On the Historical Contingency of Medical Knowledge:
An integrated historical and philosophical investigation into the development and epistemic status of knowledge about heart failure

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Abstract

A commonplace view amongst philosophers is that the function of the heart is obviously to pump blood around the body. Given this, one might think that facts about whether patients’ heart are failing to function properly are simple to discover, fully determined by the given nature of human physiology, and independent changing human cultures. I argue against this view, noting that doctors in the present day do not believe that the function of the heart is simply to pump blood. I argue that medical knowledge about heart failure is not progressing towards an ultimate truth, but rather that this knowledge is historically contingent. To do this I explore the development of knowledge of heart failure over the last two centuries, focusing particularly on the early nineteenth century research of James Hope, and the early twentieth century research of James Mackenzie. I compare the facts produced about heart disease by these two historical actors, noting that they produced very different ways of understanding heart disease, and diagnosed heart disease in different ways. For instance, Hope argued that the stethoscope was a powerful diagnostic tool, whereas Mackenzie argued that the stethoscope had probably done more harm than good. I analyse these differences using Ludwik Fleck’s work, drawing especially on his account of the active and passive elements of knowledge. I argue that if Fleck’s epistemology is read as a form of conventionalism, and augmented using the work of Henri Poincaré, then these active and passive elements of knowledge are suitable tools for analysing how Hope and Mackenzie produced the facts that they did. Using Fleck in this way, I will argue that medical knowledge can be historically contingent and objective, at once invented and discovered. I also use my longue durée account to argue that the way heart failure is understood today cannot be explained without taking the full sweep of this history into account.
Consequently, I argue that medical history is valuable to the evaluation of diagnostic practices in the present day.
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Introduction
This thesis is an integrated historical and philosophical investigation into the nature and production of medical knowledge about heart failure. It combines historical research into the development of medical knowledge and philosophical reflection on the implications of this history for medical epistemology.

The central questions addressed in this thesis relate to the possibility and consequences of the historical contingency of medical knowledge. By historical contingency I do not simply mean that what is accepted as knowledge changes over time. Rather, I mean that medical knowledge is not the inevitable result of the way the world is\(^1\). This is to claim that there are many alternative forms of medical knowledge, which are as correct and as successful as each other. It is to claim that knowledge of medicine could have legitimately developed differently to how it actually has developed.

In my discussion of historical contingency and medical knowledge I make frequent use of the notions of *discovery* and *invention*, which I define in terms of *the freedom to choose how things are*. If medical knowledge is completely discovered, then it is fully determined by the way the world is in itself. If our knowledge is entirely discovered, and we intend to represent the ‘world in itself’ faithfully, we have no choice in this matter, as this knowledge is fully determined by the way nature is. For the purpose of this thesis, I identify the condition of being ‘discovered’ with a limitation of our freedom to choose how things are. On the other hand, if knowledge of disease is completely invented or made up, then

\(^1\) The connection between historical contingency and the lack of inevitability has been made by Ian Hacking (1999: 69). Referring specifically to Andrew Pickering’s (1984) work on *Constructing Quarks*, Hacking says that the claim that knowledge of quarks is historically contingent does not mean that scientists could have erroneously come to believe that quarks do not exist, but rather than it would possible for scientists to develop an alternative and yet equally successful and legitimate physics without proceeding in a “quarky way”. I adopt this view of historical contingency here.
it is the choices made by people that constrain this knowledge, not the natural world. I identify the condition of being ‘invented’ with having the freedom to choose how things are.

The thesis that all aspects of medical knowledge are historically contingent is inconsistent with the view that any aspect of medical knowledge is discovered. It is not, however, inconsistent with the view that all aspects of medical knowledge are invented. The view that all aspects of medical knowledge are invented is an unacceptable epistemic thesis, as it entails that medical knowledge is whatever people choose that it is. If this were the case, it would be impossible to make progress in medical practice, and there would be little to no point in doing medical research. In arguing that medical knowledge is historically contingent I am not claiming that medical knowledge is entirely invented. Rather, my thesis is that all aspects of medical knowledge are at once invented and discovered.

Arguing that medical knowledge is historically contingent in this sense may seem unnecessary, especially given the scholarship that has discussed the historical contingency of other areas of scientific knowledge (Kuhn 1996; Shapin and Schaffer 1985; Pickering 1984; Chang 2004). In scholarship on medical knowledge, however, prominent philosophers and historians deny that key aspects of medical knowledge are historically contingent.

Consider Paul Thagard’s (1999) work on medical knowledge, which focuses on the production of knowledge about the role of Helicobacter pylori in causing stomach ulcers. Thagard argues in favour of a form of medical realism, in which it is possible to discover truths about diseases and their causes. “By the term medical realism, I mean that diseases and their causes are real and that scientific
investigation can gain knowledge of them” (Thagard 1999: 81). Whilst he concedes that various versions of anti-realist might be valuable when studying other sciences like physics, Thagard argues that medical knowledge is entirely discovered:

“Whatever plausibility anti-realism might have for exotic theories such as quantum mechanics, the roles of instruments and experiments in the ulcers and bacteria case support medical realism. I argue that ulcers and H. Pylori bacteria are real entities independent of any mental and social constructions and that the theoretical claim that H. Pylori is an important causal factor in ulcers can be accepted as true” (Thagard 1999: 81).

This is not to say that Thagard believes that medical knowledge can be produced simply by some logically rigorous process, and he explicitly rejects such views (1999: 219). Thagard also rejects what he calls the postmodern view that knowledge reduces to a system of competing interests and power relations:

“[T]he traditional view of science as logic and the postmodern view of science as power are both inadequate for understanding how science develops...It should be obvious by now that science is a complex psychological, social, and physical system” (Thagard 1999: 219).

I should emphasise that Thagard’s views are perfectly compatible with fallibilism – the truth may be other than what researchers currently believe. Nevertheless, Thagard argues that there are truths about the natural world, which are true because they reflect the way the world is in itself, and that can be to be discovered by researchers. Thagard argues that denying this is almost delusional:

“Are scientists deluded in thinking that systematic observation, painstakingly controlled experiments, and rigorous hypothesis evaluation can teach us about the world? The delusion lies instead in those who think that science is just another semiotic exercise like literary criticism or fashion design” (Thagard 1999: 239).

Thagard is certainly not alone in holding that at least some aspects of medical knowledge are discoverable. Many philosophers of medicine take this to be the
case (see chapter 3). Perhaps many would not say, as Thagard does above, that
timelessly correct disease categories can be discovered, but many do say that
biological facts that researchers take to be relevant to disease classifications are
objectively discoverable (see chapter 3). Some prominent medical historians also
agree that facts about disease status are timeless and discoverable. To see this,
consider Mark Harrison’s (2015) reflections on writing *longue durée* history of
disease:

“If our aim is solely to understand disease as contemporaries did, then its
biological identity is relatively unimportant. But the insistence that we must
avoid using modern disease categories prevents us from charting the
spread of disease or explaining the rise and fall of epidemics and their
relationship to economic, political, and environmental changes. These are
surely legitimate questions. They are also vital if we are to attempt
anything more than a localized study. Otherwise, there would be no reason
to place the “French disease” or the “Neapolitan disease” within the same
analytical frame, let alone *firangi roga* or any of the other names used to
describe syphilis in Asia. If cross-cultural comparisons are to be attempted,
or long-distance connections explored, then it is clearly useful to establish
the identity of the disease in question” (Harrison 2015: 648-649).

Harrison claims that it is possible, at least in theory, to use modern disease
categories to establish the identity of the disease with which patients in history
were actually suffering. This entails that it is possible to discover these timeless
disease identities, and that present-day researchers have managed to do this.
Adrian Wilson (2000) has commented that this “naturalist-realist” approach to the
historiography of disease is quite common amongst historians, although it is not
universal (see chapter 3). Other historians do argue that all aspects of medical
knowledge are historically contingent (Wilson 2000; Cunningham 2002). My point
is that this claim is controversial, and worth attending to.

In this thesis I focus on three related questions about medical epistemology:
1) Are all aspects of medical knowledge about heart failure historically contingent?

2) Does the historical contingency of this medical knowledge mean that it is impossible to make progress in medical practice?

3) How does the historical contingency of medical knowledge affect discussions about the development and evaluation of medical knowledge?

By addressing these questions I show how integrated history and philosophy of medicine can be of use to medical practice whilst at the same time addressing related debates in the history and philosophy of medicine.

A diagnosis is both the process by which what ails a patient is recognised, and a category describing a sort of patient (Jutel 2017: 156). I use the term in both senses in this thesis, as do the historical actors to which I refer. The diagnosis of heart failure refers both to the diagnostic category of heart failure, and to the process by which heart failure is recognised. This thesis makes use of longue durée historical investigation into the development of present day knowledge of the diagnosis of heart failure (in the categorical sense of the term), from the early 1800s to the present day. I combine this longue durée approach with a more geographically and temporally focused investigation into the work of two key actors in this history: the British physicians James Hope and James Mackenzie. I use this history to argue that medical knowledge is historically contingent.

I argue that Ludwik Fleck’s epistemology is useful for understanding the work of these historical actors, particularly when it is augmented with perspectives drawn from other philosophers, especially Henri Poincaré. By paying close attention to Fleck’s discussions of what he calls the active and passive elements of knowledge, I show how medical knowledge can be entirely historically contingent.
without thereby collapsing into an extreme form of relativism. I argue that knowledge of how to diagnose heart failure can be at once *invented* and *discovered*.

This *longue durée* history is organized into a series of successive iterative stages. Each stage presents what I see as a different way of approaching the diagnosis of heart failure. I have decided that these differences are important, either because they are due to the use of different diagnostic practices being used to make the diagnosis (resulting in different patients being grouped together as diseased), or because of a change to how heart failure was explained physiologically, or both. I argue that how heart failure is diagnosed in later stages is profoundly affected by how heart failure was diagnosed in earlier stages. Consequently, I argue that in order to understand why heart failure is diagnosed in the way it is today, it is necessary to pay attention to how knowledge of heart failure has developed through these earlier stages. As history is so important for understanding why heart failure is diagnosed the way it is today, history can also be used to inform arguments about how best to diagnose heart failure in the present day.

