroid patients because the central tool of the thyroid clinic, the TFT blood test, is ultimately incompatible with their subjective desire to eradicate symptoms (i.e. the rejection of symptoms by the medical profession as a reliable measure of disease means that symptoms are not the focus of clinical intervention). Therefore, the desire of dissatisfied patients to rid themselves of such symptoms is not sufficiently addressed by established tools and practices of the clinic.

In spite of this, the technical deficit of such established tools does not result in the silencing of the desire for a symptom-free life. In fact the technical deficit of thyroid function tests (i.e. their ability to account for thyroid type symptoms in subclinical and euthyroid individuals) actually enables dissatisfied patients to construct orders of thyroid biology that have the potential to rid them of their symptoms. That is, the inadequacy of current thyroid function tests, coupled with the indeterminacy of the evidence used to justify them, results in the ability of patients to negotiate solutions to their problems. To be precise, the thyroid treatment gap results in a situation where patients and doctors deploy their orders of thyroid biology, and specifically hierarchies of evidence, to enter a process whereby they negotiate and try reach compromises or even reconcile the ambiguities of the disease and each other.

As Rabinow notes, the development of the concept of biosociality was motivated by the empirical realisation that molecular control over the genome had the potential to remake nature through technique. This ability to intervene on the progression of disease within our genes led Rabinow to claim that:

it is not hard to imagine groups formed around chromosome 17, locus16,256, site 654,376 allele variant with a guanine substitution. Such groups will have medical specialist, laboratories, narratives, traditions, and heavy panoply of pastoral keepers to help them experience, share, intervene and 'understand' their fate. (1996: 102)

As Rabinow suggests, the ability to intervene in human health at the molecular level has had significant implications for how individuals relate to themselves and others who might share their faulty genes. He predicted that the techniques that promise control of genetic pathology would have the social effect of catalysing a collective aspirational quest uniting individuals who share faulty genes, and those who care for them, in order to gather information and potentially develop cures. In contrast, as demonstrated throughout this chapter, the technologies of thyroid disease do not facilitate this level of intervention. The inability of blood tests and symptomatic schemas to cure – or even to accurately diagnose -- thyroid disease, at first glance, *disables* patients as individuals intervening in their disease. On closer inspection, this technical deficit actually facilitates the ability of patients to intervene in their thyroid disease via the construction of

a thyroid biology that can meet their needs and support their desired medical interventions.

In conclusion, it is clear, that 'definitive biochemical' and 'partial symptomatic' evidence are not wholly separable. If we were to reflect on the constellations of evidence within the treatment of thyroid disease, it tells us as much about the epistemological landscape of contemporary biomedicine as it does about the 'biology' of thyroid disease. This tension between biochemical and symptomatic evidence is a specific example of the tension between evidence-based and patient-centred models of clinical care. The importance placed on biochemistry strives to rid clinical practice of inconsistent and opinion-based clinical decision-making, whilst the privileging of symptoms strives to attend to the individual experiences and therapeutic needs of the patient.

What is clear from the data collected by this study is that patients and clinicians deploy the uncertain aspects of thyroid disease to construct particular orders of thyroid biology. That is, the gaps left by the indeterminate relationship between biochemical and symptomatic evidence, are filled with versions of thyroid biology that deploy this uncertainty to satisfy a range of needs, rights and responsibilities. Subsequently, thyroid disease is revealed as a weak context for medicalisation because it is impossible for the medical profession to discipline thyroid disease into a biochemically circumscribed disease category. Instead, thyroid disease becomes a location around which a number of thyroid biologies can be negotiated and enacted, all of which are intersected by additional social, biological, political and bureaucratic trajectories.

Dissatisfied patients implant their lingering, ambiguous symptoms into the gap left by the disjunction between the model of the thyroid pituitary axis and the symptomatic evidence of thyroid disease. The ambiguous symptoms experienced by patients following treatment are thus transformed into 'proof' that their individual thyroid disease is not represented by the formal biochemical reference ranges currently used to diagnose and manage thyroid disease. Consequently, an order of thyroid biology is constructed that attends to each individual's experience of thyroid disease through symptoms. Moreover, such patients bolster this particular order of biology further by co-opting strategies employed by health policy

initiatives that facilitate 'patient choice'. Often patients seek referral to a Consultant in order to bypass the 'generalist' advice provided by their G.P. and to secure specialist expertise that can account for their complex biology. Therefore, dissatisfied patients do not reject medical expertise completely. Instead they negotiate the terms of thyroid disease by restructuring and altering its formal biomedical categories and creating a biological order that relates to their symptomatic experience. In the concluding chapter I turn to an examination of attempts to coordinate the instruments, drugs, procedures and material surroundings of the clinic to particular orders of thyroid biology among many of the actors introduced in this study so far. In doing so, I attempt to further characterise the process that might be described as 'technically deficient biosociality' in order to trace its implications in both new and not so new directions.

Chapter 6

Awkward compromises: practicing thyroid disease

Well, the way I think of this is that if I'm writing a national guideline, you have to be very black and white in what we say so that's what I call the macro level, i.e., you're talking about a big population. You can then go down to the micro level, which is you in your own hospital and what you might do here... then the nano level is what you do with an individual patient in your office. So, I do different things, at three levels... Consultant, weekly outpatients' thyroid clinic.

There is a difference between what is written, in the form of clinical guidelines, and what is practiced within the clinic. However, as the Consultant quoted above implicitly acknowledges, these differences are an inevitable consequence of the treatment of thyroid disease. As he states, guidelines have to be clear and not context-dependent – 'very black and white' - as they are widely disseminated and are produced in order to regulate practice across a large population. However this does not mean that within the practice of a hospital department or during an individual consultation, guidelines are – or indeed should be - strictly adhered to: guidelines are produced in broad terms so that they may be applicable to varied situations and localities and employed differently within them. This is particularly the case when attending to thyroid disease and is due to the ambiguity that surrounds how patients experience it and the indeterminacy of the evidence used to inform and justify diagnostic and treatment strategies. In fact, as referred to at the end chapter two the very existence of the guidelines (see Association for Clinical Biochemistry et al. 2005) for the diagnosis and treatment of thyroid disease are themselves an attempt to reconcile the varied and competing accounts of thyroid disease and the practices used to attend to it within the NHS²². Therefore, guidelines are not merely a collation of the best available scientific evidence that can be used to inform and improve practice. They are, as the creation of the guidelines for the diagnosis and treatment of thyroid disease can attest, generated from and a dynamic response to the complexity, variability and contingency

22 In a section of the guidelines entitled 'The Need for National Guidelines' the authors state that it is clear that the different stakeholders of thyroid disease (patients, clinicians, laboratory workers) 'uncertain about many aspects of thyroid function testing' (Association for Clinical Biochemistry et al. 2005: 13). Hence 'the need for national guidelines for something as common as thyroid function testing is self-evident.

of practice across the healthcare landscape. This is clearly apparent in the above quote from the Consultant where the varied and often competing scales and levels of thyroid disease are navigated by in an array of ways through the deployment of the guidelines.

As suggested in the previous chapter, the high level of ambiguity that characterises thyroid disease means that it is difficult to discipline through established medical models: symptoms and experiences of thyroid disease vary to such a degree that broadly applicable terms of practice cannot be established. The authority of the biochemical model derives from its status as a source of objective evidence, but in the case of thyroid disease this diagnostic procedure and its corresponding treatment protocols are constantly undermined by the ambiguous and *indeterminate* relationship within and between categories of symptomatic and biochemical evidence. The validity of a 'medicalised', or specifically 'biochemicalised' model of thyroid disease, therefore, is weakened and replaced by diverse and competing versions of medical and biological knowledge which allow for – and derive from – that uncertainty. This *variable knowledge* is co-ordinated – or choreographed – by actors to establish orders of thyroid disease which are then negotiated by clinicians and dissatisfied patients in the hope that a satisfactory model of thyroid disease can be reached and subsequent treatments can be prescribed. What we recognise as 'thyroid disease' is consequently dependant on a complex array of micro-practices that enable us to see and act upon a set of *variable conditions*. In Mol's terms, thyroid disease is 'done': it is enacted through practice – practices, Mol claims, makes disease 'visible, audible, tangible and knowable' (Mol 2002: 37).

In this final chapter I engage in a close reading of the work of medical philosopher and ethnographer Annemarie Mol to argue both that her work offers a useful framework through which to understand thyroid disease, and that thyroid disease can offer some additional dimensions to Mol's praxiographic model of disease. In particular, due to the need for both patients and clinicians to consolidate specific orders of thyroid biology in the pursuit and justification of particular treatments, they can demonstrate how the dispersed practices of doing disease, are reconsolidated, arguably something that is missing from Mol's monograph. In the context of thyroid disease such dispersed disease practices are performed with the aim of constructing rhetorical orders of thyroid biology and pathology that embody

the possibility of the fulfilment of various needs, rights, desires and responsibilities.

Tracking practice

The coordination through practice of various knowledges that 'hang together' to form what we recognize as a 'disease' is an approach to social studies of biomedicine developed by Annemarie Mol in her study of atherosclerosis. In her study Mol states:

If we no longer presume 'disease' to be a universal object hidden under the body's skin, but make the praxiographic shift to studying the bodies and disease while they are being enacted in daily hospital practices, multiplication follows. In practice a disease, atherosclerosis, is no longer one. (Mol 2002: 5)

According to Mol a disease is not an object or thing. It is 'as much the practices that intervene in it: the two [practices and objects] go together' (Mol 2002:156) For example, as Mol demonstrates in the case of atherosclerosis, the plaques that build up in arteries and cause them to harden can only become visible through the practice:

'A microscope is used to look at plaque, while plaque, if it is to be practically relevant in a hospital, needs a microscope (and dissection, slicing and staining techniques) to make it visible' (Mol 2002: 156)

Consequently, Mol claims that such 'Praxiographic stories have composite objects' (Mol 2002: 156) - diseases. Therefore, a disease is not 'a universal object hidden under the body's skin' nor is it a 'singular object' because the practices of enacting a disease are multiple. For instance, in the pathology laboratory, atherosclerosis is the thickness of an artery, practiced through dissection, slicing and staining techniques. However in the clinic, atherosclerosis is the pulsations of arteries felt by a clinician during a physical examination and the measurement of blood pressure in the leg. Therefore, as soon as the practices of atherosclerosis are observed it becomes a multiple entity, that is, it is constituted by a number of contrasting practices throughout various locations within the hospital. This is not to suggest that such multiple enactments are merely different disciplinarily perspectives on the disease – according to Mol they *are* the disease. The ontology of a disease is these multiple practices and through enactments of coordination adjustment and shifting they somehow 'hang

together' to constitute a broad understanding of the disease and possible ways in which it can be attended to (Mol 2002: 84). As Mol describes, for example, test results are added together in order to reach a single definitive diagnosis. This addition comes in two forms:

The first form of coordination on which coherence-in-tension depends is to add up test outcomes. It comes in two varieties. One of the forms of addition projects a common object behind various test outcomes:- 'the disease'. If the projections do not overlap, one of them is made to win. A hierarchy is established and the discrepancy between the tests is explained away. The second form of addition comes with no worries about discrepancies. It does not suggest that tests have a common object. Instead, it takes tests as suggestions for action: one bad test outcome may be a reason to treat; two or three bad test outcomes give more reason to treat (Mol 2002: 84)

Therefore, coordinations are not always smooth, and test results are often contradictory. However, these tensions are, as Mol demonstrates, worked out through the course of practice and eventually grounds for action are provided. In the case of thyroid disease, coordination through hierarchy is performed in response to the tension between evidence sources (biochemistry and symptoms) that some patients' reports. Blood test results are deemed more reliable as they are considered to be 'more scientific' and separate from individual feeling of well being. Moreover, coordination with 'no worries' about discrepancies also occurs - patients who display more than one established clinical symptom and have 'perfectly dysfunctional' thyroid biochemistry fulfil the criteria of a 'classic case' of thyroid disease. This was observed in the only male patient interviewed for the study. Robert, an accountant in his early forties, relayed during his interview that his level of TSH was 217 m/UL, an according to his Consultant a 'record' level. He then described that after he had taken thyroxin for about two weeks he managed to loose about 30 pounds in weight. Therefore, this patient was 'perfectly dysfunctional' and also 'perfectly treated' as his blood test results demonstrated overt hypothyroidism that went on to be treated successfully in a short space of time.

The second form of coordination, according to Mol, is the 'calibration of test outcomes' (2002: 64). This consists of making the results of different tests

calibrate with each other in order to provide a singular robust diagnosis. That is 'the threat of incommensurability is countered in practice by establishing common measures' (Mol 2002: 85). Subsequently, the various tests are made to speak of the same object, which can then be used to localize and quantify 'the disease'. For example, as discussed in detail previously, the focus of thyroid disease is the negative feedback loop between the pituitary and the thyroid gland - the thyroid pituitary axis. This focus means that the action of thyroid hormones at a cellular level is mostly ignored. As the Consultant points out, there is probably a lot of 'silent stuff' going on at this level. However, as there is no evidence to back these theories, the tests for thyroid disease focus on this 'evidence based' explanation.

According to Mol, therefore, it would be expected that over the course of diagnosing and treating thyroid disease the very practices that account for its multiplicity are those through which it becomes a manageable object that can be attended to in a clinical environment. Therefore, although Mol's study concerns atherosclerosis, it demonstrates the importance of taking the praxographic aspects of disease more seriously. As Mol claims:

...'[A]fter the shift from an epistemological to a praxographic appreciation of reality, telling about what atherosclerosis is isn't quite what it used to be. Somewhere along the way the meaning of the word 'is' has changed. Dramatically. This is what the change implies: the new 'is' is one that is situated. It doesn't say what atherosclerosis by nature, everywhere. It doesn't say what it is in and of itself, for nothing ever 'is' alone. To be is to be related. (Mol 2002: 53-54)

This focus on clinical practice for the social study of biomedicine is something that Steven Hass and Stefan Timmermans consolidate in a recent article that appeared in the *Sociology of Health and Illness*. They define practice as follows:

'Practice refers here to the actual contingent, situated process of performing tasks, doing work together, and transforming something into something different. An analysis of practice concerns, who does what, when, where, and with what consequences. By following clinicians and patients around, we can map the ways specific diseases foreshadow trajectories that are simultaneously deeply clinical, social, therapeutic, iatrogenic, political, and bureaucratic. Biology is no longer the invisible canvas for social action but both biology and social arrangements are continuously recreated as intertwined entities...(Hass and Timmermans 2008: 7).

Mol's provides an excellent means through which to analyse thyroid disease, specifically the ways in which the indeterminacy of evidence is enacted in order to construct particular orders of thyroid biology. Moreover, in return, this study adds to the purchase of Mol's work as it provides a situation where the closure of enactments and coordinations can be observed - an issue, in my opinion, that is not substantially addressed in Mol's monograph. That is, the tension that is indicative between the symptomatic and biochemical evidence used to diagnose and treat thyroid disease is so great and provides such an unsatisfactory explanation, that both dissatisfied patients and clinicians censor such ambiguity, and create robust orders of thyroid biology and pathology in order to reach satisfactory modes of explanation. Dissatisfied patients evaluate and coordinate evidence to a model of thyroid biology that acknowledges a pathological basis for their symptoms in spite of 'normal' or 'borderline' blood test results. Clinicians evaluate and coordinate evidence to a model that can satisfy the epistemological and economic constraints of evidence based practice. Specifically, they privilege biochemical evidence over and above, and sometimes in spite of symptoms, because symptoms cannot be linked directly to underlying thyroid pathology. In the following section of this chapter enactments that coordinate these orders of thyroid biology and pathology will be described. The first is in terms of how thyroid biology is coordinated to the temporalities biochemical and symptomatic orders of thyroid biology. The second is in terms of how particular symptoms are attributed and un-attributed – medicalised and de-medicalised – in relation to these two competing orders of thyroid biology.

Thyroid and Time

If we return to a description of the negative feedback mechanism between the thyroid and pituitary, gland referred to in chapter four, the relationship is conceived as being incredibly straightforward. As this description from *Understanding Thyroid Disorders* demonstrates:

In healthy people the amounts of T3 and T4 are maintained within narrow limits by a hormone known as thyroid stimulating hormone (TSH) or thyrotrophin. TSH is secreted by the anterior pituitary gland, which is a pea-sized structure hanging from the under surface of the brain, just behind the eyes and enclosed in a bony depression in the base of the skull. When thyroid

disease causes thyroid hormone levels in the blood to fall, TSH secretion from the pituitary is increased; when thyroid hormone levels rise, TSH secretion is switched off – a relationship known as 'negative feedback', familiar to engineers and biologists. (Toft 1995: 1)

However, if we look at the following quote from Alex, a hypothyroid patient in her mid-thirties, the relationship between a disturbance in this mechanism, visualised through the results of a thyroid function blood tests and the onset of disease experienced through symptoms, is not so simple. Specifically the symptoms of dysfunction can be present for a number of years, and often it is unclear that they are the result of a medical problem:

A: ...interestingly one of my friends, who's a nutritionist, she, she wanted to ask me if I had it about 10 years ago, but she didn't like, she thought it would be rude to ask me, so she didn't ask me.

M: 'So what, why, what made her think that?'

A: 'Coz my eyebrows are partly missing.'

M: 'Oh and that's it, the left, the outer third?'

A: 'Yeah. The outer third of my eyebrows are missing and she spotted that and she wanted to ask me.'

M: 'Is that the only thing she spotted, just that?'

A: 'Apart from I was really fat. Emm, no it could have been that, I was a lot fatter.'

M: 'Were you?'

A: 'I was a lot fatter, yes. I think I was, probably. I was about 12 stone at that point. It wasn't the heaviest I've been. I've been a lot heavier than that.'