In this introduction I reflect on the methods I have used to integrate the history and philosophy of medicine (section 1). I also take the opportunity to address historiographical concerns that have been raised about the possibility of writing *longue durée* histories of disease (section 2), before discussing the contribution made by this thesis to the historical and philosophical literatures on medicine (section 3), and summarizing the arguments presented in the individual chapters of this thesis (section 4).
1 – Methodological reflections
The way that I have integrated history and philosophy of medicine in this thesis was not formally planned, but rather was something that grew out of my experience of medical training and practice. It is helpful to present the development of my methodological approach in connection with these experiences, before introducing the philosophical tools I selected to address the problem I encountered in practice.

1.1 – The growth of my approach to integrated history and philosophy
My original training is as a veterinary surgeon. I spent five years training at the Royal Veterinary College, London, graduating in 2006. I then spent three years working in veterinary practice, which included the completion of an equine neonatal internship in the U.S.A.. My daily work in mixed practice in the UK included surgical and anaesthetic procedures, radiographic investigations, managing the routine medical problems encountered in general small animal practice, assessing colicing horses, and dealing with difficult calvings. Throughout my training and practice, I was struck by the lack of attention paid by my teachers and colleagues to the justification of how they practised. This was particularly the case for the justification of diagnostic practices. In my experience, very little attention was paid to the question of why it was that the information gathered using some diagnostic practices (e.g. the patient’s history, clinical examination, radiography, blood tests, or some combination of these) was considered vital to making a particular diagnosis, whereas other information gathered using different diagnostic practices was not. Different practitioners would disagree about the most valuable diagnostic practices for the diagnosis of a particular disease, and would be unable to articulate why they believed that they were right, and others were wrong. I spent a considerable amount of time
and effort in practice trying to uncover the reasons for making diagnoses in one way rather than another. Time and time again, my attempts were frustrated.

The central problem was that recommendations about all the characteristics that could be used to distinguish patients with a disease from patients without it were presented as if they were produced by carefully observing populations of patients and recording which characteristics were displayed by patients with a disease and which were not. It ought to be immediately apparent that such recommendations cannot be the result of careful observation alone. For, in order to observe the differences between patients with and without a disease, one must first be able to distinguish diseased and non-diseased patients. In other words one must have prior knowledge of at least some of the distinguishing characteristics of disease in order to make empirical observations about the distinguishing characteristics of disease. This prior knowledge cannot itself be produced simply by carefully observing populations of patients.

My teachers and colleagues in practice made no attempt to acknowledge the need for this prior knowledge, and simply kept on discussing the characteristics of different diseases as if they were all associated with the patients with the disease by careful observation alone. Consequently I was very dissatisfied with how I saw diagnostic practices being evaluated.

As I could not satisfy myself about the best way to diagnose disease by reviewing evidence about diagnostic accuracy in the present, I decided to explore medical history, to see how certain diagnostic practices had come to be accepted as valuable for the diagnosis of certain diseases. To this end I enrolled on an MSc degree in the history of science, medicine and technology, jointly hosted at Imperial College London and University College London.
I was immediately exposed to points of view about medicine that were entirely foreign to me from my medical training, and which helped make sense of the difficulties I had faced in practice. The most important of these was that medical knowledge might be *historically contingent*. On this view, there are no timeless and universal truths about the diseases with which patients suffer. Rather, diseases, and all aspects of knowledge about diseases, are understood to change with human culture. Therefore, there is no universal truths about which diagnostic practices detect these diseases most accurately.

This suggestion was at once liberating and frightening. On the one hand, it was liberating because it explained why the justification of diagnostic practices had proved so difficult. Hitherto, I (along with my colleagues and many other people across the medical professions) had been trying to uncover universal truths about which characteristics could be used to distinguish patients with different diseases. If there are no universal truths to uncover, then it was small wonder that this task had frustrated me. Perhaps this task had not been accomplished because it was *impossible* to accomplish.

On the other hand, this suggestion was frightening because if medical knowledge is historically contingent, it is not clear how this knowledge is constrained by anything apart from human culture. Without universal truths about the ‘world in itself’ to constrain our knowledge, what prevents knowledge from being anything that researchers in different cultures agree that it is? If knowledge is historically contingent, how is it possible to be wrong? If it is not possible to be wrong, then doctors cannot make mistakes, and cannot improve their practice. On this view the whole project of trying to learn how to care more effectively for patients is undercut, and the practice of medicine dies.
Despite these concerns, I was impressed by the thesis that medical knowledge is historically contingent. Where medical historians had focused on how and why doctors had diagnosed and treated disease in the way that they had, decisions to change how patients were managed had convincingly been shown not to be the inevitable results of some empirical finding. My own work on the historical development of knowledge of heart failure further satisfied me that claims about the historical contingency of medical knowledge were credible. At the same time, however, it was also apparent that this history contained many examples of observations which conflicted with researchers’ expectations. Indeed, such conflicts provided one of the main drivers for the changes researchers have made historically to how heart failure has been diagnosed and understood. So even though I did not see universal truths being discovered in this history, it was also clear that medical knowledge is not whatever researchers agree that it is.

Of course, it is not possible to establish beyond a shadow of a doubt from the historical record that there are no universal truths to be found. No amount of history will be able to exclude the possibility that universal truths may be uncovered in the future. And yet, this does not mean that history has no role to play in philosophical discussions about medical knowledge. Medics (and indeed philosophers) who are enamoured with the view that medical knowledge should seek to capture, or at least to closely approximate, universal truths about the diagnosis of disease do not hold that this should be some futuristic dream. Rather, claims about the accuracy of a diagnostic practice are claims that the universal truth about the accuracy of this practice is known right now, in the present. Medical history can help assess claims about whether or not researchers have actually captured universal truth, or come close to doing so, because if researchers have done so then it should be possible to point to the historical
episodes where this was done. If no such episodes can be found, then this undercuts the philosophical assumption that medical knowledge accepted in the present corresponds to the ‘way the world is in itself’.

Once history had made the view that present day medical knowledge is comprised of universal truths untenable, then it became reasonable to think again about whether or not it is helpful to keep searching for diagnostic categories that correspond to the ‘way the world is in itself’\(^2\). Why should researchers continue this difficult and unsuccessful search? One very good reason to stick to the notion of universal truth is that the alleged historical contingency of medical knowledge is frightening, as discussed. But what if historical contingency was not frightening? What if it was possible for medical knowledge to be entirely historically contingent without it thereby being reduced to whatever researchers decide that it is? If a way could be found to accept this contingency without adopting an extreme form of relativism, then contingency would become something that needs to be recognised, understood, and taken account of, but not *feared*. If such an epistemology was available then researchers could be freed from the burden of trying to show that what they believe corresponds to the ‘way the world is in itself’, which causes the difficulties discussed above.

In my view, the epistemic positions available in discussions of the concepts of health and disease in contemporary philosophy of medicine are not quite suitable for my purposes (see chapter 3). Consequently, I returned to my historical findings on the development of heart failure, to explore how medical knowledge

\(^2\) I am not claiming that the only reasonable reaction to the observation that current medical knowledge is not comprised of universal truths is to reject the possibility of universal truth. One might respond like George Canguilhem (1989: 269), who after he showed that the concepts of the normal and the pathological were historically contingent suggested an approach to disease that he believed was timeless and absolute. Nevertheless, I believe a reasonable response to this observation is to reconsider the possibility of finding out about the ‘world in itself’. 
was produced in this case. By looking closely at parts of my *longue durée* history of heart disease, whilst at the same time making use of a number of philosophical resources, I sought to uncover an epistemological account that could cope with these demands.

I chose to focus on the first two iterative stages in my longer history, which taken together comprise the transition between James Hope’s and James Mackenzie’s distinctive ways of diagnosing heart disease. It was important to look at a transition between two stages, as opposed to a single stage in isolation, because comparing different ways of diagnosing heart disease throws many things that might be taken for granted and thus camouflaged into sharp relief. In Fleck’s terminology, looking at at least two stages allows me to do some *comparative epistemology* (1979: 38). It was also apparent to me that it was impossible to understand what the researchers were doing in one stage without exploring what the researchers were doing in the stage before it. Studying Mackenzie turned out to be particularly rewarding because in his published work he reflected at length on what he thought was wrong with earlier ways of understanding heart disease, why he thought it was wrong and what he thought should be done about these mistakes. Mackenzie is a fascinating and important figure in the history of medicine, as well as a philosopher of medicine in his own right, and he deserves to have more scholarly attention paid to his work. Mackenzie is also a key player in developing the diagnosis of heart *failure*, as opposed to the diagnosis of heart *disease* (Lawrence 1985), making his work particularly relevant to the question of how heart failure is diagnosed in the present.

Looking closely at these episodes, it became clear that the actors’ own accounts of how they had produced their knowledge of heart disease/failure are incomplete. Both Hope and Mackenzie held that their knowledge was founded on
raw observations, which were unaffected by any assumptions or intuitions about the nature of heart disease. Comparing the work of these two actors shows that this was not the case. In order to understand how knowledge was being produced in these episodes, I needed to select and use philosophical tools from outside contemporary philosophical discussions of health and disease. The main tool I selected was the epistemology of Ludwik Fleck, focusing particularly on his discussions of the active and passive elements of knowledge. Even though Fleck’s epistemology is a very useful tool for the task at hand, I found that it was not quite up to the task. Having used Fleck to help make sense of the history, I then used the history to augment Fleck’s epistemology. To do this I drew on other philosophical tools in addition to Fleck’s epistemology, and I should expand on why I chose the tools that I did.

1.2 – My philosophical toolbox

The plague to which Woods refers is foot and mouth disease (FMD), a disease characterized by blistering and sloughing of the skin of the tongue, nose, mouth, udder and feet of livestock. An outbreak of FMD in 2001 led to the slaughter of 5% of the UK national herd and flock. Woods provides a longue durée history of how the response to an outbreak of FMD has changed in the British Isles since the early 1800s, right up to the present day. She argues that the response to an outbreak is not determined by the nature of the disease, but rather is historically contingent – manufactured by man (Woods 2004: xiv). Not content with doing purely descriptive work, Woods also shows how present day policy of how to
respond to an outbreak of FMD is shaped by this history. She uses this history to understand why present day policy is as it is, and then to evaluate it. She says that her “account is written in the hope that readers will take heed of the past and use it to make sense of recent events and to plan their future response to FMD” (Woods 2004: xvi). Woods starts with a medical problem (the response to an outbreak of FMD), then seeks to understand that problem using history, and uses this understanding to make normative decisions about how to respond to the problem in the future.