What is interesting here is that Alex is suggesting that there was potentially a decade between her developing an under-active thyroid gland, with consequent symptoms, and it being diagnosed and treated. Therefore, although the biochemical narrative of thyroid disease is helpful, it is also problematic because its straightforwardness is often too 'theoretical' in relation to patients' experiences of the disease, i.e., the simple cause and effect process presented by the biochemical narrative of thyroid disease does not map onto the slow and non-specific onset of symptoms that can occur over a period of months and even years. The central location of this disconnection is the temporality of these two descriptions of thyroid disease. The description of the relationship between the thyroid and

pituitary glands is decidedly lacking in time, or to be more specific, an embodied context that provides a narrative that is within the experience of patients. Consequently the cause, onset and effect of such a dysfunction seems regimented, consistent, and therefore, artificial. Subsequently, the relatively straightforward story of how the thyroid gland fails, which is often communicated to patients in the accelerated time of the fraught and busy clinic, does not match the embodied experience of ambiguous symptoms.

This anomaly is further compounded by what the sociologist Everett Hughes (Hughes 1984: 346) identifies as the incommensurability between the 'routine' of the doctor and the 'emergency' of the patient. The routine manner in which the consultant approaches 'yet another' case of thyroid disease is directly opposed to that of the patient, who will be coming to terms with an event where their 'normal' health has been disrupted. This is particularly meaningful in the case of thyroid disease (hyperthyroidism and hypothyroidism), as it will have to be managed for life via a daily dose of thyroid hormone replacement (or in the case of patients with Graves disease, after Radio Iodine treatment). As Alex goes on to state in her interview, this disassociation between the model of disease, the symptoms that have been suffered over a long period of time and the treatment for the condition, seem to be under-acknowledged during the clinical encounter:

A: I'm the kind of person that says no, I don't want to take anything. I've never taken tablets in my life, why do I want to start now? And especially if someone tells you you've got to take them for the rest of your life. It's just like, no thanks.

M: It's a big deal, yes?

A: Yeah, no, I just walked away and said no I don't want that. I was absolutely adamant. So I thought, well, I'll do a bit of research. I need to come to terms with it on my own not someone sitting there saying, 'oh don't be silly. Off you go. Take the tablets. Off you go.' And you've only got 5 minutes to discuss an illness that's going to affect the rest of your life. That's all you've got with the doctors. They don't give you anymore time than that.

This disassociation between the routine work of the clinician and this critical event in the life of the patient is further demonstrated by the different approaches to the prescription of thyroxin, which has to be taken for life. Where clinicians see thyroxin as a replacement for what a damaged thyroid gland no longer makes, many patients (such as Alex) experience thyroxin

as a drug - as something that isn't natural - which artificially supports their metabolism in the long-term, meaning that their disease, whilst managed and hopefully asymptomatic, is nonetheless permanent. As Alex claims, she usually 'is the type of person that says no I don't want to take anything'. Therefore, she felt like she needed to 'research' thyroxin in order 'to come to terms' with a disease that she would have for the rest of her life. The seriousness of such of such a decision, as Alex demonstrates, seems to be undermined by the time of the clinic, where...' You only have '5 minutes to discuss an illness that's going to affect the rest of your life'. The straightforwardness, or the assumed 'obviousness' of the relationship between the thyroid and pituitary gland is reflected in the time that a clinician will take to explain thyroid dysfunction and consequent treatment. Again this is an example where the biochemical model of thyroid disease cannot relate to how patients experience thyroid disease temporally. It assumes that this 'simple mechanism' can be quickly and easily remedied, even though such a treatment will be required for life, often after the experience of years of symptoms that have frequently been unidentified and undiagnosed.

As one study found (McMillan et al. 2006), the communication of the diagnosis and treatment of thyroid disease can have a substantial impact on patient satisfaction. This questionnaire-based survey found that half the patients who took part (N=138) reported negative experiences of treatment around the time of diagnosis. This dissatisfaction was centred on delays in diagnosis and prescription of T4 treatment and/or lack of information provided about the condition and treatment. The study concluded that more consultation time may be required initially when a patient is told that they have a condition that requires life long treatment. Therefore, the clash between the routine of the doctor and the crisis of the patient impacts on the quality of care patients feel that they receive. As Emily, a hypothyroid woman in her sixties explains:

'... and then I told him. I actually took him a list [of symptoms and questions], he was so rushed off his feet he really didn't give a monkeys, to be honest with you. So much for National Health..... and this time when I went in he was very perfunctory, 'Well it's alright, you're in the right dosage now. Fine, go to your GP test in a year...'

As Emily and also Alex can demonstrate, the temporality of thyroid disease is not only multiple, but in conflict. On the one hand the Consultant is

required to spend time with his patients in order to explain and reassure them about their thyroid disease and its treatment. On the other hand, he is also required to make sure that he sees as many patients during the clinic and so not keep them waiting in the corridor for too long. He is required to give both efficient and quality care, a task that, as Emily can attest to, is almost impossible and almost always ends in failure.

The measurement of the relationship between the pituitary and thyroid gland derives its authority from an individual's thyroid function being compared against biochemical reference ranges that denote particular levels of function obtained from an epidemiological population. The rhetorical robustness of such results deems them 'unequivocal'. Therefore, when they are related to the ambiguity of symptoms, they appear to be definitive because they provide a clear 'evidence based' explanation of symptoms. However, it is clear from talking to patients, such as Alex and Emily, the certainty of blood tests provide an awkward explanation of a disease that has, in their experience, been messy, nebulous and embodied through symptoms that have seeped into their everyday lives over a number of years. Therefore, blood tests seem an inappropriate means through which to explain and attend to thyroid disease because they are too far removed from this experience 'of not being quite right'. Moreover, as Alex claims, the clinical endpoint of this model – the prescription of synthetic thyroxin for life – seems crass, as it does not relate to the creeping temporality of the period leading up to diagnosis, nor the 'event' of being told that one has to take medication for life. The conflict of these two temporalities and the privileging of the biochemical model of thyroid disease in current clinical practice guidelines, means that patients are not only disconnected from the explanation of their symptoms, but also their treatment. The biochemical model of thyroid disease does not put them at the centre of care – they are not treated, their blood test results are. As Alex reflects, when discussing a private doctor she went to see, and who prescribed her additional T3 to supplement the standard T4 replacement:

'Because the thrust of it is, a doctor who's practicing and who is, who suffers, (I think he's over, over or underactive himself,) one of the key issues that he fought against is the way people are badly treated in this country when they've got a thyroid complaint, and that they are very badly dealt with. The thrust of it is, why they're badly treated is because, one of his answers is that the doctors are quite blinkered and they say, 'here are your blood results and you must be feeling like this because that's what your results say. They're not actually listening to you.

And I know that from other people that have said, 'Well I'm feeling terrible, but my blood results are great and therefore I must be feeling great and that's not the case'. They're treating the blood result and not the person.

The decentralisation of their personal experiences, in the diagnostic and treatment process, goes against current idioms of patient centred medicine that are espoused by health policy initiatives, such as the 'Patient Choice Initiative' (The Department of Health 2006) in the United Kingdom. Consequently, in terms of the rhetoric of patient choice, Alex and Emily are not receiving good care because all choice, or more specifically, the opportunity to be included and relate to clinical decisions made about their care, does not occur due to the mechanistic and straightforward character of the biochemical model of thyroid disease. Hence this 'lack of care' or to be more specific, the subjugation of symptoms as evidence in order to pin down thyroid disease into an acceptable evidence based object, causes some patients to reject the biochemical model of thyroid disease. The inability of such a model to account for individual patient experiences leads some dissatisfied patients to redress this imbalance through the construction of an order of thyroid biology and pathology that can account for their individual experience of symptoms. Perhaps, unsurprisingly, such constructions draw on current rhetoric of patient centeredness and patient choice that permeate contemporary health policy discourses. However, disciplining of thyroid disease through the idiom choice, can also cause problems for its diagnosis and treatment.

As Annemarie Mol claims in her recent book *'The logic of care: health and the problem of patient choice'* (Mol. A 2008) that although the inclusion of patient choice is a widely celebrated ideal, it can actually jeopardise good care rather than ensure it. As Mol goes on to demonstrate through an ethnography of a Dutch diabetes clinic, and by progressing the conceptual work developed in *The Body Multiple*, bodies with disease are impossible to control - they are often unpredictable and erratic (Mol 2008: 12). This characterisation of disease is confirmed by the data collected by Mol and by this study of thyroid disease. Moreover, it is one that is also recognised by health care workers. For example, as the Consultant reflects in the introductory quote to this chapter, there are different and competing orders of thyroid disease, some of which fall outside of the explanatory models and technologies that have been developed to attend to them. This

approach to thyroid disease is what Mol calls the 'logic of care' (2008: 2) as Mol states:

The care that I will come to talk about, is not opposed to, but includes, technology. And the technology that I will come to talk about is not transparent and predictable, but has to be handled with care (2008:5)

However, this understanding of disease is eroded by what Mol calls 'the logic of choice' that characterises current approaches to health care. The injection of choice into health services means that the language of the market is mobilised: patients are customers and diseases are products that consist of, for example, expected levels of kindness and attention (IMol 2008:18). As Mol claims, the problem with interpreting diseases as products is that the often erratic and unpredictable course – or process - of disease cannot be accounted for. In the context of thyroid disease, the transformation from process to product is even more acute: it responds not only to the patient consumption of health care, but also to another order of knowledge - the standardisation of medicine, specifically the 'biochemicalisation' of thyroid pathology.

On the one hand, patients such as Alex and Emily are using the idiom of choice that saturates NHS health service delivery (Wanless 2004), asking for care that is tailored to them as individuals. At first glance it is an odd thing to criticise, as giving patients the choice to select treatments that are appropriate for them as individuals appears to positively democratise health care delivery and transforms patients from passive objects of medicine into subjects who have a right to make choices about their own health. However, in order that patients can choose an intervention that suits them, thyroid disease and its treatment needs to be pinned down – and so transformed into a product - and yet the ambiguity of thyroid disease evades such definition. Thyroid disease 'as a product' therefore directly contradicts and clashes with its ambiguous character, and leaves Alex and Emily with a set of expectations that cannot be met. It is sometimes impossible to predict if the treatments prescribed for thyroid disease will alleviate the many non-specific symptoms of the disease – symptoms that may or not be caused by thyroid dysfunction in the first place.

In addition, the purpose of the biochemical model of thyroid disease is to eliminate subjective accounts of disease and transform thyroid pathology

into a clean evidence-based object. The idiom of standardized medicine, specifically its ability to provide an objective account of thyroid disease, contradicts the choice model of health care. The choices patients could make about their disease management are removed by the over-standardisation of thyroid disease. The grey areas that are apparent between forms of evidence and the diagnostic categories of disease, are glossed over and ignored in order to provide a clear biochemical identity of the thyroid that is robust enough to be applicable across the general population and that results in a standard treatment regime that can be implemented in a uniform manner across the health service. Subsequently, the disciplining of thyroid disease by order of patient choice and standardization, creates two competing accounts of the disease, neither of which can fully allow, or account for, the ambiguity of thyroid disease.

If we return to the competing temporalities of thyroid disease, the constructions of an alternative and 'symptoms and person first' order of thyroid biology and pathology can be observed. For dissatisfied patients, whose thyroid function falls on the borders of the reference ranges, it is claimed that the measurement of TSH does not represent the action of their thyroid hormones. As outlined in the previous chapter, many believe that the relationship between biochemical measures and symptoms is questionable because the model is only looking at a 'snapshot' of function. I.e. only how much TSH and T4/3 is being produced at the time of the blood test and not how thyroid hormones are being utilized at a cellular level for a sustained period of time.

As introduced in chapter five, Thyroid U.K. is a patient organization that campaigns on behalf of people who are experiencing symptoms of thyroid disease, but whose blood test results are 'normal' or 'borderline'. They also provide support for individuals who still suffer symptoms after treatment. They claim, with regard to thyroid function tests, that:

With all of these tests, your results could be anywhere within the range and you would be classed as 'normal'. If you are at the very edge of the range, either at the bottom or at the top, you could be classed as 'borderline'. Neither you nor your doctor truly knows what your normal is, if you did not have a blood test done before you became ill. There are also particular reasons why the blood tests remain in the normal range. If you are not converting from T4 to T3 or if your cells are not taking up the T3 normally, your T4 levels and your TSH levels will still show as normal. (Thyroid U.K)

This rehearses a more general disparity between the individual experience of thyroid dysfunction and the population-derived reference ranges that are commonly used to define thyroid health (by defining the action of their thyroid hormones at a cellular level, i.e. how they are utilised (or not) once they have either been produced by the gland or supplemented via replacement thyroxine). The problem identified by Thyroid U.K. and other such groups can be broadly interpreted as the inability of the biochemical model to fully account for the functions of the thyroid beyond the thyroid pituitary axis. Not only, as Thyroid U.K. claim, is this model unable to relate to symptoms, but it also excludes the action of thyroid hormones once they have been produced/supplemented. Therefore, for dissatisfied patients and sympathetic clinicians, the issue of thyroid function has two dimensions – production and consumption. As Thyroid U.K. state in the 'why blood tests go wrong' section of their website:

...All these tests are subject to errors which have to be carefully borne in mind when interpreting them:

The blood tests themselves are not sensitive enough and each laboratory that undertakes them uses different methods and may have different reference ranges.

They represent a snapshot of levels of thyroid hormones in the bloodstream which are subject to daily, even hourly, variation, so that the time of day and circumstances of the test may cause inaccuracies.

In hypothyroidism the circulation is slowed to a variable degree, interfering with accurate estimation.

In hypothyroidism the blood is subject to a degree of concentration, also resulting from the slowed circulation, and this has the effect of raising blood levels above their true figure. Most important is the slowed T4 to T3 conversion, together with the slowed uptake of T3 into tissues affected by low metabolism, so that the mechanisms within the cell to aid the passage of thyroid hormones into the cell are damaged. The action of the cellular power source, the mitochondrion, is similarly slowed. This means blood levels may be raised because thyroid hormone is not being used in the normal way. Many doctors lack the basic training in thyroid medicine to interpret the results of the tests correctly. Frequently they hope that the laboratory results will do the interpretation for them; but without a full clinical history, and perhaps other tests available, the interpretation may be wrong.' (Thyroid U.K: 2007)

The broad criticism made by groups such as Thyroid U.K. against the standard thyroid function blood test is of its inability to place the disease in context and interpret it as a process - more specifically, as a process that conforms not only to their biological theories of thyroid function, but also to their temporal experience of symptoms. For example, as outlined above, blood tests only provide a 'snapshot' of thyroid function where there is a 'concentration' of thyroid hormones in the blood and do not necessarily reflect the true 'circulation' and 'action' of thyroid hormones in the body. This snapshot, it is claimed, could be the reason why they have symptoms in spite of euthyroid blood test results i.e. there is something else going on with the thyroid and thyroid hormones that is not picked up by this 'snapshot test'.

Thyroid urine tests are an alternative means of measuring thyroid function that may better provide a 'full picture' than blood tests. Alternative thyroid practitioners and dissatisfied patient activists consider thyroid urine tests to be more reliable because they measure the consumption of thyroid hormones over time (24 hours). As described on the IWDL Genova Diagnostics website (a private testing laboratory endorsed by such dissatisfied patient groups), the thyroid urine test is useful because:

The diagnosis of hypothyroidism is usually made almost exclusively from measurements of Thyroid Stimulating Hormone (TSH) and Thyroxin (T4) levels found in blood tests. However this method is thought to be largely ineffective at diagnosing cases of milder hypothyroidism, more accurately termed 'thyroid dysfunction'. Laboratory blood test techniques give information only about the hormonal status of a patient at a particular point in time. The elevation of hormone levels in urine, however, assess tissue exposure to thyroid hormones over a 24-hour period.

The urine thyroid test therefore serves as a valuable tool for detecting thyroid dysfunction that may otherwise go undetected through standard blood tests. It is important to use this test as an adjunct to other indicators of thyroid function, such as body temperature, symptomology and standard blood thyroid tests (IWDL Genova Diagnostics 2006)

This test, therefore, embodies a biological order of thyroid disease that relates to the idiom of patient choice. It reflects their individual embodiment of disease and provides an explanation for their disease as they experience it. It is a model that can account for those elements of the formal

biochemical model of disease with which such activist groups are dissatisfied.

Whilst these fringe practices claim greater authority through deeper understanding of thyroid hormone action, their approach is not practiced by the established clinical thyroid community, including the clinic that was observed for this study. Within the clinic and the pressurized environment of the NHS, only 'the proven' evidence-based methods of diagnosis and treatment are used. As the Consultant pointed out when talking about such alternative diagnostic and therapeutic approaches to thyroid disease:

So, in the current era of evidence based medicine, it is actually very difficult... you would not get a chemotherapy drug on that basis. You wouldn't start a new [unclear] breast cancer prophylactics or a new heart attack drug. So if you apply the same principles that we have to everything else, then just because one or two people report feeling better, I'm afraid, that doesn't stand up.

The Consultant calibrates and coordinates thyroid disease to the time of the established model of the thyroid pituitary axis – in spite, as he admits, of the fact there may well be a lot of 'silent stuff' going on that such a model cannot account for. However, as demonstrated above, there is an alternative order of thyroid disease that is also bought into the clinic. This order harnesses a version of thyroid biology which has a temporal narrative that relates to the quality of life and health expectations of individual patients. Moreover, it harnesses the possibility of the 'silent stuff' that maybe occurring and uses it as a further justification that supports their order of thyroid biology.