Chang adopts a similar position to Woods, in that he argues that a complex interplay of history, philosophy and science can a play a role complementary to that of science itself (2004: 235-250). Chang argues that any group of researchers must take some things they claim to know for granted, in order to get on with their work (2004: 237). Questioning this taken for granted knowledge is often prohibited within the relevant scientific discipline, and this can lead to an unhealthy dogmatism. Chang envisions a role for integrated history and philosophy in exploring taken for granted beliefs, expressly for the benefit of the relevant science. The goal of this way of integrating history and philosophy of science is not to inform historical and philosophical descriptions of how science works, or philosophical prescriptions about how to do science, but rather to contribute to the production and use of scientific knowledge itself. Particularly by fostering a critical awareness, as Chang calls it (2004: 243), of how present day knowledge of diagnosis was produced, my goal is to show how integrated history and philosophy of medicine in its complementary mode can be used to inform medical practice in the present day.

Chang takes this further by highlighting the philosophical questions that are thrown up by the use of history to inform scientific practice. By focusing on
apparently straightforward scientific claims, like ‘it is possible to measure temperature using thermometers’ (Chang 2004), and ‘water is the substance with the chemical formula H₂O’ (Chang 2012a), Chang highlights that significant philosophical puzzles emerge by asking the epistemic question how is it known that these apparently uncontroversial claims are true? Chang argues that looking at history of thermometry and the chemical analysis of water reveals that this knowledge was not as straightforward to produce as might be expected (Chang 2012b: 112-118). At the same time, history focused on exploring philosophical puzzles about how knowledge is produced can challenge commonplace philosophical assumptions about the production of knowledge and contribute to the historiography of science. For Chang, the understanding of concrete historical episodes is enriched by making use of abstract conceptual tools developed by philosophers, and I make extensive use of this approach in this thesis (Chang 2012b: 110, 122; see also his use of Percy Bridgman’s operationalism in Chang 2004: 142-158, and 2012a: 188-192). Furthermore, Chang argues that this history can be used to generate new philosophical ideas about how knowledge has been produced (2012b: 121). For instance, Chang describes how his notion of epistemic iteration, emerged from his historical work (2012b: 115). Instead of following the more widely practiced methodology of using an historical case study to inform a philosophical debate, Chang has developed a more complex way of integrating history and philosophy, which involves a dialogue between historical and philosophical claims, goals and methods.

Fleck also employed a complex admixture of history, philosophy and medicine in his own work, as is reflected in the structure of Genesis (1979). The first and third chapters are more historical, whilst the second and fourth chapters reflect philosophically on the historical material presented before them, setting up a
dialogue between history and philosophy as the work proceeds. The first chapter presents a *longue durée* history of syphilis, and the third a more temporally and geographically focused history of the development of the Wassermann reaction. Fleck thus made use of both *longue durée* and more focused history in his philosophical work, a technique that I adopt. As it is beyond the scope of a single thesis to explore every transition that I have described in my *longue durée* history in detail, I focus on one transition in order to produce insights that may be applied to other transitions. I have chosen to focus on the first transition from Hope to Mackenzie because this is where the view that heart *failure* should be the focus of diagnostic attention first became prominent.

Another virtue of Fleck’s work is that he problematized the biological reality of disease itself, whereas Woods does not. Like Fleck, I am focusing and reflecting on how the knowledge of the diagnosis of a particular disease has developed. I do not focus on how societies have responded to a disease (like Woods 2004, or Rosenberg 1962), or how concepts of health and disease have developed (like Canguilhem 1989), or on how disease entities have come and gone (like Grmek 1989), or on how the cultural meanings of the same disease has changed over time (like Jackson 2009).

Like Chang, Fleck chose a claim that was taken to be beyond dispute (the purported relationship between the Wassermann reaction and syphilis) as the focus of his work. Fleck did this because he hoped that by showing that this allegedly indubitable fact was a man-made, historically contingent, cultural

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3 Woods says that “[a]lthough FMD [foot and mouth disease] is a ‘natural’ entity, which indisputably possesses certain biological characteristics, the reason why it is so feared, and the reason why its appearance causes such devastation, is because of the methods used to control it. Those methods are man-mad. It is the thoughts and actions of humans that have ‘manufactured’ FMD into the particular political, economic, social and psychological problem it is today” (2004: xiv). Woods thus problematizes the response to FMD, but not the biological reality of the disease itself.
product, this would encourage suspicion about other facts about the diagnosis of disease and in other areas of knowledge. I follow Fleck by choosing to focus on facts about heart failure, which (in some circles) are also taken to be uncontroversial facts beyond the province of dispute. It is my hope that suspicion of other facts about the diagnosis of disease might follow a fortiori from this work.

Fleck also organized his historical work into iterative stages, and emphasized the importance of comparing these different stages with one another (thereby doing what he called comparative epistemology), as this made the historically contingent and taken for granted aspects of knowledge more apparent.

Even though I have said that epistemic positions presented in contemporary philosophical discussions of health and disease were of limited value to me, some of this literature did make good suggestions about where to look for an epistemology that would be of use to me. In particular, H. Tristram Engelhardt (1996: 228, footnote 1) has suggested that a valuable medical epistemology would be a sort of Kantianism, but with "an Hegelian accent". In such an epistemology, the Hegelian accent is that the synthetic a priori knowledge would not be fixed for all people in all cultures, as Kant would have it, but rather would be historically contingent. Engelhardt has not managed to articulate such an epistemology, but this suggestion points the way towards philosophers who have.

Henri Poincaré’s conventionalism, for instance, does just this. Poincaré re-cast several areas of knowledge that Kant held were synthetic a priori as conventions, which Poincaré argued can and do change with human culture. However, Poincaré’s epistemology is hampered by his commitment to raw observations. Fleck’s epistemology resembles Poincaré’s in many ways. There are strong parallels between Poincaré’s conventions and laws and Fleck’s active and
passive elements of knowledge. Fleck improves upon Poincaré’s epistemology by rejecting the possibility of raw observations. However, Fleck’s epistemology suffers also from problems that Poincaré handles more easily. In particular, Fleck’s account of his epistemology sometimes lapses into an extreme form of relativism, which is unacceptable given the historical episodes in question and given the goals of medicine. Poincaré’s epistemology never makes such lapses, and articulates how objective knowledge is still possible even in the absence of raw observations. So although Fleck is an improvement over Poincaré, it is still useful to read Fleck with Poincaré’s conventionalism in mind.

The longue durée history I present has distinctly Kuhnian features. As Kuhn (1957) did, I trace the development of knowledge over an extended period of time. I argue that successive shifts in how heart failure was understood were precipitated by experiences that conflicted with researchers’ expectations, which is similar in many respects to Kuhn’s account of paradigm shifts precipitated by anomalies. Kuhn also sought to develop an epistemology in which scientific knowledge is neither a description the given world in itself, nor a fabrication concocted by researchers. Paul Hoyningen-Huene (1993) argues that, on Kuhn’s view, knowledge of the world is knowledge of a phenomenal world, not the world in itself, and is somewhere in-between a discovery and an invention:

“First, our use of the expression “to gain access to the phenomenal world” should neither suggest that the phenomenal world has an existence independent of and prior to the community, and thus needs only to be discovered by its members, nor be taken to imply that the community creates the phenomenal world entirely according to its own dictates; the constitution of the phenomenal world is neither purely passive reception nor purely active invention. On the contrary, the inseparability of genetically subject-sided from genetically object-sided moments of a phenomenal world implies that gaining access to a phenomenal world is something that lies between the two poles, “discovery” and “invention”” (Hoyningen-Huene 1993: 65).
As I too seek such a middle ground between discovery and invention, my work is done in a Kuhnian spirit. However, even sympathetic and careful students of Kuhn (like Hoyningen-Huene) concede that he did not manage to articulate such an epistemology:

“Kuhn’s theory of world construction aims exactly at answering this question: how do the subjects of knowledge constitute their world of appearances? I hasten to add that this theory cannot really be found explicitly and fully worked out in Kuhn’s writings” (Hoyningen-Huene 1989: 395).

Hoyningen-Huene takes on the task of reconstructing this epistemology from “hints” Kuhn leaves throughout his work (Hoyningen-Huene 1989: 395). In my view, this task is made difficult because Kuhn does not provide a philosophical tool that is equivalent to Fleck’s active and passive elements of knowledge (see chapter 7). As this feature of Fleck’s epistemology is so valuable to me, I have chosen to adapt Fleck’s work to my purposes, rather than Kuhn’s.

2 – Historiographical concerns about writing longue durée history of disease
In recent years, writing longue durée histories of disease has become very popular. Both Oxford University Press and Johns Hopkins University Press have published a series of books on the biography of disease. Authors contributing to these series focus on telling the story of a particular disease over an extended period of time, linking together many different ways of understanding sick people together.

Even so, the possibility of writing longue durée history of particular diseases has recently been questioned, and in my view for good reasons⁴. For instance Roger Cooter (2010; 2013) and Claudia Stein (2014) have both forcefully argued that

⁴ I am not arguing that such histories should not be written, but rather recognising the challenge that writing long histories of disease presents serious historiographical challenges, chiefly that this form of history seems to encourage anachronism.
writing biographies of disease is inherently anachronistic. The central challenge of writing *longue durée* history of disease is to follow how a disease changes over an extended period of time. To do this historians must establish continuities between different ways of understanding disease in different cultures at different times. Biographers of disease use several different techniques for establishing this continuity, but Cooter and Stein argue that they must all result in anachronism.

One way of establishing continuity is to take some earlier ways of understanding sick people as prototypes for later, more correct, ways of understanding sick people. This approach introduces anachronism by adopting progressivist and whiggish historiography. Another way is to re-classify patients in the past according to present day ways of understanding sick people. This approach introduces anachronism because present day ways of understanding sick people were not available to historical actors. Yet another way is to identify different ways of understanding sick people together according to some set of criteria, like the appearance of a particular clinical syndrome, or the use of a particular disease label. This approach is anachronistic as it is the historian in the present that

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5 Tattersall (2009), Wilson (2010), Weatherall (2010) are examples of recent historical work on the history of disease that takes such an approach.

6 Examples of this approach can be found in Jones (2010), Jackson (2009), Packard (2007), and Dubos and Dubos (1953). There is an ongoing debate about the legitimacy of making retrospective diagnoses in the history of disease (see Wilson 2000; Cunningham 2002; Mitchell 2011; Muramoto 2014; Packard 2016). I am not arguing that writing history by making retrospective diagnosis is valueless. I agree with Jackson (2002) that a diverse range of historiographical approaches to disease are valuable. It is fascinating to learn from Jones (2010) about the connections between the eighteenth and nineteenth century wool industry and what we now think of as anthrax. In trying to inform current malaria management policy using history, Packard (2007: xvii) is doing very important work. Even so, Packard admits that his work “is grounded in the assumption that a disease, that we now call malaria, has existed as a distinct biological entity for centuries”, so that he can examine “the conditions that have shaped its distribution over time and space as well as efforts to control it” (2016: 198). The point is that even though this approach is valuable, it is not a suitable for my purposes, because of the anachronism that opponents of the practice retrospective diagnosis have identified.

7 Adrian Wilson’s (2000) history of pleurisy uses this approach, as do Woods (2004), Jackson (2009), Hamlin (2014), Temkin (1945) and Rosenberg (1962). In this approach, the historian latches on to some criteria that are sufficiently important to unite many different ways of understanding sick people as different instances of the same thing (e.g. the epileptiform seizure for Temkin’s epilepsy, or the
chooses the supposedly timelessly important and ahistorical criteria to serve as the essence of the disease in question. The production of connected narratives that unite several different ways of understanding sick people together stands accused of “transplanting into the past the hidden or potential existence of the future” (Cooter 2013: 167; Stein 2014: 58; quoting Latour 2000: 250).

In order to write *longue durée* history of disease, historians need to identify changes across several different ways of understanding sick people. In order to identify change, the historian must also identify continuity between these different ways of understanding sick people. Unless continuity is established between different ways of understanding sick people, showing that these are different to one another does not establish that one changed into another, only that they are different. For history we need change, and for change we need continuity, for otherwise all we have is difference. For historians to identify continuity, it seems that they must decide which similarities they think are important, and thereby introduce anachronism, which is ahistorical. The possibility of writing *longue durée* histories of disease appears to reduce to absurdity.

The potential absurdity of writing *longue durée* histories of disease is troubling for me in this context. This thesis relies on historical work that follows the development of heart failure across boundaries between different ways of understanding sick people. If such historical work is absurd then this thesis is also absurd. As I want to use history to inform debates about how to diagnose disease presence of a wheeze, a cough, and spasmodic respiratory distress for Jackson’s asthma). Wilson (2000) objects to using any medical criteria as trans-historically constant, as he says that this robs the disease of its history, and betrays a historiographical essentialism. And yet, Wilson’s history is vulnerable to the same criticism, as he connects all the different pleurisies he does together because they share a label – pleurisy. Why should this common label be taken to reveal multiple instances of the same disease, rather than multiple different diseases with the same label? The same could be said for Rosenberg (1962), Woods (2004), and Hamlin (2014). If the historian is responsible for deciding which similarities between different ways of understanding sick people are timelessly important, then this introduces anachronism.
in the present day, so I must not write this history assuming that knowledge of disease in the present day is timelessly correct. My challenge, then, is to explain how it is possible to establish continuity between different ways of understanding sick people without identifying similarities that I think are timelessly important from the present, thereby introducing anachronism.

As it turns out, this is quite simple. If it is historical actors themselves, and not historians, that establish the links between different ways of understanding sick people, then there is no problem. If a medical historian is able to show that one way of understanding sick people was adopted because people in a different culture at an earlier time adopted a different way of understanding sick people, then a lineage for how this knowledge developed can be built up. Like this, continuity can be established between different ways of understanding sick people without introducing anachronism or slipping into whiggish historiography.

This is what I have done to produce the longue durée history of heart failure I present in this thesis. I am principally concerned with how the disease is diagnosed, so I focused on the practices used to distinguish the patients who are taken to have the disease from those who are taken not to. Specifically, I am most concerned with those practices that are used to define which patients do and do not have the disease. These practices constitute the operational definition of the disease, the practices that are taken to trump all others. Having determined how the diagnosis should be made according to present day actors, I then persistently interrogated this medical literature to try to uncover why it is these actors believe this should be the case. Often they provide some reason for this, usually by citing other medical literature from a slightly earlier period to support their views. Upon examining this cited literature, I often found that the researchers involved adopted quite different diagnostic practices to diagnose heart failure than do the present
day researchers. This earlier method for making the diagnosis of heart failure often resulted in a somewhat different group of patients receiving the diagnosis than would be diagnosed if the practices accepted in the present day were used. Consequently, the present day researchers often explicitly reject the set of practices endorsed by the earlier researchers they cite. In this way, this procedure can uncover different ways of diagnosing, and thus understanding, sick people, which are nevertheless linked together by the historical actors themselves. Continuity is established between these different ways of understanding sick people without me having to identify some timelessly important similarity between them, and without the assumption that the change from the earlier to the later way of understanding sick people was inevitable. The procedure was repeated by persistently interrogating the literature cited by the earlier researchers in support of their views, and in this way I constructed an intellectual and practical lineage of different ways of understanding and diagnosing heart failure.

It is useful to give an example of this procedure in action. To produce this history I start in the present day with a widely accepted way of making the diagnosis of heart failure as articulated in the European Society of Cardiology (ESC) guidelines (2012). As I describe in more detail in the next chapter, the ESC define heart failure as symptomatic cardiac dysfunction – both symptoms and signs of the disease and evidence of cardiac structural pathology or cardiac dysfunction (usually obtained through echocardiography) have to be present for a diagnosis to be made. The ESC say that there are two important forms of cardiac dysfunction – systolic dysfunction (problems with the heart squeezing) and diastolic dysfunction (problems with the heart filling). Focusing on diastolic dysfunction, I looked for the literature the ESC cited in support of their views for what diastolic dysfunction is, how it is measured and why they think it exists.
Focusing on this last question, the ESC cite a review paper (Borlaug and Paulus 2011), who in turn cite another paper from Robert Soufer and colleagues (1985). This work is described as one of the “seminal studies” that identified this form of cardiac dysfunction (Borlaug and Paulus 2011: 671). Soufer et al (1985: 1032) made the diagnosis of heart failure using clinical criteria alone, without the need to demonstrate an abnormality of cardiac structure or function. Soufer et al (1985) therefore diagnose heart failure in a way that the ESC (2012, 2016) currently does not endorse. Furthermore, the ESC does not endorse the purely clinical diagnosis of heart failure (made using symptoms and signs alone) because they believe it to be inaccurate (see next chapter). The ESC (2012; 2016) and Soufer et al (1985) adopt different ways of diagnosing, and thus understanding, patients with heart failure. Nevertheless, these ways of understanding sick people are linked together, because the ESC (2012; 2016) claims to do what they do in part because Soufer et al (1985) did what they did.

Proceeding in this way I built-up a network of published material stretching back to the early nineteenth century. In reality this network is too complex to present in detail, or even to investigate in its entirety. I have had to exercise judgement in deciding which works represented the most important literature in this network. These decisions are guided by the goal of my research, which is to investigate changes to diagnostic practices. Consequently, my focus was on those papers cited to support the use of diagnostic practices. I also had to make judgements about which work was most important to decisions regarding the choice of diagnostic practices. Again this involved the exercise of judgement, guided by factors such as frequency of citation, and claims that a piece of research is ‘seminal for’ or ‘the first to identify’ something.
Following Chang (2004), I have also organised the long history of heart failure into iterations, which I refer to as ‘stages’. This organization is done by me, and not necessarily by my historical actors. I have grouped together work that employs different ways of understanding and diagnosing heart failure according to differences that I believe are important. Different ways of diagnosing heart failure often result in significantly different groups of patients are diagnosed as having the disease. I am also guided by significant changes to the physiological explanation for heart failure, as this has influenced how patients are diagnosed with heart failure historically. Actors sometimes do recognise the divisions I am imposing, as is apparent when they explicitly set out their own work and views in opposition to the views of other earlier actors. And yet, sometimes they do not recognise the differences, and claim agreement with other actors that I argue have a different understanding of how to diagnose heart failure. In these cases I impose this organization into stages anachronistically, as this is necessary to make the presentation of this history intelligible. Even though I recognise this anachronism, I want to emphasize that it functions to introduce discontinuity that historical actors may not recognise, not to introduce continuity that actors may not recognise, which is the problem described above. I concede that others who may undertake a similar project on heart failure in the future may see things differently, but I am confident that how I present the results of my work is reasonable and informative. I would welcome the discussion that further work in this vein would make possible. I provide footnotes through the long history I present to qualify my decisions about how to organize this history.

There are important limitations to the historiographical approach I employ in this thesis. The longue durée history covers two centuries, and is presented in a single chapter. It is necessarily an overview of many of the changes that have
taken place in the historical development of knowledge of heart failure, and how they affect each other. Consequently, I have not made a more in depth study of the wider cultural ramifications of different ways of understanding heart failure. I have also not discussed how changes to society beyond my narrow focus on medical knowledge influenced this history of heart failure. Given the goals of this thesis, I have necessarily sacrificed depth for breadth. One of the central claims I am making is that past ways of understanding sick people influence present day ways of understanding sick people. The way doctors diagnose heart failure today cannot be understood without taking account of the historical development of this knowledge. This claim is impossible to substantiate without attending to a longer and therefore shallower view.

Having said this, I do not think sacrificing depth for breadth significantly diminishes the case I make. My arguments are well supported even by an overview of the historical development of knowledge of heart failure. I am confident that further study of this history will deepen researchers’ understanding of historical contingency of medical knowledge, and continue to support my arguments, even if the significance of some of the episodes I describe is diminished by further work.

I have argued that it is important for me to avoid whiggish historiography. Even though the history I have produced is not a story of progressive development to near-perfect knowledge in the present, it still has many of the features which are sometimes taken to be characteristic of whiggish historiography. I have produced this history by starting in the present and working back into the past. This approach does produce a history which highlights the ways of understanding sick people which constitute the ancestors of ways of understanding sick people that exist in the present. This is at the expense of ways of understanding sick people
that turned out to be historical dead ends, and which therefore exert little influence over present day practice. The methods I use are not designed to recover and re-evaluate knowledge in these historical cul-de-sacs. Even so, I would hope that demonstrating that medical knowledge is historically contingent, and thereby promoting a pluralism in medical diagnostics, might promote historical work that does seek to recover such forgotten and potentially productive knowledge.