The practice of these thyroid biologies, in Timmerman's words, are foreshadowed by trajectories that are 'deeply clinical, social, therapeutic, iatrogenic, political, and bureaucratic.' (Hass and Timmermans 2008: 7) The ambiguity of thyroid pathology is made available to medical knowledge and intervention by stabilising it biochemically, an approach which is in turn influenced by attempts to standardise and rationalise medical practice. In response, patients for whom this formal model is inadequate, deploy the idiom of 'patient choice' embedded in current healthcare policy initiatives (also ironically intended to rationalise the health service) in order to have their disease understood through a biological order which relates to their individual experience of the disease. The cooption of this market driven approach to healthcare means that these dissatisfied patients (intentionally

or not) become customers who demand a product (diagnosis, treatment, level of service) that addresses their individual needs. Subsequently, the biology of thyroid disease becomes intertwined with these particular social arrangements. Because it is vague and ambiguous it provides a context where the negation of these competing orders is possible. Thus thyroid disease, is again revealed as a weak context for medicalisation, or specifically, biochemicalisation, because its biology is so non-compliant.

In the following section, these various orders of thyroid disease will be discussed in terms of the symptoms of the disease and their medicalisation and de-medicalisation in relation to thyroid pathology. It will be demonstrated that the tensions between these varied orders are such that their negotiation is often fruitless and fails to co-ordinate an approach to identifying and treating thyroid disease that is workable.

Medicalising and De-medicalising

The process of de-medicalising and re-medicalising certain types of evidence is an aspect of thyroid disease most apparent within the practice of the clinic. In the clinic, symptoms are frequently de-medicalised and deemed the result of something other than thyroid disease. This is especially the case for hypothyroid (both primary and post RI) individuals who complain of the persistence of symptoms in spite of thyroid biochemistry that falls within the reference range. In similar cases encountered as part of this study, the Consultant would simply confirm that the blood test results were within the reference range and that any continuing symptoms were not caused by thyroid dysfunction. For the Consultant, treatment outside of the parameters set by the thyroid pituitary axis was thoroughly unethical. However, he admitted to adjusting the position of the TSH within the reference range in order to provide treatment with a placebo effect (i.e. He moved TSH levels up to the high end of the reference range by administering thyroxine because some patients believed that this higher end of the reference range made them feel better). However, the Consultant was adamant that there were limits to this practice of 'fooling a patient'. As he explains:

The problem with the placebo effect, you might say well, why don't we just use it then is that it's, it's not sustained. So, if you have a bad relationship with your husband and you attribute

that to your thyroid status, if you correct that thyroid status and you believe this will improve. If you have a bad relationship with your husband, a year or two later it's going to reappear so I just think there's something fundamentally wrong in a sense, fooling a patient for not making them address what is the issue. Why someone should be told that actually, you know it's not their thyroid really, but here's some T3 and it will get better.

Therefore, although he is manipulating the level of TSH by prescribing a little extra thyroxin he is doing it to make biochemical measures of thyroid function more convincing. These adjustments still do not mean that the patient's thyroid function falls outside the reference range. Consequently, in spite of this manipulation the Consultant can still be considered to be practicing ethically and within clinical practice guidelines. However, this practice also indicates that there is something inadequate about the biochemical model. That is, if there is no discernable difference in symptomatic presentation between patients whose thyroid function fall at different points within the reference range, and the placebo effect is unsustainable, why does the consultant engage in this practice? Moreover, by deploying this tactic isn't he in fact treating patient preferences and beliefs and not thyroid pathology? It is impossible and un-helpful to suppose why such practice occurs. However, what this vignette demonstrates yet again, is the slippage between biochemical and symptomatic orders of evidence - the indeterminacy between and the impossibility of making these two orders account for each other. As a consequence, it is perhaps no surprise that patients find such evidence frustrating and nonsensical. In spite of claims to the otherwise it is clearly not definitive as it is manipulated in multiple ways through the course of diagnosis and treatment. Therefore, although the rhetorical models of thyroid biology and pathology presented by the professional clinical organisations and affiliated patient groups (e.g. The BTA and BFT) and dissatisfied patients groups and sympathetic clinicians (e.g. Thyroid UK, The TPA, Dr Barry Durrant-Peatfield and Dr Gordon RB Skinner) have the persona of neat ordered entities, they are in fact borne out of the very ambiguity and indeterminacy of thyroid disease and its associated evidence. They are *attempts* to consolidate the ambiguity of thyroid disease, that draws on the available discourses of the clinic (for example EMB and patient centeredness), and transform it into an object that can be intervened upon. They are what Mol (2002: 69), drawing on the work of STS scholar John Law (Law 1994), would call 'modes of ordering', mini discourses that are embodied and performed by patients and clinicians, and

that interact with each other during the course of navigating and acting upon thyroid pathology.

As has been consistently demonstrated throughout the thesis, such attempts often fail and can never quite fully account for the non-compliant biology of thyroid disease. As a result orders of thyroid biology 'do the best they can' to explain and develop strategies to attend to thyroid disease. This can be seen in another vignette provided by the Consultant in an interview. In the following extract he describes a consultation in which a post RI woman complains of symptoms despite receiving a maintenance dose of thyroxin that has rendered her thyroid biochemistry within the reference range:

I saw a girl privately last week who feels a lot worse now with the normal thyroid function than she did when she had Graves disease. So all I could say to her was that there were psychological issues. But even then, her test was normal, and it can be up to a year before you feel like that [normal]. She clearly was having panic attacks because I did a breath challenge and she went all panicky and that reproduced the symptoms. So there was a clear anxiety issue. She was young girl in her 30s, single; I didn't quite know what was going on. But there's quite a lot of psycho-pathology around as well, so she wasn't one of life's copers at this point and, um, and it's when you say 'well just go on a holiday', you know there were lots of reasons why she couldn't do that...I'm thinking well these people are just... it's just desperation for a physical label. People aren't prepared to accept psychological diagnosis. I mean, with thyroid disease I don't think we do it very well but with diabetes we have a psychologist on board and I would say, whenever I send anyone she would say 100%, a little depressed so, and if you look at Addison's disease, which is another hormone deficiency, steroid hormone, if you do quality of life test they're twice as bad as the general population.

This negotiation of orders of potential intervention involves what Mol would call the 'calibration' of thyroid disease to the thyroid pituitary axis. That is, the Consultant attributes the continuing psychological symptoms to a 'psycho-pathology' which is present in spite of normal blood tests and, which is therefore, a separate issue to the thyroid disease (that is 'no longer present'). However, the Consultant's account also reveals a significant amount of uncertainty as to what is actually 'going on'. The de-medicalising and re-medicalising of psychological symptoms thus calibrates this woman's thyroid disease to the thyroid pituitary axis only in part. As the consultant describes, all he could tell her was that there 'were psychological issues' but that even then 'he wasn't sure what was going

on'. The ensuing 'desperation for a physical label' demonstrates not only feelings of stigmatization bought about by a psychological label, but also the failure of the model of the thyroid pituitary axis to provide a physical or biological label. As the Consultant's account demonstrates, diagnosing and managing thyroid disease is fraught with uncertainty and ambiguity. The indeterminacy that is left by the model of the thyroid pituitary axis means that the Consultant's expertise is questioned, not only by patients, but also by himself. In this instance, the Consultant relays the limits to the certainty with which he can calibrate symptoms to thyroid disease.

The medicalisation, specifically, biochemicalisation of thyroid disease is also shown to be problematic when treating patients with biochemistry outside of the reference range, but who display no symptoms. In contrast to the 'problem population' of those who claim an under-active thyroid gland through the manifestation of symptoms and borderline biochemistry, this population has the disease but very few symptoms that are considered to be perceptible and/or problematic. As Beth, a Graves patient in her mid-twenties, explains:

You know, I just, I didn't, I just thought it was me. Because I am a bit manic. I am a bit all over the place. I've always been like that, um, a bit kind of like oh, about doing ten things at one time. But I think that feeling unwell on the tube, I just attributed it to me running late and it being hot. But I never really thought that, I didn't think anything of it, the sweating and that kind of thing, I've got to get out of here, like, because my heart's beating really fast. And every night I was, kind of, finding it really hard to get off to sleep because it was always at night that it was worse. So, it all kind of started to fit into a bit of a picture, that perhaps I'd acclimatised slightly to, or I just attributed it to just normal stress and normal stresses of everyday life.

Furthermore, the Consultant explains, and as evidenced by the Colorado Disease Prevalence Study (Canaris et al. 2000) discussed in chapter five, sometimes there is little rhyme or reason as to why some people are symptomatic and others are not:

Basically all of us are miserable and most of us are gaining weight, most of us have dry hair, losing hair, dry skin. You know, they're all things we all have and just because your TSH is four, five, three, two or one doesn't really make much...I have seen people with a TSH of more than 100 that feels fine so, you know...

Again, this Consultant acknowledges the weakness of the biochemical model of thyroid disease: he claims that thyroid function tests were originally developed to locate the thyroid pathology underlying thyroid type symptoms. By stating that 'I have seen people with a TSH of more than 100 that feel fine so, you know...' is the Consultant acknowledging that the link between thyroid biochemistry and thyroid disease can be as uncertain as the link between symptoms and thyroid disease. However, even though he is aware of this disparity, he perseveres with the coordination of thyroid disease to the biochemical model because it is the only one available to him. Given that he is not prepared to offer treatment that is contrary to the guidelines, he has no other options. Although he is aware that the current tools used to diagnose thyroid disease may be insufficient for some patients, there is no evidence-based alternative currently at his disposal. He goes on, then, to suggest how little he and his colleagues are able to do for patients who are within the biochemical reference range of healthy thyroid function yet whom complain of symptoms:

So I think that, you know, as my, I've got a very, the top thyroidologist in America and I was at a meeting with him last week and I said what do you do when a person with a TSH of 1.5 on Thyroxin has symptoms of hypothyroidism? He says there's nothing I can do.

The pressure brought about by the insufficiency of the thyroid pituitary axis model is heightened by the dissatisfied patients who take information about alternative biologies of thyroid disease to the clinic. As the Consultant explains, in cases such as this he feels cornered:

Maybe there's a way of dressing it up, but actually I'm not sure. I mean, usually in that sort of consultation I'm thinking if I get away without a complaint here I've done well. With that sort the focus is on escaping alive and you know, I know I'm never going to help them without even saying anything...

The Consultant's desperate wish to 'escape alive' is caused by the response of dissatisfied patients to the inertia of the biochemical model of thyroid disease when it is applied to their order of thyroid disease. As discussed in the previous chapter the lack of an explanation for their symptoms causes distress and a worsening of their symptoms, even the development of new symptoms related to emotional and mental stress. For instance, as Diana Holmes' petition to the Society of Endocrinology states,

current established treatment strategies are considered to constitute 'emotional abuse' by the medical profession. Moreover as the TPA claim on their website:

Medical practitioners shelter themselves behind test results, which are routinely used to claim more than they test. They refuse to listen to their patients or even credit the evidence of their own eyes, leaving their patients to remain ill for the rest of their lives. It is because of this disgraceful state of affairs that TPA-UK was founded (Thyroid Patient Advocacy UK 2005)

Therefore, at the point where the biochemical order of thyroid disease cannot provide an evidence-based alternative to explain symptoms, in spite of normal biochemistry, and symptoms are unacceptable evidence in terms of evidence based practice, both dissatisfied patients and clinicians, who feel they are under attack, fill this gap with their own modes of ordering. That is, the nature of thyroid disease is so anomalous the patient-expert divide becomes to some extent, symmetrical, as both sides are frustrated with the inadequacy of approaches to thyroid disease and attempt to construct alternative explanatory models that are relevant to them. The result of this failure to coordinate thyroid disease to particular biological and medical orders means that negotiation between these orders becomes impossible. This failure generates a cycle of blame in which these orders become increasingly dogmatic and oppositional.

Pathological Blaming

As emphasised in chapter five and again in this chapter, the evidence that constitutes the diagnostic categories of thyroid disease is indeterminate and uncertain, and as a result that diagnosis can become a stressful and conflictual process, but also, as I have suggested, *itself a source of suffering*. This is possibly the most important reason why 'thyroid disease' cannot be understood as a neutral and singular pathological fact, but is arguably more usefully understood as a set of biosocial orders that dynamically relate to each other. Returning to Rabinow's concept of biosociality introduced in chapter two, – it is possible to suggest that dissatisfied patients and clinicians engage in what Mol would call 'modes of ordering' thyroid biology and pathology through particular evaluations and deployments of evidence. However, in contrast to the original

formulations of biosociality, such practices are not a result of new found molecular control over biology. They are a result of a lack of technique and control. 'Modes of ordering' thyroid biology are, therefore, an attempt to make sense of the explanatory gap that characterises thyroid disease through the fulfilment of an array of rights, desires, needs and responsibilities, such as: choice over health care intervention, a symptom free life, the ability and responsibility to practice within evidence-based guidelines, that are to hand within the governmental discourses of healthcare in the United Kingdom. Consequently, the 'local biology' of thyroid disease is not geographical, but located around the non-compliant biology of thyroid pathology, which eludes all attempts at standardisation (either through biochemistry and evidence-based medicine or symptoms and patient centred medicine). Specifically, the 'local biology' of thyroid disease is one of non-compliance that eludes any straightforward 'medicalisation', 'biochemicalisation' or more generally standardisation.

Thus such biosocial ordering demonstrates that thyroid disease provides a weak context for medicalisation, because such orders are in part the result of a symmetrical relationship between doctors and patients, bought about by the ambiguity of thyroid disease. That is, due to the indeterminacy of the disease, both clinicians and dissatisfied patients can produce convincing accounts of its biology and pathology. However, as has also been demonstrated, both of these accounts are partial. Thus the power to define and discipline the disease is highly dispersed, but also, ultimately elusive. As a consequence such modes of ordering do not result in a smooth process of negotiation and eventual treatment. Sometimes such biosocial orders are so contradictory, negotiation between them and attempts to fill the thyroid diagnosis and treatment gap fail. Specifically, the trajectories of patient choice and medical standardisation that foreshadow the thyroid clinic, shut down the ambiguity of thyroid pathology. That is, the politics embedded in these orders, and in the relationship between them, again makes it impossible to account for the ambiguity of thyroid disease. The insufficiency of their terms renders consultants 'lost for words' and patients dumbfounded, distressed and consequently instigates the further retreat into their preferred mode of ordering in the hope that an explanation may eventually be provided.

The inability of either order to fully account for thyroid disease results in the production of two rhetorical models around which dissatisfied patients, sympathetic clinicians and the established clinical thyroid community collect. The established evidence-based biochemical order relies on thyroid biochemistry to 'clear-up' the non-specificity of thyroid type symptoms and reach a 'definitive' and scientifically reliable diagnosis. On the other hand, the non-establishment 'symptoms first' order considers thyroid biochemistry to be poor evidence of disease because it cannot account for the symptoms in a sizeable minority of patients with borderline or normal blood test results, often even after 'successful treatment'. Subsequently, at this point these two orders can no longer be negotiated and they are transformed into two polarised positions around subjectivity/symptoms/choice and objectivity/biochemistry/standardization. Clinicians accuse dissatisfied patients, who question the established diagnosis and treatment strategies, of wasting time and having unrealistic health expectations. With equal vehemence, patients blame clinicians for being unsympathetic and practicing 'technocratic' medicine. The frustration that these extreme oppositional models reveal saturates the day-to-day business of the clinic. As the consultant describes his approach to 'difficult' patients: 'I know I'm never going to help them without even saying anything because I know what peoples' ideas are...'

So what are the implications of this cycle of blame when it is clear that these conflicting biosocial orders of thyroid disease all have some degree of 'truth'. Thinking back to Adriana Petryna's (2003) insightful response to her informants' accounts of health and disease in her ethnography of post-Chernobyl Ukraine, it is useful to recall her observation that the role of such studies is not to apportion blame, but to 'paint a clearer picture of the dynamic interplay between scientific and social orders, and how those orders come to define actual conditions of health' (2003: 12). For example, if we return look at what some of the dissatisfied patients say about their suffering, it seems to revolve around the feeling that they are never optimally treated and are left to struggle with residual symptoms on their own. For instance, as Briony, a hypothyroid woman in her late thirties, who feels that her thyroid function fluctuates in spite of consistent blood test results within the reference range, muses:

I guess I am sort of conscience of the weather. I'm thinking oh, what if it turns cold am I going to start suffering more because your body won't be able to respond as fast, it will be slower to

respond. I did, erm, we had a terrible long winter and spring, when it was just cold endlessly and I it did really feel that. I don't know if other people were moaning about it as much as I did but...

As Briony demonstrates, there is a constant worry about when symptoms will appear and if they are due to a disturbance in thyroid function. It is this uncertainty and consequent worry that seems to contribute to the feelings of ill health in such patients. This liminal state of thyroid disease therefore becomes the way in which physical feelings and sensations are analysed and expressed, as for example, feeling the cold becomes inextricably linked to the spectre of under-treated thyroid disease, as well as the changing of the seasons. This constant wrestle with such liminal and ambiguous categories of thyroid disease seems to seep into all parts of these patients' lives. For example, as the following quote from Briony demonstrates:

But I'm feeling incredibly tired, incredibly tired. I just know that even if I have 9 hours sleep, I had 9 hours sleep Saturday, you know Saturday morning I woke up after 9 hours and I just felt drained. Sunday I did the same and I just felt drained. And I know that I'm really struggling to get out of bed and that isn't, you know that it's just not working. If I have 8 hours sleep and I still can't get out of bed then I know it's not right. It's not right. Because I know that when I was a little bit overactive I would be ping, wide awake, after 10 minutes. I'm not a morning person, now maybe it's because of thyroid problem that I'm not. I know I never have been, but this morning I was just thinking, god I must try and get out of bed. Even after I've taken the tablets, to be honest, it was still like, I don't know why. The chopping and changing of the weather, maybe that's got something to do with it, because you're body's got to constantly adjust and readjust 'coz, oh, it's one minute it's hot, next minute it's cold, next minute its hot. So that could be something to do with it. I don't know, but I'm not, I'm really not right at the moment. But it's surprising, given that after taking the same as I was when I was slightly over.

In this interview extract the focus on thyroid disease is expressed through the amount of sleep Briony gets and feels she needs. She is constantly questioning herself. Does she feel tired because of the symptoms of under-treated thyroid disease or is it because she's just not a 'morning person', she never really has been after all? But is this in itself because of her thyroid problem? Or is it because of 'the chopping and changing of the weather?' Ultimately this line of questioning ends as many explanations of thyroid disease do, with utter uncertainty. As Briony concludes, 'I don't know, but I'm not, I'm really not right at the moment'. Therefore, this extract demonstrates the ambiguity and frustration that surrounds the definitions of

thyroid health actually contributes to the ill health of patients who find themselves in this position. Specifically, as demonstrated by Briony and throughout the preceding two chapters, thyroid disease provides a context where the imperative of scientific discourses to standardise, has failed. Moreover, what results is not a coherent discourse of power but a multiple and discordant discourses of power, or 'modes of ordering', that attempt to muddle through the unusual landscape of this discordant pathology.

In conclusion, it is clear there are no broadly applicable terms of thyroid disease. The thyroid pituitary axis, which has been developed in an attempt to establish the boundaries of thyroid health, is constantly undermined by the indeterminacy of the evidence it generates (blood tests results), but also, the indeterminacy of its relationship to symptomatic evidence. Therefore, the deployments of particular evidentiary models of thyroid disease can perhaps be best understood as the choreographing and coordination or 'modes of ordering' of these ambiguous forms of evidence through a set of complex micro-practices. To be precise, thyroid disease can be defined as a range of 'attempts' that 'do the best they can' to develop a satisfactory model of disease, through the management and evaluation of the available, albeit partial and ambiguous, evidence.

As has been described, biochemical and symptomatic evidence is coordinated to particular temporalities in an attempt to prove their intrinsic authority and justify specific diagnostic and treatment strategies. On the one hand, the thyroid pituitary axis, and as a consequence a blood test result, is considered to a better and more reliable source of evidence because it can be extracted from the subjective and social milieus of the illness experience. The straightforward cause and effect narrative of this 'simple' modulated mechanism is lacking in time and as a consequence can side-step the subjective nature of individual patients narratives and get at the 'real' thyroid pathology contained within the gland. On the other hand, for dissatisfied patients and sympathetic clinicians, symptomatic evidence is more authoritative *because* it can relate to narratives of symptomatic experience. That is, the truth of symptoms is derived from their ability to coordinate with the subjective and social milieus of the illness experience.

Therefore, the logics contained in these orders are polarised and often mean that they and their protagonists are unable to relate and negotiate with each other. For instance, it became clear that the simple straightforwardness of the biochemical model espoused by the Consultant, often left patients with an unsatisfactory explanation of their thyroid disease and a feeling that the care they were receiving was inadequate. That is, his simple explanation did not do justice to their experience of suffering and the prospect of having to take thyroid replacement therapy for life. In response, the Consultant was at a loss about how to respond. He was offering them a clear 'evidence based' explanation of their disease and 'subsequent' proven and 'effective' treatments. What more could he do? The failure of these two orders and groups to relate to each other and feelings of dissatisfaction are most effectively shown by the practice and results of medicalising and de-medicalising symptoms. As described, the Consultant only attributed symptoms to thyroid disease when biochemical evidence was present. However, he was often left flummoxed and troubled by the continued complaints of patients. He became lost for words because the biochemical model he deployed could not explain this gap, and nor could, as far as he was concerned, the un-substantiated biomedical theories presented by dissatisfied patients and sympathetic clinicians. As a consequence, it seems to be the case that the suffering that thyroid disease produces is, in part, a result of the inability of either of these orders to fully account for it.

In spite of the production of technologies such as blood tests and clinical practice guidelines, attempts to standardise thyroid pathology have failed. It is clear that both patients and clinicians are aware that the tools through which they attempt to understand thyroid disease are inadequate. However, in the absence of anything else they muddle through as best they can in order to attend to the disease. What is being maintained through this process of 'muddling through', therefore, is not a rule driven model of disease constructed by medical authority, but a collection of dispersed practices that gather around and attempt to intervene upon this non-compliant pathology. That is, the 'local biology' of thyroid disease can be characterised as a range of often unsuccessful attempts by clinicians and patients to medicalise, standardise or more accurately 'biochemicalise' and 'biologise' the non-compliant biology of thyroid disease that results in varying degrees of success.

Chapter 7

Conclusion: making biology meaningful and the management of disease

Well, I can't understand. I mean, I can't believe I've made a career out of it. Because I mean, a lot of the people I trained with just take the piss. It either goes up or it goes down [thyroid function][...]They can't believe what goes on. But I think if we were in the Second World War, I don't think we'd be worried about a TSH of 5.5. Consultant, weekly outpatients' thyroid clinic

As the consultant reflects above, treating thyroid disease should be simple. If thyroid function becomes too low and the production of thyroid hormones is insufficient, thyroid replacement therapy should be prescribed. If thyroid function becomes excessive, the gland should be 'shut down' with a dose of radioactive iodine and thyroid replacement therapy should be prescribed. However, the thyroid does not just go up or down – it can sit on the borders, or leave the realm of biochemistry altogether and seep into subjective assessments of health. It may even be a far more complex pathology than current 'evidence based' diagnostic tools and treatment strategies can account for. What is clear is, due to the complex relationship between the indeterminate aspects of thyroid disease, the ability of the medical profession and patients to control and/or discipline it, is limited and elusive. Even the Consultant, who has observed thousands of patients with thyroid disease through the course of his career, questions his expertise, due to the continuously non-compliant nature of thyroid pathology. On one hand, he 'can't believe' he has a 'made a career' out of such a simple and straightforward disease. On the other, his colleagues 'can't believe what goes on', and 'take the piss' out of how such a simple disease is so difficult to manage. The Consultant attempts to explain this situation by inferring that thyroid pathologies themselves have not changed. They are as straightforward as they have always been, but what has changed, is patient expectations of health and the health service. By suggesting that 'if we were in the Second World War, I don't think we'd be worried about a TSH of 5.5', he is inferring that the increased health awareness of the population is detrimental to the delivery of health care. To be more precise, as the endocrinologist Dr Anthony Weetman claims, there is the suggestion that:

[...] we [clinicians] are practising in the age of post-modern medicine. A cardinal feature of post-modernism is the derogation of objective facts, which are the defining characteristic of science and the replacement of scientific certainty with the view that reality can have multiple meanings. (Weetman 2006: 231)

Hence, patients, often through access to information found on the Internet, will refuse to believe what doctors tell them, will claim they have thyroid disease and ask for subsequent treatment strategies, for which, as yet, there is no sound evidence base.

As has been demonstrated at points throughout the thesis, biomedical knowledge about thyroid disease is by no means comprehensive and certain. Using the praxographic approach, developed by Annmarie Mol, and through an analysis that charts how formal biomedical knowledge (contained in documents such as clinical practice guidelines) interacts with the practices of the clinic and the bodies of patients, it becomes clear that thyroid disease is at times singular and straightforward, but at others, multiple and complex. For example, genetic predispositions to thyroid disease are well established, yet almost tangential to their diagnosis and treatment. A goitre is a 'classic' physical sign and symptom of thyroid disease, but can also be considered a 'normal abnormality' that does not necessarily impair thyroid function. Some patients found that their disease could be sufficiently explained and treated through the application of the biochemical model of thyroid function. However, others found that this model did not represent their thyroid disease sufficiently, and as a consequence not only failed to alleviate their suffering, but increased it through the frustration and distress caused by the failure to define and attend to it through the methods currently favoured within the NHS.

It can be argued, therefore, that the standardisation of thyroid disease through biochemically, circumscribed states of normalcy and pathology, is a successful strategy of intervention. It clears up the ambiguity of thyroid disease by disciplining it into a clear-cut set of diagnostic entities that can satisfy the requirements of EBM. Specifically, it provides a population-based category of disease that ensures that treatment is only given to patients who can be diagnosed with certainty, or to be more precise, certainty derived from rigorous scientific methodology. However, this 'mode of ordering' thyroid disease is also insufficient. The certainty that is

apparently derived from biochemically ascribed categories of thyroid disease, when investigated qualitatively, is at times highly uncertain and results in unsatisfactory treatment outcomes. In particular, the population-based biochemical reference ranges, used to denote states of thyroid function, cannot account for the expression of thyroid pathology, specifically the experience of symptoms, in all patients. Often patients who fall on the borderline between healthy and unhealthy thyroid function and who experience symptoms, or those who have received treatment and also still experience symptoms, are not *dysfunctional enough* in terms of the tenets of standardised care. In particular, their experience of symptoms does not satisfy the evidentiary requirements of the biochemical mode of ordering thyroid disease, because symptoms are considered to be *anecdotal and non-specific* and therefore unreliable. Consequently, such patients inhabit a liminal category between health and disease; they can demonstrate the 'classic' clinical symptoms of thyroid disease but not the biochemical evidence that is needed to justify treatment within the contemporary NHS, espoused in clinical practice guidelines (Association for Clinical Biochemistry et al. 2005).

The limits of the biochemical mode of ordering ironically provide a location through which dissatisfied and frustrated patients can potentially overcome their unsanctioned experience of thyroid pathology and suffering. As described in chapter five, such patients draw on the palpability of their symptoms and current rhetorics of patient-centred medicine and develop an order of thyroid biology, pathology and treatment that can account for *their* biochemically, no-compliant thyroid disease. Therefore, the limits of the biochemical order of thyroid disease are revealed and re-ordered by such patients. Specifically, a mode of ordering is developed, where the hierarchy of evidence that privileges the results of thyroid function tests, over and above the symptoms, is reversed. In contrast to the biochemical mode of ordering, signs and symptoms are considered to be more an authoritative and reliable form of evidence *because* they are embodied by patients and can be clearly observed and touched by clinicians during the clinical encounter. That is, the embodiment of symptoms, far from making them unreliable, makes them undeniable evidence of thyroid disease. Consequently, patients who order their thyroid disease in this way are able to attribute their suffering to an underlying thyroid pathology and potentially negotiate treatment for their symptoms.

The strategic re-ordering of the dominant order of thyroid biology by dissatisfied patients and clinicians is particularly meaningful in terms of the concept of medicalisation. Irving Zola's (1972) original formulation of this concept can be defined as the process through which the normal aspects of everyday life and individual behaviour become placed under the control of medical expertise and subject to various biomedical and technical solutions. Therefore, the 'biologisation' (described in detail at the end of chapter five) of non-specific symptoms, by dissatisfied patients, is an interesting empirical addition to this concept. The non-specific symptoms of thyroid disease, that are also normal feelings and sensations we all experience at one time or another, are medicalised or more specifically 'biologised' by patients and transformed into 'irrefutable' evidence of disease. It is the authority and expertise of patients (their embodied experience of suffering) and subsequent re-ordering of thyroid biology and pathology, that transforms such non-specific symptoms into a medical problem. However, in contrast to the process of medicalisation, this process of 'biologisation' is practiced by patients in order to contest the formal medical model of thyroid disease and transform it into a pathological entity that fits with their experience of symptoms and desire for a symptom-free life. Specifically, the authority that enables this process comes not from the medical profession, but from patients who are dissatisfied and frustrated by the inadequacy of the standardised biochemical model thyroid disease.

As a consequence, this reformulation of the concept of medicalisation, through 'autobiologisations' preformed by patients, has interesting implications for the concept of biosociality and more generally, theories of governmentality. Rabinow's now famous claim that groups will collect around specific pathologies, identified by molecular biology, and gather together a multitude of experts in order to 'understand' their 'fate', (1992: 102) are in part a result of the ability to engineer our vitality, but also, reflective of what Rose calls the governmental technologies of 'advanced liberalism' (2006: 25), where the power of the state over the management of human health has devolved to the individual citizens and 'quasi-autonomous regulatory bodies' (Rose' 2006: 25). That is, hand in hand with the ability to maximise life and health, comes responsibility of citizens to maintain their health through practices of 'care of the self'. Governmental technologies, that 'regulate at a distance' (Rose. 2006: 25), through various

non-state regulatory bodies, for example, medical associations, or through tools such as clinical practice guidelines, are also an attempt to intervene upon and control the health of the population. In the classic Foucauldian sense, both of these governmental technologies share the same imperatives - the management of human health and reproduction - at the level of the individual and the population.

What is interesting, in the case of thyroid disease, is the rupture between these two abstract systems of governance. Although dissatisfied patients and sympathetic clinicians collect around and remake thyroid pathologies, (in an attempt to maximise health and act upon themselves through the language and practices of biomedicine), they do so in response to the technical deficit of current diagnostic and treatment strategies for thyroid disease. As described in chapters five and six, the indeterminacy of the evidence used to diagnose and treat thyroid disease, results in a number of practices, in particular the reversal of the hierarchy of symptomatic and biochemical evidence. This reversal, however, is in direct conflict with the evidence-based treatment strategies that are offered by the NHS and, as has been described, results in the polarisation of these two orders of thyroid biology. In this sense, the 'biologisation' that patients perform is both a form of biocitizenship, in the sense that they are attempting intervene upon their biology in order to optimise their health, but also an example of how these subjective health practices actually clash with other technologies of governance, i.e. EBM.

As I conclude, I would like to suggest that in the thyroid clinic, the assumption that 'the care of the self' is intrinsically and straightforwardly linked to the internalisation of broader neoliberal structures of governance, is an inaccurate description of what is going on. That is, although such patients are practising what Rose calls 'somatic ethics' (2006: 26), they are doing so in a way that is out of step with other forms of governance. Whilst dissatisfied patients attempt to optimise their health, due to the lack of control they and the medical profession have over thyroid pathology, they are doing so by drawing on a range of *uncertain* theories of thyroid disease. Therefore, in the thyroid clinic, the relationship between the governance of the individual and the governance of the population is far more stratified and unintended than theories of governmentality suggest.

The results of these various and often contradictory attempts to standardise and order the elusive and non-compliant nature of thyroid biology, is a situation where these competing orders end up 'muddling through', in attempts to reach some form of agreement and grounds for therapeutic action. That is, using and adapting Lock's term, the 'local biology' of thyroid disease can be characterised as a range of often unsuccessful attempts by clinicians and patients to medicalise, standardise, 'biochemicalise' and 'biologise' the non-compliant biology of thyroid disease. This troubled relationship between the individual and epidemiological plains of thyroid disease, often stabilises the traditional categories of power, in particular categories of gender. It is clear that the anecdotal evidence provided by the largely female population of dissatisfied patients, is often attributed to the character of the irrational, over emotional and 'hormonal' female patient (for example see Daggett: 2000). Thus, as a number of studies (Bharadwaj 2008, Gibbon 2008, Roberts 2008), and Rabinow (1996: 103), himself pointed out, biosocial practices are capable of reinforcing traditional categories, such as gender and ethnicity, as well as questioning and reformulating them. However, the latent uncertainty that remains, due to the current inadequate biomedical model of thyroid disease, also results in the continual undermining of this category and diagnostic categories of thyroid disease more generally. The uncertainty that remains is typified by the Consultant's underlying feeling of 'not quite knowing what is going on'.

This thesis has consisted of an investigation into the diagnosis, treatment and management of thyroid disease. In doing so it has described how this disparate, non-compliant 'biology' eludes any straightforward 'medicalisation', 'biochemicalisation' -- or to be precise, standardisation. In lieu of any successful standardisation, thyroid disease is a context of failed standardisation, in which procedures, speech, acts, documents and guidelines are relied upon, even though everyone knows they are inadequate. As a consequence of being under pressure to be coordinated to an effective and efficient clinical treatment system, thyroid disease becomes ambiguous and ambivalent, hit and miss, awkward, contested and disappointing for clinicians and patients.

In the introduction to this thesis I asked if there was something about the contemporary clinical encounter that made this well established, common and straightforward disease unnecessarily so complicated. Ironically, as I

hope I have demonstrated, what seems to have complicated thyroid disease are a number of strategies that compete with each other in their attempts to discipline this non-compliant biology, the health practices of patients and the clinical practices of clinicians. EBM and the more general audit culture of the NHS makes thyroid biology accountable. However, in doing so, it clashes with the patient-centred business model that also governs the NHS, as it often cannot satisfy the experiences of individual patients. The failure of both of these models to fully discipline thyroid biology and pathology creates neither an accountable nor enterprising approach to diagnosing and treating thyroid disease. Instead, what is left is a system where the gaps left by these competing rationales are muddled through by patients and clinicians in an attempt to reach compromises over health care. Therefore, it could be said that what has changed about the contemporary clinical encounter, embodied and revealed by the vagueness of thyroid disease, is the inclusion of a number of strategies to govern health, all of which jostle for position in the process of trying to reach a consensus. Thyroid biology and pathology is to some extent a 'matter of choice', but it is also a set of population-based biochemical reference ranges. It is the battle between these evidentiary sources and broader systems of governmentality that largely define the conditions of thyroid health and the health of NHS patients more generally.