3 – Contribution made by this thesis
This thesis makes contributions to several areas of historical, philosophical and medical literature. Two books have been written on the history of heart failure – Saul Jarcho’s (1980) *The Concept of Heart Failure from Avicenna to Albertini*, and Martin Cowie and Michael Holland’s (2002) *A Concise History of Heart Failure: from dropsy to the randomised trial*. Both of these are *longue durée* histories, but they are written assuming progressivist and whiggish historiography. Cowie and Holland start their history in ancient Egypt, and Jarcho starts his in the twelfth century. Both authors examine records of people who today might be thought to have heart failure, and discuss how gradually over time these patients were recognised as such. This approach leads both authors to concentrate on different sources to those that I select. Indeed, Jarcho finishes his history where mine begins, at the beginning of the nineteenth century. Neither book is interested in the question of whether knowledge of heart failure is historically contingent. This is not to diminish the value of these histories. Multiple approaches to historiography are valuable (Jackson 2002). Nevertheless, the historiographical approach adopted by these authors is not suited to the goals of my thesis, and consequently a new history had to be written.

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8 In the preface to his book, Fleming (1997: xii-xv) reviews the ten histories of cardiology written in the twentieth century, noting the whiggish character of each. Fleming also argued that this was not necessarily a bad thing: “Much has been said in this preface about Whiggish historiography and several
My thesis also focuses on the work done by James Hope and James Mackenzie, both of whom are underrepresented in contemporary scholarship on the history of medicine.

Some historians make the claim that medical history can be used to inform medical decision making, and that this is the case because medical knowledge is historically contingent (Jones et al 2015). These historians have not developed the necessary philosophical arguments to make this case strongly, as they do not show how the historical contingency of medical knowledge does not necessarily lead to extreme forms of relativism. My thesis addresses this problem directly. My argument that the development of knowledge over long periods of time should be taken into account if present day views are to be understood also informs a conversation about how exactly medical history can inform medical decision making.

There are many philosophers of medicine who hold that at least some aspects of medical knowledge are not historically contingent. Many philosophers of medicine do argue that disease categories change as culture and values change, and that evaluative decisions must be made to determine what the function of an organ is. Even so, most philosophers of medicine today do not recognise the historical contingency of basic facts about a patient’s biological state. Facts about how and why patients suffer and die are taken to be timeless descriptions of a patient’s physiological state. This is particularly true about physiological knowledge about heart disease. Many philosophers of medicine appear to

examples have been quoted from previous writers. The present author is only too well aware that further examples can be found in his own writing but takes comfort from a remark by D.L. Hull, quoted by Mayr, “if we are not prepared to interpret the past in terms of the present, why should we care about the past?” I have argued that one reason to try to avoid whiggish historiography is because I am trying to learn about the present by looking at the past.
reached conclusions about the physiological role of the heart without checking their conclusions against medical practice, and have not noticed that their conclusions differ from mainstream medical opinion. By attending closely to biomedical practice, specifically regarding the development of knowledge of heart failure, my thesis problematizes these basic biological facts, and argues that they too are historically contingent.

My thesis also informs wider discussions in the philosophical literature about the epistemic status of facts. Over the last decade some philosophers have argued forcefully against various versions of “relativism” and “constructivism”, advancing instead their thesis of “absolutism” (Boghossian 2006; Seidel 2014). These authors argue that the truth or falsehood of factual claims about the world is not relative to anything. Rather, whether claims are true or false is fully determined by the way the world is in itself, independently of any considerations of human culture (Boghossian 2006: 12-13). This is not to say that researchers cannot be mistaken about what the facts are, or about how to reliably produce them (Seidel 2014: 21). It is only to say that researchers are absolutely correct or absolutely mistaken about such matters. Furthermore, these authors also advance the claim that if knowledge of the world is not absolute then people may as well believe anything they want (Boghossian 2006: 4-5). On this view there is no middle ground between a search for absolute truth and extreme forms of ‘believe anything you want’ relativism. In this thesis I argue for a form of relativism about medical facts and knowledge that is sensible, not self-refuting, productive and useful. I show how such an epistemic position can avoid the difficulties produced by the search for universal and timeless truth in diagnostic medicine, whilst at the same time avoiding the pitfall of extreme relativism.
I am not able to address the arguments made by absolutists directly in this thesis. This work is not designed first and foremost to address abstract arguments about the epistemic status of facts. Rather, it is designed to address concrete instances of problems that arise out of the philosophical and medical literatures on heart disease and heart function. Nevertheless, I develop abstract philosophical theses about the epistemic status of medical facts, drawing heavily on Ludwik Fleck’s arguments that there are no universal and timeless facts. This thesis therefore provides important preliminary research for future work that contributes to discussions of epistemic absolutism and relativism.

This thesis makes important contributions to the philosophical literature on Ludwik Fleck’s epistemology. Fleck is often read as an extreme form of relativist, who argued that facts are completely determined by the thought style of a group of researchers. I argue that much of Fleck’s epistemological work is incompatible with this view, and that Fleck can usefully be read as a conventionalist who found a way to reject the possibility of timeless facts without thereby accepting such an extreme form of relativism. Furthermore, when Fleck’s epistemology is discussed in the philosophical literature, these discussions tend to focus on his account of thought styles and thought collectives. This is at the expense of other important aspects of his epistemology, like his discussion of the active and passive elements of knowledge. Fleck’s epistemology is also most frequently discussed as a curiosity in the history of philosophy, and as a forerunner of modern science and technology studies, and rarely put forward as an important and useful philosophical tool (Peine 2011: 489-490). It is no longer quite true to claim that “[n]o one has tried to build their own work using the research of Fleck” (Wettersten 1991: 496), as Andrew Pickering’s “dialectic of resistance and accommodation” was inspired by Fleck’s epistemology (Pickering 1995: 22). Nevertheless, scant
reference to Fleck is made in mainstream philosophy of medicine today.\textsuperscript{9} This thesis contributes to Fleck scholarship by using his epistemology to make sense of the production of medical knowledge of heart failure over the last two centuries, and by focusing on the relationship between the active and passive elements of knowledge.

This thesis focuses on developing a conventionalist reading of Fleck’s epistemology in the context of the development of knowledge of heart failure. As discussed, I see this work as developing a suggestion made in the philosophical literature on health and disease. It is beyond the scope of this thesis to compare the epistemology I produce to the work of other scholars who also seek a middle way between the production of knowledge of the ‘world in itself’ and extreme relativist positions. Consequently, I leave the comparison of the epistemology developed here with work such as Hilary Putnam’s (1977) “internal realism”, Helen Longino’s (1990) “contextual empiricism” and Ron Giere’s (2006) “perspectival realism” for future work.

\textbf{4 – Summary of chapters}

In chapter 1, I substantiate and expand upon the key claims I have made in setting up my thesis. I show that some historians do claim that medical knowledge is historically contingent, and that medical history can be used to inform medical decision making. I show that many contemporary philosophers disagree with this, and see the diagnosis of heart disease and heart failure as particularly unproblematic. I show that the confidence of many philosophers in these matters is problematic, because according to medics the function of the heart is not what philosophers assume that it is. Finally, I draw attention to controversy amongst

\textsuperscript{9} In three contemporary textbooks in the philosophy of medicine (Gifford et al 2011; Solomon et al 2017; Marcum 2017) there is only one brief reference to Fleck’s work (Brochhausen 2017: 183-184).
medics about how to diagnose heart failure, and to the trouble that is encountered in this medical literature as a result of searching for the universal truth about how to diagnose this disease.

In chapter 2, I present my longue durée history describing the development of knowledge of heart failure over the last two centuries. I use this history to argue that the development of knowledge of heart failure has been historically contingent. Knowledge did not have to proceed in the way that it did given the observations researchers made. Nevertheless, I argue it is also clear from this history that knowledge of heart failure could not have developed in any way these researchers’ wanted. I argue that present day knowledge of heart failure is both discovered and invented.

In chapter 3, I review the contemporary philosophical literature on concepts of health and disease in search of an epistemology that can cope with all aspects of medical knowledge being both invented and discovered. Even though some of this literature certainly points the way towards such an epistemology, I argue that none of the available views are entirely suitable for my purposes.

In chapter 4, I look more closely at the work of James Hope. It is a commonplace view that practitioners of “hospital medicine” (Bynum 1994: 25) in this period, like Hope, were only interested in using clinico-pathological correlation to make accurate pathological diagnoses and not in explaining the patient’s symptoms or prognostication. I argue against this commonplace view, by pointing out that Hope certainly did pursue explanation and prognostication, and that he used clinico-pathological correlation to do this. I examine the evidence and arguments that Hope deployed in support of the view that auscultation was a powerful diagnostic tool that could be used to accurately detect heart disease. I draw
attention to how by the end of the nineteenth century, in line with Hope’s views, it was widely accepted that murmurs heard at auscultation were a sure sign of heart disease.

In chapter 5, I look more closely at the work of James Mackenzie. Mackenzie’s work is often understood as spearheading a change from the study of structural abnormalities to the study of functional abnormalities. I argue that this is an unsatisfactory way to view Mackenzie’s work. Instead, I argue that his work is better understood as spearheading a change from the “hospital” medicine of the early nineteenth century to a fusion of Jewson’s (1976) “laboratory” and “bedside” medicine. I examine the evidence and arguments that Mackenzie deployed against the view that murmurs detected at auscultation were a sure sign of disease, and in support of the view that the practice of auscultation had done more harm than good. I draw attention to Mackenzie’s arguments regarding the untrustworthiness of knowledge produced in hospitals, and to his arguments that doctors should re-frame their understanding of heart disease as heart failure.

Hope and Mackenzie both argue that certain facts, like facts about the diagnostic power of auscultation, are obviously true to those who observe the world closely enough. They also appear to contradict each other. In chapter 6, I argue that if a form of conventionalism is applied to medicine, both of these claims can be taken as true, because the apparent differences are the result of Hope and Mackenzie adopting different conventions. Inspired by Engelhardt’s (1996: 228) idea that a form of Kantianism “with a Hegelian accent” can be applied to medicine, I provide a detailed comparison between Poincaré’s and Fleck’s forms of conventionalism. I argue that the role played by conventions in the production of knowledge means that facts, like those produced by Hope and Mackenzie, should be understood as invented.
Fleck articulated his conventionalism in terms of the active element of knowledge (which I compare to Poincaré’s conventions) and the passive element of knowledge (which I compare to Poincaré’s laws). In chapter 7, I argue that, so long as Fleck is read as a conventionalist, the relationship between the active and passive elements of knowledge is such that the passive element of knowledge should be taken to be both invented and discovered. Using the facts produced by Hope and Mackenzie, I argue that although the active element of knowledge is necessary to produce passive facts, it is not sufficient. The passive element of knowledge is therefore not fully determined by the collective will of researchers. Locating this resistance counts as making a discovery. Instead of looking at facts as universal truths, Fleck looks at facts as the resistance to the will of researchers, and shows how facts are both invented and discovered. Following Poincaré, I argue this passive resistance should be viewed as a form of objectivity.