References

- ADAMSON, C. (1997) *Existential and clinical uncertainty in the medical encounter: an idiographic account of an illness trajectory defined by Inflammatory Bowel Disease and Avascular Necrosis*. *Sociology of Health and Illness*, 19, 133-59
- ANGELL, M. (1996) *Evaluating the Health Risks of Breast Implants: The Interplay of Medical Science, the Law, and Public Opinion*. *N Engl J Med*, 1513-1518.
- ARKSEY, H. (1998) *RSI and the experts : the construction of medical knowledge*, London, UCL.
- ARMSTRONG, D. (1983) *Political anatomy of the body : medical knowledge in Britain in the twentieth century*, Cambridge, Cambridge University Press.
- ARMSTRONG, D. (1984) The Patients View. *Social Science & Medicine*, 18, 737-744.
- ARMSTRONG, D. (1995) The Rise of Surveillance Medicine. *Sociology of Health & Illness*, 17, 393-404.
- ARMSTRONG, D. (2002) Clinical autonomy, individual and collective: the problem of changing doctors' behaviour. *Social Science & Medicine*, 55, 1771-1777.
- ARMSTRONG, D. (2007) Professionalism, Indeterminacy and the EBM Project. *BioSocieties*, 2, 73-84.
- ARMSTRONG, D. & CALDWELL, D. (2004) Origins of the Concept of Quality of Life in Health Care: A Rhetorical Solution to a Political Problem. *Social Theory & Health*, 2, 361-371.
- ARMSTRONG, P. & ARMSTRONG, H. (1996) *Wasting away : the undermining of Canadian health care*, Toronto ; Oxford, Oxford University Press.
- ASSOCIATION FOR CLINICAL BIOCHEMISTRY, THE BRITISH THYROID ASSOCIATION & THE BRITISH THYROID FOUNDATION, (2005) UK Guidelines for the Use of Thyroid Function Tests.
- ATKINSON, P. (1988) *Discourse, Descriptions and Diagnoses: Reproducing Normal Medicine*, Kluwer Academic Publishers.
- BANKOFF, G. (1947) *The Conquest of the unknown* [S.I.], Macdonald.
- BANTING, F. G. & BEST, C. H. (2007) The internal secretion of the pancreas (Reprinted from the *Journal of Laboratory and Clinical Medicine*, vol 7, pg 251-266, 1922). *Indian Journal of Medical Research*, 125, A251-A266.
- BARRY, A., OSBORNE, T. & ROSE, N. S. (1996) *Foucault and political reason : liberalism, neo-liberalism and rationalities of government*, London, Routledge.
- BAUMAN, Z. (1991) *Modernity and ambivalence*, Oxford, Polity.
- BAYLISS, R. I. S. & TUNBRIDGE, W. M. G. (1998) *Thyroid disease : the facts*, Oxford, Oxford University Press.
- BECK, U. & RITTER, M. (1992) *Risk society : towards a new modernity*, London, Sage Publications.
- BECKETT, G. (2003) Thyroid function tests and reference ranges. *BTF News* 7.
- BELKIN, G. S. (1997) The technocratic wish: Making sense and finding power in the "managed" medical marketplace. *Journal of Health Politics Policy and Law*, 22, 509-532.
- BERG, M. (1995) Turning a Practice into a Science - Reconceptualizing Postwar Medical-Practice. *Social Studies of Science*, 25, 437-476.

- BERG, M. (1998) Order(s) and Disorder(s): Of Protocols and Medical Practices. IN MARC BERG AND ANNEMARIE MOL (Ed.) *Differences in Medicine: Unraveling Practices, Techniques and Bodies*. Durhan and London, Duke University Press.
- BERNARD, C. (1974) *Lectures on the phenomena of life common to animals and plants*, Springfield, Charles C Thomas
- BHARADWAJ, A. (2008) Biosociality and biocrossings: Encounters with assisted conception and embryonic stem cells in India. IN GIBBON, S. & NOVAS, C. (Eds.) *Biosocialities, genetics and the social sciences : making biologies and identities* London, Routledge.
- BORELL, M. (1985) Organotherapy and the Emergence of Reproductive Endocrinology. *Journal of the History of Biology*, 18, 1-30.
- BORELL, M. (1987) Biologists and the Promotion of Birth-Control Research, 1918-1938. *Journal of the History of Biology*, 20, 51-87.
- BOSTON WOMEN'S HEALTH BOOK, C. (1974) *Our bodies, ourselves : a book by and for women*, New York, Simon and Schuster.
- CANARIS, G. J., MANOWITZ, N. R., MAYOR, G. & RIDGWAY, E. C. (2000) The Colorado thyroid disease prevalence study. *Arch Intern Med*, 160, 526-34.
- CANGUILHEM, G. (1978) *On the normal and the pathological*, Dordrecht, Holland ; Boston, D. Reidel Pub. Co.
- CANGUILHEM, G. & DELAPORTE, F. (1994) *A vital rationalist : selected writings from Georges Canguilhem*, New York, Zone Books.
- CANNON, W. B. (1932) *The Wisdom of the Body*, pp. xv. 312. Kegan Paul & Co.: London; printed in U.S.A.
- CHISTIAKOV, D. A. (2005) Immunogenetics of Hashimoto's thyroiditis. *J Autoimmune Dis*, 2, 1.
- CLIFFORD, J., MARCUS, G. E. & SCHOOL OF AMERICAN RESEARCH(SANTA FE N.M.) (1986) *Writing culture: the poetics and politics of ethnography: a School of American Research advanced seminar*, Berkeley, University of California Press.
- CLYDE, P. W., HARARI, A. E., GETKA, E. J. & SHAKIR, K. M. (2003) Combined levothyroxine plus liothyronine compared with levothyroxine alone in primary hypothyroidism: a randomized controlled trial. *Jama*, 290, 2952-8.
- COCHRANE, A. L. (1973) *Effectiveness and efficiency : random reflections on health services*, [London] ([3 Prince Albert Rd, NW1 7SP]), Nuffield Provincial Hospitals Trust.
- COHN, S., DYSON, C. & WESSELY, S. (2008) Early accounts of Gulf War illness and the construction of narratives in UK service personnel. *Social Science & Medicine*, 67, 1641-1649.
- COOPER, L. (1997) Myalgic encephalomyelitis and the medical encounter. *Sociology of Health & Illness*, 19, 186-207.
- CORNER, G. W. & ALLEN, W. M. (1929) PHYSIOLOGY OF THE CORPUS LUTEUM: II. Production of a Special Uterine Reaction (Progestational Proliferation) by Extracts of the Corpus Luteum. *Am J Physiol*, 88, 326-339.
- CRAWFORD, R. (1980) Healthism and the Medicalization of Everyday Life. *International Journal of Health Services*, 10, 365-388.
- CROSSLEY, M. (1998) 'Sick Role' or 'Empowerment'? The ambiguities of life with an HIV positive diagnosis. *Sociology of Health and Illness*, Vol.20 No. 4 1998.
- CUSSINS, C. (1996) Ontological choreography: Agency through objectification in infertility clinics. *Social Studies of Science*, 26, 575-610.
- DAGGETT, P. (2000) The perils and pitfalls of unnecessary thyroid hormone replacement. *Brit. Thyroid Found. Newsl*, 1-2.

- DANIELS, G. H. C. & DYAN, C. M. (2006) *Thyroid Disorders*, Oxford, Health press Limited.
- DAYAN, C. M., SARAVANAN, P. & BAYLY, G. (2002) Whose normal thyroid function is better--yours or mine? *Lancet*, 360, 353.
- DEGOWIN, R. L., LEBLOND, R. F. & BROWN, D. D. (2004) *DeGowin's diagnostic examination*, New York, McGraw-Hill Professional ; London : McGraw-Hill.
- DEPARTMENT OF HEALTH (2004) *The NHS improvement plan : putting people at the heart of public services : executive summary*, London, Department of Health.
- DEPARTMENT OF HEALTH (2003) Copying letters to Patients: Good practice guidelines.
- DOUGLAS, M. (1966) *Purity and danger: an analysis of concepts of pollution and taboo*, London: Routledge & Kegan Paul.
- DUDEN, B. (1991) *The Woman beneath the skin : a doctor's patients in eighteenth-century Germany*, Harvard University Press.
- DUMIT, J. (2006) Illnesses you have to fight to get: Facts as forces in uncertain, emergent illnesses. *Social Science & Medicine*, 62, 577-590.
- DURRANT-PEATFIELD, B. (2006) *Your thyroid and how to keep it healthy*, London, Hammersmith Press.
- EHRENREICH, B. A. E. & ENGLISH, D. (1973) *Witches, midwives and nurses. A history of women healers ... Reprinted*, Detroit: Black & Red.
- ERIKSEN, T. H. (2001) *Small places, large issues : an introduction to social and cultural anthropology*, London, Pluto.
- ESCOBAR-MORREALE HF, DEL RAY FE, OBREGON MJ & GM.; D. E. (1996) Only combined treatment with thyroxine and triiodothyronine ensures euthyroidism in all tissues of the thyroidectomized rat. *Endocrinology*, 2490-2502.
- ESCOBAR-MORREALE, H. F., BOTELLA-CARRETERO, J. I., DEL REY, F. E. & DE ESCOBAR, G. M. (2005) REVIEW: Treatment of hypothyroidism with combinations of levothyroxine plus liothyronine. *Journal of Clinical Endocrinology and Metabolism*, 90, 4946-4954.
- EVANS-PRITCHARD, E. E. (1937) *Witchcraft, oracles and magic among the Azande*, Oxford, Clarendon Press.
- FAUSTO-STERLING, A. (1985) *Myths of gender : biological theories about women and men*, New York, Basic Books.
- FEATHERSTONE, M., HEPWORTH, M. & TURNER, B. S. (1990) *The body : social process and cultural theory*, London, Sage.
- FLYNN, R. W. V., MACDONALD, T. M., MORRIS, A. D., JUNG, R. T. & LEESE, G. P. (2004) The thyroid epidemiology, audit, and research study: Thyroid dysfunction in the general population. *Journal of Clinical Endocrinology and Metabolism*, 89, 3879-3884.
- FOUCAULT, M. (1976) *The birth of the clinic : an archaeology of medical perception*, London,.
- FOUCAULT, M. (1981) *The history of sexuality / Vol.1, An introduction*, Harmondsworth, Penguin.
- FOX, R. C. (1980) The Evolution of Medical Uncertainty. *Milbank Memorial Fund Quarterly-Health and Society*, 58, 1-49.
- FRANKFORD, D. M. (1994) Scientism and Economism in the Regulation of Health-Care. *Journal of Health Politics Policy and Law*, 19, 773-799.
- FRANKLIN, S. (1997) *Embodied progress : a cultural account of assisted conception*, London, Routledge.
- FRANKLIN, S. & ROBERTS, C. (2006) *Born and made : an ethnography of preimplantation genetic diagnosis*, Princeton, Princeton University Press.

- FREIDSON, E. (1972) *Profession of medicine : a study of the sociology of applied knowledge*, New York, Dodd, Mead & Company.
- FREIDSON, E. (1988) *Profession of medicine : a study of the sociology of applied knowledge : with a new afterword*, Chicago, University of Chicago Press.
- GEERTZ, C. (1988) *Works and lives : the anthropologist as author*, Cambridge, Polity Press.
- GIBBON, S. (2007) *Breast cancer genes and the gendering of knowledge : science and citizenship in the cultural context of the "new" genetics*, Basingstoke [England] ; New York, Palgrave Macmillan.
- GIBBON, S. & NOVAS, C. (2008) *Biosocialities, genetics and the social sciences : making biologies and identities*, London, Routledge.
- GIDDENS, A. (1990) *The consequences of modernity*, Cambridge, Polity in association with Blackwell.
- GOLDING, T. C. (1952) A Brief History of Thyroid Disease. *Historical Bulletin*, 16.
- GOOD, B. (1994) *Medicine, rationality, and experience : an anthropological perspective*, Cambridge ; New York, Cambridge University Press.
- GORDON, D. R. (1988) Clinical Science and Clinical Expertise: Changing Boundaries Between Art and Science in Medicine. IN LOCK, M. M. & GORDON, D. R. (Eds.) *Biomedicine examined*. Dordrecht; Boston, Kluwer Academic Publishers.
- GRAVES, R. (1835) New observed affection of the thyroid gland in females. (Clinical lectures.). *London Medical and Surgical Journal*, 7, 516-517.
- GULL, W. W. (1873- 1874) On a cretinoid state supervening in adult life in women. *Trans Clin Soc Lond*, 7, 180-185.
- HACKING, I. (1986) Making Up People. IN HELLER, T. (Ed.) *Reconstructing Individualism*. Stanford University Press.
- HACKING, I. (1995) The Looping Effect of Human Kinds. IN D., S. (Ed.) *Causal Cognition: An Interdisciplinary Approach*. . Oxford, Oxford University Press.
- HACKING, I. (2002) Kinds of People: Moving Targets *Proceedings of the British Academy*, 151 285-318.
- HAHN. ROBERT A & KLEINMAN. ARTHUR (1983) Biomedical Practice and Anthropological Theory: Frameworks and Directions. *Annual Review of Anthropology*, Vol.12 (1983),pp.305-333.
- HALL, R. & STANBURY, J. B. (1967) Familial studies of autoimmune thyroiditis. *Clin Exp Immunol*, 2, Suppl:719-25.
- HARAWAY, D. J. (1990) *Simians, cyborgs, and women : the re-invention of nature*, London, Free Association.
- HARINGTON, C. R. & BARGER, G. (1927) Chemistry of Thyroxine. *Biochem. J.*, 21, 169-183.
- HASS, S. & TIMMERMANS, S. (2008) Towards a sociology of disease. *Sociology of Health & Illness*, 30.
- HAUG, M. (1973) Deprofessionalization: an alternative hypothesis for the future. . *Sociological Review Monograph*, 2, 195-211.
- HAYDEN, L. C. & SACHS, L. (1998) Suffering, hope and diagnosis: On the negotiation of chronic fatigue syndrome. *Health Affairs*, 2 pp. 175-193.
- HELEN LAMBERT AND ROSE HILARY (1996) Disembodied Knowledge: Making Sense of Medical Science. IN BRIAN, I. A. A. W. (Ed.) *Misunderstanding Science*. Cambridge, Cambridge University press.
- HOLMES, D. (2002) *Tears Behind Closed Door -Failure to diagnose an Underactive Thyroid: The Truth Behind the Tragedy*, Normandi Publishing Ltd.

- HOLMES, D. (2004) *We can make a difference*, available at: <http://www.thyroidtears.co.uk/wecanmakeadifference.html> [Accessed on 20th March 2006]
- HORTON, R. (1995) Canguilhem, Georges - Philosopher of Disease. *Journal of the Royal Society of Medicine*, 88, 316-319.
- HORWITZ, A. V. & WAKEFIELD, J. C. (2007) *The loss of sadness : how psychiatry transformed normal sorrow into depressive disorder*, New York ; Oxford, Oxford University Press.
- HUBBARD, R. & WALD, E. (1997) *Exploding the gene myth : how genetic information is produced and manipulated by scientists, physicians, employers, insurance companies, educators, and law enforcers*, Boston, Beacon Press.
- HUGHES, E. C. (1984) *The sociological eye : selected papers*, New Brunswick, N.J., U.S.A., Transaction Books.
- ILLICH, I. (1975) *Medical nemesis : the expropriation of health*, London,.
- INSTITUTE OF MEDICINE. COMMITTEE ON CLINICAL PRACTICE, G., FIELD, M. J. & LOHR, K. N. (1992) *Guidelines for clinical practice : from development to use*, Washington, DC, National Academy Press.
- IWDL GENOVA DIAGNOSTICS (2006) Urine Thyroid Hormone T3/T4, available at: http://www.gdx.uk.net/index.php?option=com_gpanel&Itemid=2&task=view&id=29 [accessed on 12 April 2006]
- JACKSON, I. M. D. & COBB, W. E. (1978) Why Does Anyone Still Use Desiccated Thyroid Usp. *American Journal of Medicine*, 64, 284-288.
- KAUFMAN, S. R., RUSS, A. J. & SHIM, J. K. (2006) Aged bodies and kinship matters: The ethical field of kidney transplant. *American Ethnologist*, VOL 33; NUMB 1,, 81-99.
- KEVLES, D. J. & HOOD, L. E. (1992) *The Code of codes : scientific and social issues in the human genome project*, Cambridge, Mass, Harvard University Press.
- KONRAD, M. (2005) *Narrating the new predictive genetics : ethics, ethnography, and science*, Cambridge, UK ; New York, Cambridge University Press.
- LAMBERT, H. & ROSE, H. (1996b) Disembodied Knowledge: Making Sense of Medical Science. IN WYNNE. B AND INWIN A. (Eds.) *Misunderstanding Science*. Cambridge, Cambridge University press.
- LANE, C. (2007) *Shyness : how normal behavior became a sickness*, New Haven, Yale University Press.
- LATOUR, B. (1993) *We have never been modern*, New York ; London, Harvester Wheatsheaf.
- LAW, J. (1994) *Organizing modernity*, Oxford, Blackwell.
- LEYS, C. (2001) The British National Health Service in the face of neoliberalism. . IN. ARMSTRONG, P H. ARMSTRONG & COBURN, D. (Eds.) *Unhealthy times: Political economy perspectives on health and care in Canada* Toronto, Oxford University Press.
- LIGHT, D. W. (2001) Comparative institutional response to economic policy managed competition and governmentality. *Social Science & Medicine*, 52, 1151-1166.
- LIPPMAN, A. (1992) Led (Astray) by Genetic Maps - the Cartography of the Human Genome and Health-Care. *Social Science & Medicine*, 35, 1469-1476.
- LOCK, M. (1988) New Japanese Mythologies: Faltering Discipline and the Ailing Housewife, Vol. 15, No. 1, Medical Anthropology. (Feb.,

- 1988), pp. 43-61. *American Ethnologist* Vol. 15, No. 1, *Medical Anthropology*. (Feb., 1988), pp. 43-61.
- LOCK, M. (2001) The Tempering Of Medical Anthropology: The Troubling of Natural Categories. *Medical Anthropology Quarterly*, 15 (4), 478-492.
- LOCK, M. (2005) Eclipse of the gene and the return of divination. *Current Anthropology*, 46.
- LOCK, M. M. (1994) *Encounters with aging : mythologies of menopause in Japan and North America*, Berkeley, University of California Press.
- LOCK, M. M. (2002) *Twice dead : organ transplants and the reinvention of death*, Berkeley, University of California Press.
- LOCK, M. M. & GORDON, D. (1988) *Biomedicine examined*, Dordrecht ; Boston, Kluwer Academic Publishers.
- LOCKE, J. & GOLDIE, M. (1994) *Two treatises of government*, London, Everyman.
- MACGREGOR, A. (1961) Why Does Anybody Use Thyroid Bp. *Lancet*, 1, 329-&.
- MARCUS, G. E. (1995) *Technoscientific imaginaries : conversations, profiles, and memoirs*, Chicago, University of Chicago Press.
- MARCUS, G. E. & FISCHER, M. M. J. (1999) *Anthropology as cultural critique : an experimental moment in the human sciences*, Chicago, London, University of Chicago Press.
- MARKER, R. E., WAGNER, R. B., ULSHAFFER, P. R., WITTBECKER, E. L., D.P.J, G. & C.H, R. (1947) Steroidal sapogenins. *Journal of the American Chemical Society* 69 (1947) pp. 2167-2171.
- MARTIN, E. (1987) *The woman in the body : a cultural analysis of reproduction*, Beacon Press.
- MARTIN, E. (1994) *Flexible bodies : tracking immunity in American culture : from the days of polio to the age of AIDS*, Boston, Beacon Press.
- MARTIN, E. (1998) Fluid Bodies, Managed Nature,. IN BROWN. B AND CASTREE N (Ed.) *Remaking Reality: Nature at the Millennium*. London, Routledge.
- MARTIN, E. (2007) *Bipolar expeditions : mania and depression in American culture*, Princeton, Princeton University Press.
- MAY,C., RAPPLEY,T., MOREIRA,T., FINCH,T. & HEAVEN,B. (2006) Technogovernance: Evidence, subjectivity, and the clinical encounter in primary care medicine. *Social Science & Medicine*, 62, 1022-1030.
- MCKINLAY, J. & STOECKLE, J. (1988) Corporatisation and the social transformation of doctoring. *International Journal of Health Services*, 18, 191-205.
- MCMILLAN, C., BRADLEY, C., RAZVI, S. & WEAVER, J. (2006) Psychometric evaluation of a new questionnaire measuring treatment satisfaction in hypothyroidism: The ThyTSQ. *Value in Health*, 9, 132-139.
- MEDVEI, V. C. (1993) *The history of clinical endocrinology : a comprehensive account of endocrinology from earliest times to the present day*, Carnforth, Parthenon Pub. Group.
- MOL (2002) *The Body Multiple: Ontology In Medical Practice.*, Durham, NC: Duke University Press.
- MOL. A (2008) *The Logic of Care: Health and the problem of patient choice* London, Routledge.
- MOREIRA, T. (2005) Diversity in clinical guidelines: the role of repertoires of evaluation. *Social Science & Medicine*, 60, 1975-1985.
- MOREIRA, T. (2007) Entangled evidence: knowledge making in systematic reviews in healthcare. *Sociology of Health & Illness*, 29, 180-197.

- MOREIRA, T., MAY, C., MASON, J. & ECCLES, M. (2006) A new method of analysis enabled a better understanding of clinical practice guideline development processes matter. *Journal of Clinical Epidemiology*, 59, 1199-1206.
- MURRAY, G. R. (1891) Note on the treatment of myxoedema by hypodermic injections of an extract of the thyroid gland of a sheep. *BMJ*, 2, 796.
- MYKHALOVSKIY, E. & WEIR, L. (2004) The problem of evidence-based medicine: directions for social science. *Social Science & Medicine*, 59, 1059-1069.
- NATIONAL PATIENT SAFETY AGENCY (2007a) About NRES, available at: <http://www.nres.npsa.nhs.uk/applicants/about/NRES.htm>. [accessed on 31st October 2008]
- NATIONAL PATIENT SAFETY AGENCY (2007b) Launch of National Research Ethics Service. *International Journal of Pharmaceutical Medicine*, 1 1-113.
- NAVARRO, V. (2002) *The political economy of social inequalities : consequences for health and quality of life*, Amityville, N.Y., Baywood Pub. Co.
- NETTLETON, S. (1995) *The sociology of health and illness*, Oxford ; Cambridge, Mass, Polity Press.
- NETTLETON, S. (2006) 'I just want permission to be ill': Towards a sociology of medically unexplained symptoms. *Social Science & Medicine*, 62, 1167-1178.
- NHS DIRECT (2006) NHS Direct Online Health Encyclopaedia, Patient choice, available at: <http://www.nhsdirect.nhs.uk/articles/article.aspx?articleId=572>. [accessed on 20th November 2006]
- OAKLEY, A. (1980) *Women confined : towards a sociology of childbirth*, Oxford, Martin Robertson.
- OAKLEY, A. (1984) *The captured womb : a history of the medical care of pregnant women*, Oxford, Basil Blackwell.
- OUDSHOORN, N. (1990) Endocrinologists and the conceptualization of sex, 1920-1940. *Journal of the History of Biology*, 23, 136 - 186.
- OUDSHOORN, N. (1994) *Beyond the natural body : an archaeology of sex hormones*, New York ; London, Routledge.
- PARKES, A. S. (1966) Rise of Reproductive Endocrinology 1926-1940 - Sir Henry Dale Lecture for 1965. *Journal of Endocrinology*, 34, R20-&.
- PARSONS, T. (1952) *The Social System*, pp. xviii. 575. Tavistock Publications: [London;] printed in the U.S.A.
- PAUNKOVIC, N., PAUNKOVIC, J., PAVLOVIC, O. & PAUNOVIC, Z. (1998) The significant increase in incidence of Graves' disease in eastern Serbia during the civil war in the former Yugoslavia (1992 to 1995). *Thyroid*, 8, 37-41.
- PETRYNA, A. (2003) *Life exposed : biological citizens after Chernobyl*, Princeton, N.J., Woodstock, Princeton University Press.
- POP, V. J., MAARTENS, L. H., LEUSINK, G., VAN SON, M. J., KNOTTNERUS, A. A., WARD, A. M., METCALFE, R. & WEETMAN, A. P. (1998) Are autoimmune thyroid dysfunction and depression related? *Journal of Clinical Endocrinology and Metabolism*, 83, 3194-3197.
- PORTER, R. (1997) *The greatest benefit to mankind : a medical history of humanity from antiquity to the present*, London, HarperCollins.
- POWDERMAKER, H. (1966) *Stranger and friend : the way of an anthropologist*, New York, W. W. Norton.
- POWER, M. (1999) *The audit society : rituals of verification*, Oxford, Oxford University Press.

- RABINOW, P. (1992) Artificiality and Enlightenment: From Sociobiology to Biosociality. IN CRARY, J. & KWINTER, S. (Eds.) *Zone 6: Incorporations*. Cambridge, Mass, MIT Press.
- RABINOW, P. (1996) *Essays on the anthropology of reason*, Princeton, N.J, Princeton University Press.
- RABINOW, P. (1999) *French DNA : trouble in purgatory*, Chicago, London, University of Chicago Press.
- RAPP, R. (1999) *Testing women, testing the fetus : the social impact of amniocentesis in America*, New York, Routledge.
- ROBERTS, C. (2002) 'A matter of embodied fact' Sex hormones and the history of bodies. *Feminist Theory*, vol. 3(1): 7-26.[1464-7001(200204) 3:1;7-26; 022063].
- ROSE, N. (2001) The politics of life itself. *Theory Culture & Society*, 18.
- ROSE, N. (2003) Neurochemical selves. *Society*, 41, 46-59.
- ROSE, N. & NOVAS, C. (2004) Biological citizenship. IN ONG, A. & COLLIER, S. (Eds.) *Global Assemblages: Technology, Politics, and Ethics as Anthropological Problems*. Malden MA, Blackwell.
- ROSE, N. S. (2006) *Politics of life itself : biomedicine, power and subjectivity in the twenty-first century*, Princeton, N.J. ; Woodstock, Princeton University Press.
- SACKETT DL, R. W., GRAY JAM, HAYNES RB, RICHARDSON WS. .. (1996) Evidence based medicine: what it is and what it isn't. *British Medical Journal*, BMJ ;312:71-2.
- SCHEPER HUGHES NANCY (1994) Aids and the Social Body. *Social Science and Medicine*, 39, 7, 991-1003.
- SCHIEBINGER, L. L. (2000) Skeletons in the Closet: The First Illustrations of the Female Skeleton in Eighteenth-Century Anatomy. *Feminism and the body*. Oxford, Oxford University Press.
- SHARPE, M. & CARSON, A. (2001) Unexplained' somatic symptoms, functional syndromes, and somatization: do we need a paradigm shift? *Annals of International Medicine*, 926-930.
- SHILLING, C. (2003) *The body and social theory*, London ; Thousand Oaks, California, SAGE Publications.
- SHILLING CHRIS (2002) Culture of the Sick Role and the Consumption of Health. *The British Journal of Sociology*, 53:621-638.
- SIBBALD, B. & ROLAND, M. (1998) Understanding controlled trials - Why are randomised controlled trials important? *British Medical Journal*, 316, 201-201.
- SKINNER, G. R. B., HOLMES, D., AHMAD, A., DAVIES, J. A. & BENITEZ, J. (2000) Clinical Response to Thyroxine Sodium in Clinically Hypothyroid but Biochemically Euthyroid Patients. *J Nutr Environ Med*, 10, 115-124.
- STOCKL, A. (2007) *Complex syndromes, ambivalent diagnosis and existential uncertainty: The case of Systemic Lupus Erythematosus (SLE)*, *Social Science and Medicine*, 65, 1549-1559
- STOP THE THYROID MADNESS!! (2006) *Long and Pathetic List of Thyroid Symptoms*, available at: <http://www.stopthethyroidmadness.com/long-and-pathetic/?PHPSESSID=a21d72683c359416d23dee3406bce172> [accessed on 12 April 2006]
- STRATHERN, M. (1992a) *After nature : English kinship in the late twentieth century*, Cambridge ; New York, Cambridge University Press.
- STRATHERN, M. (1992b) *Reproducing the future : essays on anthropology, kinship and the new reproductive technologies*, Manchester U.P.
- STRATHERN, M. & ASSOCIATION OF SOCIAL ANTHROPOLOGISTS OF THE COMMONWEALTH. CONFERENCE (1995) *Shifting contexts :*

- transformations in anthropological knowledge*, London ; New York, Routledge.
- SULLIVAN, M. (2003) The new subjective medicine: taking the patient's point of view on health care and health. *Social Science & Medicine*, 56, 1595-1604.
- TANENBAUM, S. J. (1996) "Medical effectiveness" in Canadian and US health policy: The comparative politics of inferential ambiguity. *Health Services Research*, 31, 517-532.
- TEW, M. (1990) *Safer childbirth? : a critical history of maternity care*, London, Chapman and Hall.
- THE BRITISH SOCIOLOGICAL ASSOCIATION (2002) Statement of Ethical Practice for the British Sociological Association (March 2002)
<http://www.britsoc.co.uk/equality/Statement+Ethical+Practice.htm>.
- THE BRITISH THYROID FOUNDATION (2006) available at: <http://www.btf-thyroid.org/>
- THE DEPARTMENT OF HEALTH (2006) Choice Matters: Increasing choice improves patients' experiences.
- THE DEPARTMENT OF HEALTH (2009) The NHS Constitution. IN CROWN COPYRIGHT (Ed.).
- THE WORLD MEDICAL ASSOCIATION (1964) Ethical Principles for Medical Research Involving Human Subjects
<http://www.wma.net/e/policy/b3.htm>.
- THYROID PATIENT ADVOCACY UK (2005) Dr. Gordon Skinner Response to BTF, available at: <http://www.thyroiduk.org/> the blood tests pg. [accessed on 24 June 2005]
- THYROID UK (2007) available at: <http://www.thyroiduk.org/>
- TIMMERMANS, S. & BERG, M. (1997) Standardization in action: Achieving local universality through medical protocols. *Social Studies of Science*, 27, 273-305.
- TIMMERMANS, S. & MAUCK, A. (2005) The promises and pitfalls of evidence-based medicine. *Health Affairs*, 24, 18-28.
- TOFT, D. A. (1995) *Understanding Thyroid Disorders*, Banbury, Family Doctor Publications.
- TOMER, Y., BARBESINO, G., GREENBERG, D. A., CONCEPCION, E. & DAVIES, T. F. (1999) Mapping the major susceptibility loci for familial Graves' and Hashimoto's diseases: evidence for genetic heterogeneity and gene interactions. *J Clin Endocrinol Metab*, 84, 4656-64.
- TOMER, Y., BARBESINO, G., KEDDACHE, M., GREENBERG, D. A. & DAVIES, T. F. (1997) Mapping of a major susceptibility locus for Graves' disease (GD-1) to chromosome 14q31. *J Clin Endocrinol Metab*, 82, 1645-8.
- TOMER, Y., GREENBERG, D. A., BARBESINO, G., CONCEPCION, E. & DAVIES, T. F. (2001) CTLA-4 and not CD28 is a susceptibility gene for thyroid autoantibody production. *J Clin Endocrinol Metab*, 86, 1687-93.
- TRAWEEK, S. (1992) *Beamtimes and lifetimes : the world of high energy physicists*, Cambridge.
- TURNER, B. S. (1996) *The body and society : explorations in social theory*, London ; Thousand Oaks, Calif, Sage Publications.
- VANDERPUMP, M. P., AHLQUIST, J. A., FRANKLYN, J. A. & CLAYTON, R. N. (1996) Consensus statement for good practice and audit measures in the management of hypothyroidism and hyperthyroidism. The Research Unit of the Royal College of Physicians of London, the Endocrinology and Diabetes Committee

- of the Royal College of Physicians of London, and the Society for Endocrinology. *Bmj*, 313, 539-44.
- VANDERPUMP, M. P., TUNBRIDGE, W. M., FRENCH, J. M., APPLETON, D., BATES, D., CLARK, F., GRIMLEY EVANS, J., HASAN, D. M., RODGERS, H., TUNBRIDGE, F. & ET AL. (1995) The incidence of thyroid disorders in the community: a twenty-year follow-up of the Wickham Survey. *Clin Endocrinol (Oxf)*, 43, 55-68.
- VILLAR, H. C. C. E, SACONATO, H, VALENTE, O. & ATALLAH, A. N. (2007) Thyroid hormone replacement for subclinical hypothyroidism. *Cochrane Database of Systematic Reviews*, -.
- WAILOO, K. (1997) *Drawing blood : technology and disease identity in twentieth-century America*, Baltimore, London, Johns Hopkins University Press.
- WALD, D. S. (2004) Bureaucracy of ethics applications. *Bmj*, 329, 282-4.
- WALSH, J. P. (2002a) Dissatisfaction with thyroxine therapy - could the patients be right? *Curr Opin Pharmacol*, 2.
- WANLESS, D. (2001) *Securing our future health : taking a long-term view; interim report*, Great Britain, Treasury, Public Enquiry Unit.
- WANLESS, D. (2004) *Securing good health for the whole population : final report*, Norwich, HMSO.
- WEETMAN, A. P. (2006) Whose thyroid hormone replacement is it anyway? *Clinical Endocrinology*, 64:3 231.
- WEIR, R. F., LAWRENCE, S. C. & FALES, E. (1994) *Genes and human self-knowledge : historical and philosophical reflections on modern genetics*, Iowa City, IO, University of Iowa Press.
- WIERSINGA, W. M. (2001) Thyroid hormone replacement therapy. *Hormone Research*, 56.
- WILSON, E. O. (1976) *Sociobiology : the new synthesis*, Cambridge, Mass ; London, Belknap Press of Harvard University Press.
- YOUNG, A. (1995) *The harmony of illusions : inventing post-traumatic stress disorder*, Princeton, Princeton University Press.
- ZOLA, I. (1972) Medicine as an Institution of Social Control. *Sociological Reveiw*, 20: 487-504.

Appendices

1. Patient information sheet
2. Sample of clinic letter
3. BTA patient information sheet on the management of hyperthyroidism
4. BTA patient information sheet on the management of hypothyroidism
5. BFT list of hypothyroid symptoms
6. BFT list of hyperthyroid symptoms
7. Thyroid petition (raised by Diana Holmes)
8. BTA statement on Armour Thyroid (USP) and combined thyroxine/ tri-iodothyronine as Thyroid Hormone Replacement

Patient Information Sheet

Title of Study - Genetic Predispositions and Chronic Disease: A qualitative study of the implications of genetic predispositions on the definition, patient understanding and management of thyroid conditions.

You are being invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part. Thank you for taking the time to read through this information.

What is the purpose of this study?

This 12 month study will examine how genetic predispositions to autoimmune thyroid conditions influence their treatment and management. Autoimmune thyroid conditions tend to run in families, in the sense that if your parents have these conditions you are more likely to develop them, and this study will be particularly interested in this component.

This study will investigate how familial information about thyroid conditions, from the perspective of patients, is understood in terms of how it influences its diagnosis, the treatment you receive in the clinic, how you understand the disease, and how you choose to manage your condition.

Why have I been chosen?

You have been chosen because you have or are attending the thyroid clinic at the [REDACTED] [REDACTED] as a patient for treatment for your thyroid condition. If you agree to take part in the study you will be one of approximately 15 patients who will be involved in the project.