In the conclusion, I address the three questions about the historical contingency of medical knowledge asked above in the light of the work done in this thesis. I find that medical knowledge of heart failure is historically contingent, and that it is possible to make progress and produce objective knowledge in the face of this historical contingency. As it is possible for knowledge to be both historically contingent and objective, researchers can give up the search for universal and timeless knowledge about disease, and embrace a more pluralistic attitude to medical knowledge, without having to worry that they must abandon the possibility of producing objective knowledge as well. I also argue that the historical contingency of medical knowledge affects the evaluation of diagnostic practices in the present day. I apply the Fleckian framework of active and passive associations developed in my discussions of Hope and Mackenzie to the other
transitions described in the *longue durée* history. I argue that it is impossible to understand why historical actors adopted the active associations that they did without taking the work of their predecessors into account. I illustrate the effect that medical history can have on medical decision making with examples drawn from contemporary medical practice.
Chapter 1 – Problematizing heart failure: historical, philosophical and medical perspectives

Introduction

In the introduction I claim that, according to some historians, the history of medicine can inform medical decision making in the present because medical knowledge is historically contingent. I also claim that many philosophers of medicine do not agree with this view, and hold that medical knowledge of heart disease and the function of the heart is uncontroversial and firmly established. I claim that philosophers of medicine have been too quick to draw this conclusion as, if medics are to be believed, the function of the heart is not what philosophers of medicine assume that it is. I would add that how cardiac dysfunction is actually measured when diagnosing heart failure, and the characteristics of the syndrome of heart failure itself, are not as might be expected if the naïve assumptions of philosophers of medicine are taken for granted. In this first chapter I expand on these claims and show that medical knowledge of heart disease (and particularly heart failure) is not as obvious as many philosophers have hitherto assumed (sections 1 and 2).

Showing this might be enough to encourage some scholars to reconsider their views about the epistemic status of medical knowledge, and promote the view that medical knowledge may be historically contingent. However, I doubt that it is. These scholars could simply concede that we have made erroneous assumptions about how to diagnose heart disease, and about what the function of the heart actually is, and that we just need to pay closer attention to medical practice to correct these assumptions. Pointing out that some philosophers and medics hold different views about what the function of the heart is need not be taken to undermine the view that medics have managed to uncover historically
transcendent, universal facts about what the function of the heart is and about how to diagnose heart disease.

What should start to undermine the view that medics have uncovered timeless facts about heart failure is the realization that medics in the present day do not all agree about what heart failure is, how to measure cardiac dysfunction, how to diagnose the disease and which patients have it (section 3.1). Furthermore, the arguments that medics use to support their positions are unconvincing, and often incoherent (section 3.2). Medics do try to produce timeless, universal facts about which observed characteristics can be used to distinguish patients with and without heart failure. They do this by carefully inspecting patients with and without the disease to see which diagnostic practices are accurate and which are not. I argue here that this whole approach investigating how to identify patients with and without disease is fundamentally flawed. The efforts made by medics to discover timeless facts about the characteristics that can be used to distinguish patients with and without disease has lead them to produce unsatisfactory and incoherent arguments about how to diagnose heart failure. That medics are themselves struggling to produce timeless facts about how to diagnose this disease provides good reason to think again about whether this is possible at all, and about whether medical knowledge might be historically contingent.

1 – Perspectives on historical contingency and the function of the heart
Some historians and medical humanities scholars have recently claimed that medical history can inform the practice of medicine in the present day (Jones et al 2015: 623, 639-641; Lovett and Tomkins 2013: 4). Some of these scholars argue that history has this power because it is capable of “diverting the professional gaze away from the technologies of biomedical science towards the social/cultural contexts of the disease/injury experience” (Lovett and Tomkins
2013: 3). On this view, history is used to draw attention to the different ways of experiencing disease, and thereby promote compassion, empathy and humanity in medical care (Lovett and Tomkins 2013: 3-4).

More controversially, other historians argue that history should have a more dramatic role in shaping medical practice. Rather than complementing biomedical science, some historians have argued that medical history should be used to shape how biomedical knowledge itself is produced, understood and used. On this view, medical history is “an essential component of medical knowledge, reasoning and practice” (Jones et al 2015: 623). Such scholars argue that medical history alone can provide insights that “physicians must know in order to be effective diagnosticians and caregivers, just as they must learn anatomy and pathophysiology” (Jones et al 2015: 623).

This is a bold claim, and one which rests on another equally bold claim – that medical knowledge is historically contingent:

> "Medical knowledge is produced through specific social, economic, and political processes. History provides a critical perspective on the contingency of knowledge production and circulation, fostering clinicians’ ability to tolerate ambiguity and make decisions in the setting of incomplete knowledge” (Jones et al 2015: 639)\(^\text{10}\).

On this view, medical knowledge is not something that is simply discovered to correspond to a timeless, mind independent, ‘world in itself’. Rather, medical

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\(^{10}\) Similar sentiments are expressed by Lisetta Lovett and Allanah Tomkins (2013: 4), who say that “[a]n awareness of historical time can foster and enhance a tentative or provisional acceptance of scientific knowledge. This stance is well suited to twenty-first century medicine, in which change is rapid and techniques and technologies might be outmoded swiftly”. David Doukas, Laurence McCullough and Stephen Wear (2012: 335), organizers of the Project to Rebalance and Integrate Medical Education (PRIME) initiative, argue that the “study of histories of medicine and science provides students with a critical perspective on contemporary medicine by requiring them to gain an understanding of how physicians thought and acted in the past. As a result, contemporary ways of thinking become open to critical appraisal. Students can thereby challenge the naïve view that with change comes progress in all cases”. Claims that medical history is valuable to medical training due to the contingency of medical knowledge are not uncommon in the medical humanities literature.
knowledge is (at least in some sense) like fashion, culinary taste, or favourite sport. People in different times and cultures preferred to dress, eat and play differently, without any of these alternatives being more correct than the others.

If medical knowledge is historically contingent, then correct medical knowledge is determined by culture in something like the way that the correct way to dress, eat and play is. On this view, medical knowledge is invented\textsuperscript{11} in particular social, cultural and historical contexts.

So, some historians\textsuperscript{12} claim that medical history can improve medical training and practice because medical knowledge is historically contingent. I want to highlight that, although it is made by historians, this is not an historical claim. This is a claim about what medical knowledge is (e.g. a cultural product), how it is produced (e.g. in specific cultural contexts), and how it can be most effectively deployed (e.g. by remembering medical history). This is an epistemic claim about the use, production and nature of knowledge, and thus a philosophical claim.

Furthermore, this claim is problematic, because many other historians and philosophers do not hold the view that biomedical knowledge is historically contingent. Nowhere is this more evident that with respect to the attitude of some philosophers to the disease and function of the heart.

1.1 – Many philosophers think that knowledge of heart disease and function is not historically contingent

According to K.W.N. Fulford, the correct classification of patients according to their disease state is usually taken to be a matter for scientific discovery, and not

\textsuperscript{11} The historians Andrew Cunningham and Perry Williams have argued that the whole of science is an “invention”, that took place around 1760-1848 (1993: 409). They do this because they feel this term “helps fix the revised view of science as a contingent, time-specific and culture-specific activity, as only one amongst the many ways-of-knowing that have existed, currently exist, or might exist” (Cunningham and Williams 1993: 428). I use the term invention in the same way, and applying it to medical knowledge.

\textsuperscript{12} I do not claim that this view is held by all, or even a majority, of historians. As I argue in chapter 3, many historians do not problematize some aspects of disease in their work. I claim that this view is held by at least some historians and that I think it has promise and deserves close consideration.
to involve any evaluative decision on the part of doctors or patients. “In the traditional fact-centred model of medicine, as noted, diagnosis is assumed to be essentially a matter for medical science. This is because in most of medicine diagnosis does indeed appear to be value free” (Fulford 2004: 210). Fulford argues against this view, and holds that evaluative decisions are always relevant to decisions about the disease state of a patient – a view he calls the “two-feet” principle, as diagnostic decisions always stand on both facts and values (2004: 209).

However, this division between factual and evaluative considerations entails that medical scientists, without making any evaluative decisions, do discover facts that are relevant to decisions about disease status. These facts are therefore universal for all, insulated from cultural considerations, and timeless. Fulford (2004) argues that in the case of psychiatric disease, evaluative considerations are very important. But in the case of many bodily diseases, he argues that evaluative decisions are much less important, and this is what makes much of medicine appear value free (2004: 210). He argues that this is because all people, regardless of historical or cultural concerns, always have and always will disvalue many somatic conditions. He chooses myocardial infarction to illustrate his point. “A heart attack [myocardial infarction], as noted, is a bad condition by anyone’s standards. Hence the evaluative part of the diagnosis is unproblematic. Hence it can be (and properly is) ignored in practice” (Fulford 2004: 212). So, for Fulford, it is only universal, timeless, and value free facts that are relevant to deciding whether or not a patient is having a heart attack.

Other philosophers have made similar claims. H. Tristram Engelhardt has argued, for instance, that even though many diseases (including bodily diseases) are not stable across cultures, diseases like myocardial infarction “can be
straightforwardly acknowledged as diseases” and need not be termed “ego-
dystonic myocardial infarction” (Engelhardt 1985b: 173). On this view, personal
choices and cultural factors just do not enter into the decision about whether or
not a patient is having a heart attack.

Confidence that the decision about whether or not someone has a heart disease
is straightforward and not culturally informed is not reserved for myocardial
infarction. Scott DeVito argues that valvular disease of the heart is similarly
straightforward to recognise. DeVito argues that, in general, diseases are not
stable across cultures. However, he concedes that valvular disease of the heart
conforms to the views of philosophers, like Christopher Boorse, who argue that
knowledge of disease states should be stable across cultures. According to
DeVito, incompetent heart valves always reduce life expectancy, and are
therefore unproblematically considered as diseases:

“Holosystolic heart murmurs provide a good example of disease-states that
conform to Boorse’s view. A heart with a holosystolic murmur is diseased
because (i) the way the blood circulates through the heart contributes to the
continued living of a person and (ii) the difference in the way that blood
circulates in a holosystolic heart (blood is regurgitated back into the left
atrium upon contraction) produces a negative impact on the life span of
persons who have holosystolic heart murmurs as compared with those
persons who do not have holosystolic heart murmurs” (DeVito 2000: 541).

The function of the heart is also widely held to be straightforward to determine
and independent of culture. Engelhardt, who is well known for arguing that
knowledge of biological function is not simply discovered independently of social,
historical and cultural considerations, singles out cardiac and respiratory function
as things about which there will be cross-cultural agreement (1980: 44-45). Many
philosophers go even further, and claim that it is perfectly obvious that the
function of the heart is to circulate adequate quantities of blood. For instance,
Boorse claims that “it is obvious that the function of the heart is to circulate blood”
According to Elselijn Kingma “[t]he qualitative normal function of the heart is to pump blood, and the quantitative normal function of the heart is to pump blood at a certain rate, with a certain output, with regularity, etc” (Kingma 2010: 249). Lennart Nordenfelt says that “[t]he heart pumps blood. The result of the pumping, the distribution of blood, is the natural function of a heart in a human” (2006: 17). Nordenfelt adds that the function of the heart is more easily delineated than the function of other organs, which makes it the most celebrated example of an organ with a natural function (Nordenfelt 2006: 16-18).

This view is corroborated by Arno Wouters in a review of the philosophical literature on biological function. Wouters (2005) identifies five main approaches to the analysis of biological function. Despite their differences, Wouters reports that all these philosophers agree that the function of the heart is “to propel blood” (2005: 125). Indeed, Wouters claims that in this literature the function of the heart “is the philosopher’s favourite example and quite often the only one mentioned” (2005: 125). There is widespread agreement amongst philosophers that the function of the heart is straightforward to determine, discovered independently of cultural considerations, and is to circulate adequate quantities of blood. If these widely held views of philosophers are correct, then medical knowledge is not historically contingent in any practically relevant sense, at least not in the case of the diagnosis of heart disease. If the philosophers mentioned here are correct, then the historians mentioned above are wrong, and one of the main arguments used to support the view that medical history is valuable to medical practice is fatally undermined.

1.2 – These views about heart disease have been formed too quickly
Even though many philosophers hold that the function of the heart is obvious, and the diagnosis of heart disease is straightforward, it is immediately apparent
from historical and medical literature that things are not quite as simple as they have assumed. A brief survey of the historical and medical literature on heart disease and heart failure reveals that the views of many philosophers of these matters differ significantly from the views of medics. If different communities of researchers can come to quite different opinions about the diagnosis of heart disease and the function of the heart, then the historians above are perhaps correct to claim that medical knowledge is historically contingent.

The connection between myocardial infarction and heart attack, for example, is not as straightforward as it is made out to be. As Margolis (1976: 245-246) and Feinstein (1967: 68-69) have noted, occlusion of the blood vessels supplying the heart may produce no pain, just as pain characteristic of a heart attack may occur without myocardial infarction. Such instances of “silent myocardial infarction” have been reported in 9-37% of cases with forms of non-fatal myocardial infarction (Thygesen 2012: 2559). There are also gendered differences with respect to the recognition of myocardial infarction that are diagnostically important that are recognised in the medical literature:

“The current description of “typical” cardiac symptoms is based primarily on the experience of white, middle-aged men, with deviations called “atypical.” Researchers have speculated that this label contributes to misunderstandings in clinicians and lay individuals, leads to inaccurate diagnoses, and causes women to delay seeking treatment” (McSweeney 2003: 2619).

The arguments made above that the diagnosis of myocardial infarction is unproblematic and independent of culture are grounded on the assumptions that the experience of this pathology is so uniformly and severely awful that patients could neither think that what they were experiencing was not a disease, nor think that their experience was dissimilar to other patients with this pathology. As some patients with myocardial infarction are asymptomatic, and even symptomatic
patients can have sufficiently dissimilar clinical presentations as to cause gendered differences in the diagnosis of this pathology, these assumptions are untenable. The prognosis of patients with incompetent heart valves is also highly heterogeneous (Goldstone et al 2015). As I will show in this thesis, the recognition of valvular disease of the heart is historically contingent.

Some historians have convincingly argued for the role of social and cultural factors in the “making” of disease categories like coronary thrombosis and myocardial infarction (Lawrence 1992: 62, Howell 1984, Aronowitz 1998). Christopher Lawrence, in particular, has drawn attention to the work of early twentieth century doctors (including that of James Mackenzie) who recognised that blockage of the blood supply to the heart could cause angina pectoris, a syndrome of crushing chest pain (Lawrence 1992: 61). And yet, these doctors also identified that this syndrome could also occur in the absence of coronary artery pathology, just as other doctors have noticed closer to our own time (Feinstein 1967: 68-69). In contrast to the views of more recent doctors, these early twentieth century doctors did not take coronary artery pathology to be the basis of a distinct disease, but rather used the experience of angina pectoris (amongst other things) to do this (Lawrence 1992: 58,60). The lack of robust clinical-pathological correlations has left doctors with choices about how to classify the sick, and different doctors in different times have made different decisions about how to do this. Consequently, Lawrence has argued that coronary thrombosis and myocardial infarction are not “unproblematic natural entities” that have a “manifestly obvious character” (Lawrence 1992: 52).
Myocardial infarction is not as straightforward to identify as Fulford and Engelhardt suggest\(^\text{13}\).

Attending to medical literature on heart failure also reveals that many philosophers have been too quick to assume that the function of the heart is obviously to pump adequate amounts of blood around the body. If this were the function of the heart, then patients who were pumping adequate amounts of blood around their bodies should not be considered to have heart failure. Perhaps surprisingly, the commonly accepted definition of heart failure shows that this is not how heart failure is understood by doctors today. For instance, according to the European Society of Cardiology (ESC) guidelines on the diagnosis and treatment of heart failure:

“Heart failure can be defined as an abnormality of cardiac structure or function leading to failure of the heart to deliver oxygen at a rate commensurate with the requirements of the metabolizing tissues, despite normal filling pressures (or only at the expense of increased filling pressures)” (ESC 2012: 1792).

It is this bracketed clause “or only at the expense of increased filling pressures” that undermines the widely held view about the function of the heart. This clause entails that a patient can be in heart failure even if their heart is pumping enough blood to meet the requirements of metabolizing tissues, so long as the pressure of the blood within the chambers of the heart as it fills are raised. It is even possible for patients to have “high output heart failure”, where their hearts are pumping a greater than adequate volume of blood around their bodies (Metha and Durby 2009). According to doctors in the present day, the function of the

\(^{13}\) It is also worth noting that Lawrence (1992: 75) closes with these remarks which further problematize the supposedly obvious and unavoidable disease category of coronary thrombosis: “The status of coronary thrombosis as a disease entity has recently been questioned by some cardiologists who prefer to see it as an “incident”. Are we watching the beginnings of the disappearance of so definite, material and natural category as a heart disease? If so, how will future generations explain this disappearance?”
heart is not simply to pump blood, but rather to pump blood at normal filling pressures. This is at odds with the views commonly expressed by philosophers, and shows that the function of the heart is not as obvious as many philosophers have assumed that it is.

2 – The diagnosis of heart failure
2.1 – The measurement of cardiac dysfunction
Even if physiological definitions of heart failure are not what might be intuitively expected, present day discussions of the diagnosis of heart failure do not focus on physiological discussions of the function of the heart. In-line with trends in evidence-based medicine to downplay the importance of pathophysiological mechanism in clinical decision making, and with trends in psychiatry to operationalize diagnostic criteria for psychiatric diseases, medics discussing the diagnosis of heart failure tend to focus on the diagnostic practices that are actually employed to distinguish diseased and non-diseased patients. Pathophysiological definitions of heart failure, like the one above, tend to be viewed by medics today as too vague to be of much use in the clinic (Sutton and Chatterjee 2008: 1). In other words, medics discussing the diagnosis of heart failure today tend to focus on how heart failure is measured.

How doctors actually measure cardiac dysfunction when diagnosing heart failure may also surprise philosophers. The ESC does not leave doctors free to interpret the above physiological definition of heart failure as they see fit. Rather, the ESC also gives an operational definition of heart failure. The ESC specifies a set of measurements that serve as criteria for the diagnosis of heart failure, thereby specifying (as much as is possible in a document) how the diagnosis of heart failure should be carried out in practice. According to the ESC, for a diagnosis of heart failure to be made, there are three criteria that must be met. The patient
must have some of the *symptoms* (observations that the patient reports to the doctor) that are taken to indicate heart failure. Secondly, the patient must have some of the *signs* (observations the doctor makes of the patient during the physical examination) taken to indicate heart failure. Thirdly, the patient must have what the ESC describes as *objective evidence of abnormalities of cardiac structure or function* (ESC 2012: 1795). For a diagnosis to be made a patient must have either evidence of abnormal cardiac structure (such as abnormal heart valves), or a test result that demonstrates or indicates the presence of some form of cardiac dysfunction, in addition to the presence of symptoms and signs of heart failure.

If cardiac dysfunction was as closely related to the inability to circulate adequate volumes of blood as philosophers have assumed, then one might expect the measurement of cardiac dysfunction to focus on the measurement of cardiac output. This might be complemented with measurements of how well perfused the patient’s tissues are. Such measurements are possible to make\(^{14}\), and yet they play no role in the ESC’s operational definition of heart failure. The practices that the ESC does say should be used to measure cardiac function are quite complex, and require a basic understanding of cardiovascular physiology to grasp them.

The human heart has two sides: left and right. Blood flows from the left side of the heart to the tissues of the body in the *systemic* circulation, where oxygen is delivered to the tissues, and carbon dioxide is removed from them. Blood returns

\(^{14}\) For example, measurements of “cardiac index” (the ratio of cardiac output to body surface area, which is a proxy for metabolic rate) are not used by the ESC to define heart failure. Alternatively, the “Fick principle” (which is that (cardiac output times arterial oxygen concentration) minus (cardiac output times mixed venous oxygen concentration in the right side of the heart) provides a measure of metabolic rate) allows an inverted measure of cardiac output divided by metabolic rate to be made from measurements of the arterio-venous oxygen difference. This is not used by the ESC to define heart failure either.
to the right side of the heart where it is then pumped to the lungs in the *pulmonary* circulation. In the lungs, carbon dioxide is exchanged for oxygen, and the oxygen-rich blood proceeds to the left side of the heart. Here the cycle begins again, and oxygen-rich blood is once more pumped through systemic circulation to the tissues of the body (see figure 1).

Each side of the heart is divided into two chambers: the atrium and the ventricle. Blood flows from the atria to the ventricles. Blood flows from the left atrium, to the left ventricle, through the systemic circulation, to the right atrium, then the right ventricle, through the pulmonary circulation before returning to the left atrium once more.