Do I have to take part?

It is up to you to decide whether or not to take part. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to withdraw at any time without giving reason. A decision to withdraw at any time, or a decision not to take part, will not affect the standard of care you receive.

What will happen to me if I take part?

You will be interviewed in your home, or an alternative mutually agreed location, twice over a twelve-month period. These interviews will be recorded, last approximately one to two hours, and will involve a series of questions that are designed to find out about your experience of having a thyroid condition. There are no right or wrong answers to these questions and you can talk about anything that you feel is relevant to your thyroid condition and its treatment. You will not be expected to attend any additional appointments with your consultant or GP in order to take part in the study.

What do I have to do?

To take part in the study you have to agree to the interviews in your home or another mutually agreed location. The interviews will take place at a time convenient for you and there will be no additional interventions in terms of how you will be treated by your consultant for your thyroid condition.

What is the drug or procedure that is being tested?

There is no drug or procedure being tested, this study is non-clinical and involves only interviews.

What are the possible disadvantages and risks of taking part?

It is possible that during the interviews you may feel that some of the issues addressed in relation to your thyroid disease are sensitive or embarrassing. If you do feel like this you must raise it with the interviewer. In this instance you could ask the interviewer to move on to another subject or terminate the interview all together. You may also pull out of the study at any time if you choose to do so, this will in no way affect your treatment.

What are the possible benefits of taking part?

There are no direct benefits to your treatment if you take part in this study. It involves no interventions or new treatments for your thyroid condition. However, it is hoped that this study will add further to the understanding of thyroid conditions, specifically with regards to understanding how patients manage and understand the disease.

What if new information becomes available?

As this is a non-clinical study your doctor will communicate all possible new information about the treatment of your thyroid condition to you. It is not within the remit or expertise of this study to pass on clinical information or advice. If new information about the study specifically becomes available you will be informed immediately via the contact details you will have provided on your consent form.

What happens when the research study stops?

Your treatment will continue as normal as it did during the duration of the study, you will not be expected to take part in any further interviews or be observed in any additional appointments.

What if something goes wrong?

As this study is non-clinical it will not effect your treatment, therefore, there is no risk of harm and no special compensation arrangements in terms of negligence with regards to your treatment. Regardless of this, if you wish to complain, or have any concerns about any aspect of the way you have been approached or treated during the course of this study, the normal National Health Service complaints mechanism should be available to you.

In addition, there are also contact details for senior supervisory staff for the project at the London School of Economics and Political Science, these can be found at the end of this information sheet in *the contact for further detail* section.

Will my taking part in the study be kept confidential?

All information that is collected about you during the course of the research will be kept strictly confidential. Any information about you that leaves the hospital/surgery will have your name and address removed so that you cannot be recognised from it. In addition permission will also be sought from your GP for your inclusion in the study, something to which you must agree in order to take part in the study.

The [REDACTED] have approved this study, under the assurance that high standards of ethical protocol will be observed.

What will happen to the results of the research study?

All transcripts and tape recordings of the interviews will be kept in secure storage at the London School of Economics, and only those involved in the study will have access to them. If you wish you may also have copies of interview transcripts. In addition to the availability of the interview transcripts you will also be provided with a report of the study summarising its findings near the end of the project.

Megan Clinch a graduate student in the BIOS Centre at the London School of Economics and Political Science will use, the data collected to form the basis of a PhD thesis. This thesis will be approximately 70,000 to 100,00 words long and is expected to be completed by October 2007, it may also form the basis for additional papers and articles that may be published in relevant professional journals and presented at conferences. You will be informed of such instances as they occur and be sent a summary of the research when it is completed.

Who is organising and funding the research?

The research is the basis for a PhD thesis organised by Megan Clinch a student in the BIOS Centre at the London School of Economics and Political Science and it has received funding from the institution through which it is being conducted. The research has been conceived through a supervisory relationship with BIOS Associate Director Professor Sarah Franklin and sponsored by the convenor of the BIOS Centre and the Department of Sociology Professor Nikolas Rose.

Who has reviewed the study?

In addition to the review of the study by [REDACTED] the study had been reviewed via a through process by the department of Sociology at the London School of Economics and Political Science, the institution through which the PhD will be awarded.

Contact for further information

Megan Clinch a PhD student at the London School of Economics and Political Science in a research Centre called BIOS that is an affiliated to the department of Sociology is conducting the study. Attached is a leaflet on BIOS for you to look at, there is also a website www.lse.ac.uk/bios

If you would like to contact Megan to find out more about the study please do so, her details are as follows.

BIOS
The London School of Economics and Political Science
Houghton Street
London
WC2A 2AE
m.l.clinch@lse.ac.uk
Office: 0207 107 5211
Mobile: 07866 455 408

It is important that you feel that your participation in the study has been handled with respect and sensitivity with regards to your condition.

If once enrolled in the study, you do not feel the correct protocols have been followed, please contact the senior project supervisors listed below.

Professor Sarah Franklin
PhD Supervisor and Associate Director of BIOS
The London School of Economics and Political Science
Houghton Street
London
WC2A 2AE
s.franklin@lse.ac.uk
020 7955 6465

Professor Nikolas Rose
Director of BIOS, Professor of Sociology and Convenor of the Department of Sociology
The London School of Economics and Political Science
Houghton Street
London
WC2A 2AE
n.rose@lse.ac.uk
020 7955 7533

Please keep these details just in case a situation of this nature arises.

How do I take part in the study?

To take part in the study please fill out the consent form including your contact details and then return it to Megan Clinch in the stamped addressed envelope provided. Megan will then contact you to arrange a convenient time for interview.

Thank you for taking the time to read through this information, we look forward to hearing from you if you decide to take part in the study.

DEPARTMENT OF ENDOCRINOLOGY

Ext. [REDACTED]

Direct Line: [REDACTED]

Fax: [REDACTED]

E-mail: [REDACTED]

MV/ag/Hosp No. [REDACTED]

Thyroid Clinic: 18 September 2006

Typed: 27 September 2006

[REDACTED]

Dear Dr [REDACTED]

Re: [REDACTED]

**Diagnosis: Treated hypothyroidism
Past history of atrial fibrillation
Osteoporosis
Treated type 2 diabetes**

This lady's thyroid function now is fine on thyroxine 225µg daily. Her current FT4 is 22.4pmol/l with a TSH right within the middle of the reference range at 2.4mu/l. She should thus remain on this dose in the long term and I simply suggest she had an annual check of thyroid function from now on. The only other thing to correct from my previous letter is that she was on Flecainide not Amiodarone.

With best wishes

Yours sincerely

[REDACTED]

cc

[REDACTED]

Hyperthyroidism – clinical features and treatment

1. The causes of hyperthyroidism

The thyroid is a gland in the neck that produces two thyroid hormones, thyroxine (T4) and tri-iodothyronine (T3). Thyroxine is inactive and is converted by the tissues and organs that need it into tri-iodothyronine. The role of thyroid hormones, put simply, is to regulate the metabolism of virtually all cells in the body. In health, the production of these thyroid hormones is tightly regulated by the secretion of thyroid stimulating hormone (TSH; also known as thyrotropin) from the pituitary gland in the brain. When the thyroid gland becomes affected by disease, sometimes the production or release of thyroxine and tri-iodothyronine can be abnormally high, leading to increased levels in the blood; a state of thyroid overactivity known as hyperthyroidism or thyrotoxicosis. If this happens, the body's metabolism speeds up and this can be manifest by changes in various, and seemingly unrelated tissues, that are listed below. In this state of hyperthyroidism, a blood test will show an elevated amount of these thyroid hormones circulating. Conversely, the TSH level in the blood almost always becomes suppressed, because the pituitary gland senses ('sees') the abnormally high levels of thyroid hormones, which are more than is needed by the brain.

The prevalence of hyperthyroidism is about 1% and it is about six times more common in women. There are two main causes of hyperthyroidism in the UK:

- 1). Autoimmunity causing stimulation of the thyroid gland
- 2). Overproduction of hormones by benign tumours in the thyroid gland.

1). In autoimmune thyroid overactivity, the thyroid cells are stimulated by an abnormal antibody which is specifically targeted at the TSH-receptor on the thyroid gland causing stimulation of the thyroid to produce excess hormones. This also causes the thyroid cells to grow, and together with immune cells congregating in the gland, this leads to thyroid enlargement, called goitre.

An early description of this form of autoimmune thyroid disease was made by an Irish physician called Robert Graves, so it is often termed Graves' disease. Graves' disease is almost always accompanied by the presence of the TSH-receptor autoantibodies in the blood and very frequently by thyroid peroxidase (TPO) autoantibodies which may both be a useful tool for diagnosis. In addition, about a third of people with Graves' disease develop a variety of eye problems including a staring appearance, grittiness and soreness, protruding eyeballs, and (rarely) double vision or sight problems. This is termed "thyroid eye disease" or "Graves' ophthalmopathy". Cigarette smoking increases the risk of developing thyroid eye disease in patients with Graves' disease.

2). The other common cause of thyroid overactivity is that the thyroid develops one or more benign tumours (technically follicular adenomas but often simply called “nodules”) that secrete excess thyroid hormone in an unregulated manner. This nodular hyperthyroidism becomes commoner with advancing age and is termed “solitary toxic nodule” or “toxic multinodular goitre”, depending on the number of nodules.

Together these two types of hyperthyroidism account for well over 90% of all cases. Rarer causes include inflammatory conditions of the thyroid called thyroiditis, which sometimes is the result of pregnancy, viruses or drugs such as amiodarone or interferon. All the types of hyperthyroidism just mentioned are usually classified as *primary*, meaning that they result from an excess stimulation or release of thyroid hormone from the thyroid gland. Very rarely, there may be secondary thyroid overactivity as a result of a pituitary problem where the pituitary gland manufactures an excess amount of TSH (thyroid stimulating hormone). This leads to thyroid overactivity with normal or high blood TSH. More commonly blood tests that have the same pattern as primary thyroid overactivity can result from taking an excess of thyroid hormone tablets, such as levothyroxine.

2. The symptoms and signs of hyperthyroidism

Common complaints include fatigue, heat intolerance, sweating, weight loss despite good appetite, shakiness, inappropriate anxiety, palpitations of the heart, shortness of breath, tetchiness and agitation, poor sleep, thirst, nausea and increased frequency of defecation. The elderly may complain predominantly of heart problems with a fast or irregular heart beat, breathlessness and ankle swelling, whereas children tend to hyperactivity, with a short attention span. Signs include shaky and hot hands, fast or irregular heart beat, inability to sit still, flushing of the face and upper trunk, fast tendon reflexes, an enlarged thyroid gland and prominent or bulging eyes. Nowadays patients often are diagnosed at an early stage of disease, owing to increased awareness and improved biochemical testing. Therefore some patients have relatively few of the classical signs or symptoms. In addition, none of the symptoms or signs just listed is sufficiently sensitive or specific for the diagnosis of hyperthyroidism, even when combined together. Thus, it may take three to six months to diagnose hyperthyroidism, during this time the person can feel very unwell. It is not uncommon for people to worry that they have cancer, because of the associated weight loss.

3. Treatment of hyperthyroidism

Graves' disease and nodular thyrotoxicosis

Betablockers

Betablockers are a group of drugs that tend to improve some of the symptoms and manifestations of hyperthyroidism. In particular, they can improve palpitations, slow the heart down and improve tremor. They have no effect on curing the thyroid overactivity, but do make many people feel better. Betablockers should not be taken if the patient has asthma or a wheezy chest.

Antithyroid drugs

Carbimazole (Neomercazole) and propylthiouracil are antithyroid drugs that are effective in reducing the production of thyroid hormones in the majority of people with hyperthyroidism. In people with Graves' disease, treatment with one of these drugs for between 6 months and 2 years results in a long-term remission in around half of patients, once the drug is stopped.

Both drugs have the common side effects of rash and joint pains, and more rarely (less than 1 in 500 cases) a serious reduction in the circulating white blood cells (agranulocytosis) may occur during treatment. The dosage of these antithyroid drugs can either be adjusted every 6 to 8 weeks according to thyroid hormone levels in the blood, to keep the person's thyroid hormone levels in the normal range (titrated dose regimen) or kept at a fixed, higher dose and levothyroxine replacement added to maintain normal thyroid hormone levels (block and replace regimen). In nodular hyperthyroidism (solitary toxic nodule or toxic multinodular goitre), antithyroid drugs do not result in cure, just a temporary reduction in thyroid hormone levels. A more permanent solution is often sought, called a definitive treatment.

Radioiodine

Radioiodine is a radioactive isotope of iodine (^{131}I) that is taken up and concentrated selectively by the thyroid gland. In most people, this small dose of radioactivity is sufficient to gradually destroy the thyroid tissue, over 6 weeks to 6 months following a single dose. Patients with Graves' disease have a high rate of permanent thyroid underactivity following radioiodine (about 80%), whereas patients with nodular thyroid overactivity tend to preserve their thyroid function better, with only around half eventually becoming underactive. Patients are monitored for underactivity following the dose and promptly treated with thyroxine, should this develop. The common outcome of thyroid underactivity is an accepted consequence of radioiodine therapy because hyperthyroidism is a serious condition whereas replacement treatment with levothyroxine is simple and has no side effects at the correct dose.

Radioiodine is a safe treatment for thyroid overactivity, with no overall excess of cancers in many hundreds of thousands of patient years of follow up (JAMA 1998; 280: 347-355; Lancet 1999; 353: 2111-5). Patients with ophthalmopathy require careful evaluation, as radioiodine may worsen

thyroid eye disease: this can be prevented by a short course of steroid tablets. There is no damage to fertility or to hair growth, but women are advised not to become pregnant for 6 months following a dose, as the baby's thyroid could be damaged. Men should avoid fathering a child within 4 months of treatment. Following a standard dose of radioiodine, other precautions are necessary to minimise radiation exposure of others but these restrictions are usually easily accommodated by the patient. Radioiodine may trigger airport security alarms up to eight weeks following a dose and patients should carry a letter about the treatment if they travel in this period. Radioiodine is the most cost effective and certain treatment for thyroid overactivity and about 10,000 doses annually are given in the UK.

Thyroid surgery

Surgery to remove most or all of the thyroid gland (subtotal or total thyroidectomy) is another way of definitively treating thyroid overactivity. This is a straightforward operation when carried out by an experienced thyroid surgeon, with a low risk of complications. Hypothyroidism is a recognised side effect of surgery for which levothyroxine replacement will be needed, lifelong. Thyroidectomy is a good treatment option for people with a large goitre and for those with thyroid eye disease. Prior to thyroid surgery, thyroid overactivity needs to be controlled, usually with antithyroid drugs to make an anaesthetic safe. This is because an anaesthetic in a hyperthyroid person has a high risk of precipitating a dangerous hyperthyroid crisis or "thyrotoxic storm".

Treatment of thyroiditis

Many forms of thyroiditis are 'self-limiting', meaning that the overactivity recovers spontaneously and no treatment may be required. If the person has severe symptoms of thyrotoxicosis, betablockers are helpful. However, in some cases thyroiditis can be painful or prolonged and anti-inflammatory tablets or steroids may be helpful. Furthermore, in some cases a period of thyroid underactivity may follow the thyrotoxicosis, and this may require levothyroxine treatment, if causing severe symptoms.

Subclinical hyperthyroidism

In subclinical hyperthyroidism, the TSH is suppressed but the free thyroid hormone levels are normal. Endocrinologists regard this condition as a precursor of overt or clinical hyperthyroidism but there is some debate over whether this mildest of degree of hyperthyroidism should be treated (JAMA 2004; 291: 228-238, J Clin Endocrinol Metab. 2007 ;92:3-9.). Further research is being conducted in this area. At present treatment is a matter for individual clinical evaluation and discussion between patient and doctor, although there is a consensus that treatment may be worthwhile in the elderly, particularly if the heart rhythm becomes abnormal or there is thinning of the bones or low-impact bone fractures.

Hypothyroidism – clinical features and treatment

1. The causes of hypothyroidism

The thyroid is a gland in the neck which makes two thyroid hormones, thyroxine (T4) and tri-iodothyronine (T3). Thyroxine is inactive and is converted by the tissues and organs that need it into tri-iodothyronine. The role of thyroid hormones, put simply, is to regulate the metabolism of virtually all cells in the body. When there is too little thyroid hormone (hypothyroidism) the body's metabolism slows down and this is manifested by changes in various tissues that are listed below. Around 80% of tri-iodothyronine in the body is derived from conversion of thyroxine in the tissues (a process mediated by deiodinase enzymes), the remainder coming directly from the thyroid gland. Most (>99%) of the thyroid hormone in the blood is bound to proteins and is not available to cells. The free fraction of T4 and T3 in the blood is therefore a more useful measure of thyroid hormone levels than the total amount of these hormones. This is what is meant by free T4 (FT4) and free tri-iodothyronine (FT3).

The prevalence of hypothyroidism is about 2% and this is ten times more common in women. There are two main causes of hypothyroidism in the UK, namely autoimmunity and as a side effect of treatment for an overactive thyroid or for thyroid cancer. In autoimmune thyroid diseases, the thyroid cells are destroyed by white blood cells (lymphocytes) which attack the thyroid. Autoimmune thyroid disease is usually accompanied by the presence of thyroid peroxidase (TPO) autoantibodies which can be detected in the blood and are therefore a useful tool for diagnosis. In patients who have an overactive thyroid or thyroid cancer, treatment may consist of surgery or radioiodine, both of which destroy the diseased gland but inevitably result in a significant proportion of patients developing hypothyroidism.

Together these two types of hypothyroidism account for well over 90% of all cases. Rarer causes include inflammatory responses in the thyroid (sometimes as the result of viruses or drugs such as amiodarone or lithium), abnormal thyroid development in the foetus and genetic defects in thyroid function (leading to congenital hypothyroidism which should usually be picked up during neonatal screening). Iodine deficiency is still a common cause of hypothyroidism in some parts of the world but is very rarely encountered in the UK. All the types of hypothyroidism just mentioned are usually classified as *primary*, meaning that they result from direct impairment of the thyroid gland's function.

Impaired thyroid function may also occur as a result of pituitary disease, because the pituitary manufactures TSH (thyroid stimulating hormone) which is the most important internal factor controlling thyroid function. If the pituitary is damaged and cannot make TSH, the thyroid stops working. Although uncommon, such *secondary* hypothyroidism is important as the normal blood test used to test for the presence of primary hypothyroidism (namely the TSH level) can be misleading. Such patients usually have other clinical features suggestive of pituitary disease, so a careful history and clinical examination will point to the correct blood tests that need to be undertaken if hypothyroidism is suspected.

2. The symptoms and signs of hypothyroidism

Common complaints include fatigue and lethargy, cold sensitivity, dry skin and lifeless hair, impaired concentration and memory, increased weight with poor appetite and constipation. Patients may also fairly often experience a hoarse voice, tingling of the hands (carpal tunnel syndrome), heavy and, later, absent periods, deafness and joint aches. In childhood there may be delayed development and in the adolescent precocious puberty. The elderly may develop memory disturbance, an impaired mental state or depression, and in rare cases coma can occur, resulting in death if left untreated. Signs include slow movements, 'myxoedema facies' in which the face looks puffy due to the accumulation of subcutaneous fluid, cool dry skin, slow pulse rate, thinning of the hair including the eyebrows, slow tendon reflex relaxation time, slow pulse rate and hoarse voice. The thyroid may be enlarged (causing a goitre) in some patients due to accumulation of lymphocytes (Hashimoto's thyroiditis), but in others the thyroid is destroyed by the time of diagnosis and there is no goitre.

Nowadays patients often are diagnosed at an early stage of disease, due to increased awareness and improved biochemical testing. Therefore many patients have relatively few of the classical signs or symptoms just listed. In addition, none of the symptoms or signs is sufficiently sensitive or specific for the diagnosis of hypothyroidism, even when combined together.

3. Treatment of hypothyroidism

Thyroxine

Thyroxine (or levothyroxine) is the current standard thyroid hormone replacement recommended in the British National Formulary (BNF). Patients in the UK who require thyroxine can obtain an exemption certificate, which means that they do not

have to pay for prescriptions of this drug. This is the standard treatment for thyroid hormone deficiency. It has a half life of 7 days and is readily converted into tri-iodothyronine (by a process called peripheral deiodination) in the body's tissues. This same process occurs with the thyroxine secreted by the thyroid.

Goal of treatment with thyroxine

The goal of treatment in primary hypothyroidism is to reverse the symptoms of hypothyroidism by normalising the blood TSH level. The most recent UK guidelines, published by the Association of Clinical Biochemists and the British Thyroid Association (BTA) in 2005, state 'The aim of treatment should be to restore and maintain the TSH level within the reference range' (<http://www.acb.org.uk/docs/TFTguidelinefinal.pdf>).

The TSH blood test is successful in establishing the correct dosage of thyroxine because there is a feedback loop between thyroid hormone in the blood and the pituitary. When thyroid hormone levels are low, TSH levels rise, and conversely when thyroid hormone levels are high, the TSH levels fall. The pituitary is very sensitive to changes in circulating thyroid hormone levels and the amount of TSH it secretes is therefore a useful yardstick to measure how much thyroid hormone the whole body is exposed to.

Prolonged periods of overtreatment with thyroid hormone (associated with a reduction of TSH levels below the reference range) increase the risk of developing atrial fibrillation (an irregular heart rhythm associated with a risk of stroke) and bone thinning (summarised in JAMA 2004; 291; 228-238).

Measurement of serum T4 or T3 levels on their own are not recommended for monitoring thyroid hormone replacement in primary hypothyroidism, as the levels may change through the day after ingestion of a tablet and the levels do not reflect the tissue response to thyroid hormone in the way TSH does. For instance, if a patient omits thyroxine tablets for a few weeks the TSH levels will rise, but the FT4 level will be normal if the patient then remembers to take thyroxine for a day or two before attending clinic.

Subclinical hypothyroidism

In subclinical hypothyroidism, the TSH is elevated but the free thyroid hormone levels are normal. Endocrinologists regard this condition as a precursor of overt or clinical hypothyroidism but there has been considerable debate over whether even this

mildest of degrees of hypothyroidism can be associated with symptoms and whether it should be treated (JAMA 2004; 291: 228-238, J Clin Endocrinol Metab 2005; 90: 581-585). Further research is being conducted in this area. At present treatment is a matter for individual clinical evaluation and discussion between patient and doctor, although there is a consensus that treatment is usually worthwhile if repeated TSH levels exceed 10mU/L.

Use of tri-iodothyronine

Around 80% of circulating T3 arises from the peripheral tissues by deiodination of T4 and only around 20% is directly secreted by the thyroid gland. Thyroxine treatment in hypothyroid individuals is predicated on the assumption that this 'missing' 20% of T3 can be compensated for by increased peripheral deiodination.

There have been recent trials of tri-iodothyronine replacement in combination with replacement of thyroxine. However T3 given as a liothyronine (or tertroxin) tablet does not reflect a physiologically relevant replacement. Firstly, its half life is 24 hours and administration results in undesirable, non-physiological peaks of serum T3. Secondly, a molar ratio of 14:1 T4:T3, delivering around 100mcg T4 and 6mcg T3 per day, would be optimal for an average adult patient. Liothyronine tablets are 20mcg in size, making any approach to mimic normal T3 replacement extremely difficult with standard sized tablets, especially in those who still have a degree of thyroid remnant function.

Despite initially promising results in a small trial, the benefits of T3 and T4 combination treatment in patients with hypothyroidism have not been borne out by several large and more prolonged trials. Data from 11 independent randomised control trials (1216 patients) were pooled and reviewed in J Clin Endocrinol Metab 2006; 91: 2592-2599).

There was no overall objective evidence of benefit in terms of symptom scores (body weight, depression, fatigue, quality of life) or other physiological markers (serum cholesterol, triglyceride levels, low or high density lipoprotein. At present, combination treatment is not recommended by endocrinologists. Future work is needed to determine whether any benefits might occur with sustained release T3 preparations which are not yet developed for use.

Armour thyroid extract

Armour thyroid extract is desiccated animal thyroid extract which was superseded by synthetic thyroxine in the 1960s. It must be obtained from the USA. Although not normally prescribed in the UK, because it is not licensed for use, it can be given through the NHS if specific arrangements are made on a named patient basis. According to the Medicine and Healthcare Products Regulatory Agency, it is the decision of individual NHS Trusts as to whether an unlicensed product like Armour is made available on a NHS or private prescription.

Armour thyroid extract is not recommended by endocrinologists as standard thyroid hormone replacement treatment, as the amount of thyroid hormone is more variable between batches than it is in thyroxine tablets. Furthermore, the ratio of T3 to T4 in Armour thyroid extract tablets is higher than is normally secreted by human thyroid tissue, resulting in potentially harmful levels of T3 (one grain, about 60 mg, of desiccated thyroid extract contains about 38mcg of T4 and 9mcg of T3). The position of the BTA is set out in a statement (<http://www.british-thyroid-association.org/armour.htm>). Although some patients wish to take Armour, for instance, because they perceive it to be 'natural' rather than 'synthetic', there have been no scientific studies that compare it to thyroxine, and there is a theoretical reason based on the ratio of T3 to T4 to believe it could have adverse effects.

Thyroid hormone treatment in euthyroid individuals

Only one prospective study has been conducted to assess the possible benefit of thyroxine treatment in euthyroid individuals (Brit Med J 2001; 323: 891-895). In this controlled trial there was no effect of thyroxine. There are also strong theoretical reasons to believe that such treatment is futile. The full position of the BTA is summarised in a joint statement with the Society for Endocrinology (http://www.british-thyroid-association.org/thyroid_statement.pdf).

Signs and Symptoms of Hypothyroidism

Also known as Underactive Thyroid, Myxoedema and Hashimoto's Disease

- General tiredness
- Excessive need of sleep
- Increased awareness of the cold
- The skin may become dry and thick and feels cold
- The hair may begin to thin out become dry and coarse
- Unusual loss of body hair – eyebrows may become sparse, and hair on forearms short and stubbly
- Flaking, splitting nails
- The voice may become hoarse or croaky
- Constipation
- Muscle weakness, cramps and aches; difficulty climbing stairs
- Sore muscles
- Pins and needles in the fingers and hands
- In women of reproductive years the periods may become heavier and longer, but sometimes can prematurely stop
- Fertility problems – failure to conceive, miscarriage.
- Unexplained weight gain
- Puffy face and bags under the eyes, change in facial appearance
- Slow speech, movements and thoughts
- Low mood, depression
- Memory problems and lack of concentration
- Slow heart beat and slightly raised blood pressure
- Increased cholesterol
- Anaemia
- Hearing problems
- Swelling at the front of the neck
- Sensation of a lump in the throat
- Although rare, in severe cases, unsteadiness on their feet, mental disturbance and even hallucinations may be experienced
- Loss of libido / impotency

Signs and Symptoms of Hyperthyroidism

Also known as Overactive Thyroid and Graves' Disease:

- Palpitations – undue awareness of heart beat
- Rapid and sometimes irregular heart beat
- Breathlessness
- Hair loss
- Brittle nails
- Unexplained weight loss
- Swelling and or tenderness at front of throat
- Hyperactive behaviour. Children tend to be clumsy and drop things
- In children they may have grown faster than their peers so that their height is greater than normal for their age
- Difficulty sleeping
- Nervousness / Anxiety
- Irritability
- Aggressive behaviour
- Sweating
- Heat intolerance
- Thirst
- Tremor in hands and fingers
- Looseness of the bowels, diarrhoea
- Weak muscles – the upper muscles of your legs and arms are most likely to be affected. You may have difficulty in getting up from the squatting position without using your arms or find it hard to lift a heavy package down from a high shelf
- Rapid pulse
- Warm moist hands
- Increased appetite
- Lack of concentration and memory loss
- Eye pain, double vision
- Swelling or protrusion of the eyes
- Development of painless red lumps, usually on the shins
- In women of reproductive years the periods may become scant and sometimes can prematurely stop
- Impaired fertility
- Osteoporosis

- Low cholesterol
- Low blood pressure
- Loss of libido / impotency

British Thyroid Foundation - www.btf-thyroid.org

THYROID PETITION

We the undersigned [thyroid patients, families/friends] wish to lodge this petition with the General Medical Council as a formal complaint against the clinical practice of the majority of the medical profession with regard to the diagnosis and management of hypothyroidism on four counts: -

1. Over reliance on thyroid blood test results and a total lack of reliance on signs, symptoms, history of the patient and a clinical appraisal.
2. The emotional abuse and blatant disregard by the majority of general practitioners and endocrinologists over the suffering experienced by untreated/incorrectly treated thyroid patients and their lack of compassion over the fate of these patients.
3. Stubbornness by the majority of general practitioners and endocrinologists to treat patients suffering with hypothyroidism with a level of medication that returns the patient to optimum health. In addition, the unwillingness to prescribe alternative thyroid treatment for patients on individual clinical grounds. For example a combination of T4/T3, T3 alone or a natural thyroid treatment such as Armour Thyroid.
4. The ongoing reluctance to encourage debate or further research on hypothyroidism.

In addition we formally request an independent investigation into patients who are hypothyroid, which includes examination of clinical results of patients treated by private doctors (whose work is outside NHS directives), and comparative examination of clinical results of patients treated by NHS practitioners who diagnose and manage hypothyroidism.

Failure to address these issues will result in a vote of no confidence in the General Medical Council.

NAME IN CAPITALS	TOWN	POST CODE	SIGNATURE

DATE: **TOTAL NUMBER OF SIGNATURES ON THIS SHEET:**

Armour Thyroid (USP) and combined thyroxine/ tri-iodothyronine as Thyroid Hormone Replacement

A Statement from the British Thyroid Association Executive Committee

February 2007

Part 1. Use of Armour Thyroid

A. Armour Thyroid contains both thyroxine (T4) and tri-iodothyronine (T3) extracted from the thyroid gland of pigs. One grain, about 60 mg, of desiccated pig thyroid extract contains about 38mcg of T4 and 9mcg of T3, a ratio of around 4 to 1. The normal concentration of these hormones in the human thyroid is, however, at a ratio of 14 to 1. In other words, Armour thyroid extract contains excessive amounts of T3 relative to T4 when used to replace thyroid hormone in man. Moreover, as pig thyroid contains other substances apart from T4 and T3, Armour Thyroid is not a pure preparation of thyroid hormones. Historically, extracts of animal thyroid glands were the only way to treat thyroid underactivity, but since the 1950s pure synthetic thyroid hormones have been available in tablet form (thyroxine sodium [T4] and liothyronine [T3]).

B. The concentration of thyroid hormones in Armour Thyroid USP is regulated by the manufacturer to United States Food and Drug Administration (FDA) standards. Despite this, there have been significant problems with the stability of Armour Thyroid in recent years, prompting a massive recall of tablets.¹ Because of these stability problems with Armour Thyroid, there is potential for fluctuations in thyroid hormone levels in the blood of patients treated with Armour Thyroid. These fluctuations may be unpredictable and have adverse effects on patients' health.

C. There is no evidence to favour the prescription of Armour Thyroid in the treatment of hypothyroidism over the prescription of thyroxine sodium, as supplied in the United Kingdom. There has never been a direct comparison of these two treatments. The BTA committee cannot recommend a treatment with possible side-effects, when a safe and equally well-established treatment exists.

D. Armour Thyroid is not on the British National Formulary and is not a licensed therapy in the UK. Mr. G. Matthews, the Pharmaceutical Assessor of the Medicines and Health Care Products Regulatory Agency, in a letter sent to BTA dated 19 October 2005, has clarified that "The regulations on medicine allow doctors to prescribe an unlicensed medicine for a patient to meet such a special clinical need, on their own direct personal responsibility. Where these unlicensed medicines are not available in the UK they can be imported by appropriately licensed medicines wholesalers, for supply to a doctor or pharmacy, to meet these needs."

E. The cost of Armour Thyroid may be up to £20 per month, compared to an equivalent cost of £1 per month for thyroxine. Furthermore, Armour Thyroid is not eligible to be claimed on the prescription exemption certificate (FP10).

Part 2. Use of Combination Thyroxine/ Tri-iodothyronine (Liothyronine) Therapy

A. There is no currently available tablet preparation containing thyroxine and tri-iodothyronine (T4/T3) in a combination that adequately reproduces the relative quantities of these hormones produced by the human thyroid gland. Neither is there a preparation that produces a sustained release of thyroid hormones in a pattern similar to that from the human thyroid gland.

B. Having been disregarded as a therapeutic approach to the treatment of hypothyroidism since the 1970s, interest in combination thyroxine/tri-iodothyronine (T4/T3) therapy was re-ignited by a study of 31 patients published in 1999.² Although this study showed promising results, with improvement in quality of life, wellbeing and brain (psychometric) function with combination therapy, the majority of the patients in the study had been treated previously for thyroid cancer. The relatively high doses of thyroxine that formed the routine treatment for thyroid cancer (compared to a lesser replacement dose that would be normal in hypothyroidism) could have confounded the results of the study.

C. Since this initial study, there have been a further seven rigorously conducted (“randomised, double-blind, placebo-controlled”) studies, encompassing more than 900 hypothyroid patients (summarised in refs. 3 & 4). None of the subsequent studies showed a beneficial effect of combined T4/T3 therapy on measures of wellbeing, health and mental functioning. Three of the seven studies show harmful or undesirable effects of the T4/T3 combination.

D. In three of the subsequent studies of combination treatment, the patients were asked which treatment they preferred, and in two of these 3 studies more patients preferred the combination T4/T3 therapy. There is no obvious explanation for these observations, and it may or may not be a reproducible effect.

E. The BTA keeps an open mind about whether using an appropriate formulation of T4/T3 combination tablet would, in the future, provide health and quality of life benefits in the treatment of hypothyroidism for a subgroup of patients. However, based on the current evidence from rigorous studies of large numbers of patients using the currently available formulations of synthetic thyroid hormones, combined T4/T3 cannot be recommended because of a lack of benefit and a small number of undesirable and harmful effects seen on combination treatment.

British Thyroid Association Executive Committee, March 2007

References

1. An FDA enforcement removed more than half a million bottles of Armour Thyroid from US pharmacies in 2005 due to unstable concentrations of thyroid hormone in the preparation.
[www.fda.gov/bbs/topics/enforce/2005/ENF00899.html]
2. Bunevicius R, Kazanavicius G, Zalinkevicius R, Prange AJ Jr. Effects of thyroxine as compared with thyroxine plus triiodothyronine in patients with hypothyroidism. *N Engl J Med.* 1999; 340: 424-9.
3. Escobar-Morreale HF, Botella-Carretero JI, Escobar del Rey F, Morreale de Escobar G. Review: Treatment of hypothyroidism with combinations of levothyroxine plus liothyronine. *J Clin Endocrinol Metab.* 2005; 90: 4946-54.
4. Grozinsky-Glasberg S, Fraser A, Nahshoni E, Weizman A, Leibovici L. Thyroxine-triiodothyronine combination therapy versus thyroxine monotherapy for clinical hypothyroidism: meta-analysis of randomized controlled trials. *J Clin Endocrinol Metab* 2006; 91: 2592-9