At the exit of each of these four chambers, valves prevent the retrograde flow of blood (see figure 2). The co-ordinated contraction and relaxation of each of these chambers acts to pump blood forwards in the circulation. Each time the heart beats, both atria will contract together to pump blood into the ventricles, which will then contract together to pump blood into either side of the circulation. The phase of the heart's action where it is squeezing blood out of the ventricles and into the circulation is called *systole*. The phase of the heart's action where the ventricles are filling is called *diastole*. Cardiac dysfunction is often discussed in terms of systolic dysfunction (dysfunction of squeezing) and diastolic dysfunction (dysfunction of filling) (ESC 2012: 1792-1793).

Perhaps the most important measurement of cardiac function is the *left ventricular ejection fraction* (LVEF), which is the amount of blood ejected from the left ventricle during systole (ESC 2012: 1792). A reduced LVEF is often taken as evidence of systolic dysfunction (ESC 2012: 1792). There are many other measurements of cardiac dysfunction in addition to reduced LVEF. These include
Figure 1: Double circulatory system. The pulmonary circulation flows through the lungs and the systemic circulation flows through the liver, gut, kidneys and other organs. Oxygenated blood in red, deoxygenated blood in blue. (Image adapted from Shutterstock, image ID 365494934 “Circulatory system”. Copyright NelaR).

Figure 2: Anatomy of the heart. (Image adapted from Shutterstock, image ID 46345582 “Progress of blood through the heart”. Copyright Blamb).
increased thickness to the muscular wall of the heart, increased chamber size, and various measurements of blood flow that indicate that the chambers of the heart are not filling as they should (ESC 2012: 1800). These latter sorts of measurement do not need to be described in detail here, but as they indicate that the chambers of the heart are not filling properly they are taken as evidence of diastolic dysfunction (ESC 2012: 1803).

The main method used to measure cardiac function (as well as to look for structural abnormalities and muscular wall thickening) is the ultrasound of the heart, or echocardiography. The ESC has been recommending that echocardiography be used to check for objective evidence of cardiac dysfunction since the mid-1990s, arguing that “confirmation by echocardiography of the diagnosis of heart failure and/or cardiac dysfunction is mandatory and should be performed shortly following the suspicion of the diagnosis of HF [heart failure]” (ESC 2008: 2398; see also Task force on heart failure of the ESC 1995: 745; Remme and Swedberg 2001: 1532; ESC 2005: 1118). “Of the several imaging modalities available, echocardiography is the method of choice in patients with suspected HF [heart failure] for reasons of accuracy, availability (including portability), safety and cost” (ESC 2012: 1800).

Even though echocardiography is perhaps the most important method for detecting cardiac dysfunction, it is not thought to be infallible. This is particularly the case for the measurement of diastolic dysfunction. In 1995, the ESC reported that “[n]o practically useful Doppler echocardiographic guidelines are yet available for diagnosing diastolic heart failure” (Task force on heart failure of the ESC 1995: 745). In 2001, there were still “no universally accepted minimal criteria for the diagnosis of diastolic dysfunction” (Remme and Swedberg 2001: 1533). The ESC at this time recommended that if there was doubt about the diagnosis
of heart failure due to uncertainty about the presence of diastolic dysfunction, then the matter should be settled using a therapeutic trial. Here, the patient is treated for heart failure with a diuretic (a drug that acts to make the patient produce more urine and thus reduce the amount of water they have in their body), to see if this improved the patient’s condition (Task force on heart failure of the ESC 1995: 742; Remme and Swedberg 2001: 1527). If the patient improves following treatment for heart failure, then the diagnosis of heart failure is deemed warranted.

In 2005 and 2008, the ESC appears more confident about how to measure diastolic dysfunction echocardiographically, and describe three different filling patterns which they said can be used to detect and stage the degree of diastolic dysfunction (ESC 2005: 1121; ESC 2008: 2399). More recently, however, confidence in the echocardiographic assessment of diastolic dysfunction appears to have waned a little. In 2012, the ESC claimed that “no single echocardiographic parameter is sufficiently accurate and reproducible to be used in isolation to make the diagnosis of LV [left ventricular] diastolic dysfunction” (ESC 2012: 1800). The ESC also stated that the diagnosis of heart failure with preserved ejection fraction was more difficult than the diagnosis of heart failure with reduced ejection fraction. This was “because it is largely one of exclusion, i.e. potential non-cardiac causes of the patient’s symptoms (such as anaemia or chronic lung disease) must first be discounted” (ESC 2012: 1793). For a diagnosis of heart failure with a preserved ejection fraction to be made, the most recent ESC guidelines state that elevated levels of natriuretic peptides (hormones that are thought to be released as a result of an increase in pressure in the atria) should be recorded along with echocardiographic evidence of cardiac dysfunction (ESC 2016: 9). This all indicates that the ESC is not confident that echocardiographic
measurements of cardiac function in heart failure with preserved ejection fraction are sufficiently specific to warrant a diagnosis on their own.

In summary, there are two main sorts of heart failure, classified according to the sort of dysfunction detected at echocardiography. The first is heart failure with a reduced ejection fraction, comprised mainly of patients thought to have systolic dysfunction. The second is heart failure with preserved ejection fraction, thought to be mainly comprised of patients with diastolic dysfunction. And yet, the actual measurement of cardiac function is not thought to be straightforward, and echocardiography is supplemented by diagnostic practices such as therapeutic trials, and the measurement of natriuretic peptides.

2.2 - The syndrome of heart failure
All this seems at odds with the confidence of many philosophers that the function of the heart is obvious, and presumably simple to measure. The symptoms and signs of heart failure, which need to be present for a diagnosis to be made, are also different to what might be expected by philosophers and other lay people. Taking the function of the heart as simply to pump an adequate volume of blood around the body, a patient in heart failure might reasonably be expected to present in “circulatory shock” – which is a medical term for a life threatening condition resulting from under-perfusion. Such a patient might be expected to present acutely (suddenly) with collapse, fainting (syncope), turning blue (cyanosis), pallor and prolonged capillary refill times. Whilst patients in heart failure do sometimes present in circulatory shock (ESC 2008: 2391), this is not the dominant clinical picture.

Most often, heart failure is not an acute condition that arises quickly and without warning. Rather, the most common form of heart failure is chronic heart failure, where the patient’s condition gradually gets worse over a long period of time (The
Task force on heart failure of the ESC 1995: 741; Remme and Swedberg 2001: 1528). Furthermore, patients in both acute and chronic heart failure typically present with a syndrome of \textit{breathlessness and fluid accumulation} (ESC 2012: 1793, 1795). Symptoms of breathlessness include breathlessness on exertion, paroxysmal nocturnal dyspnoea (fits of breathlessness at night in bed), and orthopnoea (breathlessness induced by patients lying on their backs). Signs of fluid accumulation include pulmonary oedema (fluid collecting in the lungs), ascites (fluid collecting in the abdomen), ankle swelling, enlargement of the liver, weight gain, and jugular distension. Heart failure is not characterized by an acute syndrome of circulatory shock, as might be expected, but rather by a chronic syndrome of breathlessness and fluid accumulation.

The treatment for heart failure is also different to that which might be expected if the heart is conceived of as a simple pump. If a simple mechanical pump is not pumping enough fluid, one might expect that the appropriate action to take would be to make the pump work harder, to pump more fluid each stroke, or to pump more quickly. In the treatment of heart failure, however, the use of positive ionotropic agents (which make the heart contract more strongly each beat) such as digitalis is at best controversial (Sutton and Chatterjee 2008: 41). In contrast to this, the use of β-blockers (which act to slow the heart rate) is entirely uncontroversial. Instead of focusing on increasing the work done by the heart, treatment is often focused on relieving the fluid accumulation associated with heart failure. Doctors treat this fluid accumulation using diuretics, which is why diuretics feature in the therapeutic trial for heart failure mentioned above. The diuretics used (e.g. loop diuretics such as furosemide) do not act on the heart at all, but rather on the kidney. Another mainstay of treatment for heart failure is the angiotensin-converting-enzyme (ACE) inhibitor, which acts to block the actions
of a hormonal axis that promotes fluid accumulation. So many treatments for heart failure target renal and endocrine systems, instead of acting on the heart directly.

The diagnosis of heart disease is not as simple as many scholars have assumed. As has been shown historically, the diagnosis of heart attack and myocardial infarction are neither unproblematic nor independent of culture. If doctors are to be believed, then the function of the heart is not what many philosophers say that it is. If the heart is considered as a simple pump, then how cardiac dysfunction is measured also appears surprising. The symptoms and signs that characterize heart failure, and the treatments for this disease, are not what might be expected to follow from this commonplace philosophical opinion. All of these things contribute to the diagnosis of heart failure, which seems quite different and much more complex than many philosophers have hitherto suspected.

Returning to the discussion of the historical contingency of medical knowledge, the differences between the views of many scholars and medics about the diagnosis of heart disease does not in and of itself show that historians are correct to claim that medical knowledge is historically contingent. Scholars might simply accept that their assumptions about the diagnosis of heart disease and the function of the heart are wrong\textsuperscript{15}. Scholars may simply defer to doctors’ expertise in this matter, and return to their arguments about what the correct analysis of heart disease and function are in the light of this medical knowledge. If scholars accept that their assumptions about heart disease and function were made in haste, then the view that medical knowledge is simply discovered might be preserved.

\textsuperscript{15} It is also possible for philosophers to argue that it is the doctors who are wrong.
3 – Controversy exists amongst medics about how to diagnose heart failure
Once again, things are not so simple. Scholars cannot simply defer to the view that medics hold about the diagnosis of heart disease and heart failure, because there are important disagreements amongst medics about the diagnosis of heart disease, and heart failure in particular.

Since the turn of the new millennium, many doctors have expressed concerns about how the diagnosis of heart failure is being carried out in general practice. Some doctors are particularly concerned that general practitioners often do not refer patients they suspect of having heart failure for echocardiographic examination, so that the presence of cardiac dysfunction can be confirmed. These doctors argue that attempts to make the diagnosis clinically, thereby relying on the patient’s history and physical examination alone, leads to diagnostic error. According to one review article (cited by ESC 2012 and ESC 2016)¹⁶:

“Both over diagnosis and under diagnosis occur commonly in clinical practice. Among the patients thought to have heart failure by their General Practitioners, a significant percentage do not have any demonstrable abnormality of cardiac function on objective testing. Thus they are incorrectly labelled and inappropriately treated. Only 25 to 60% of the patients referred for evaluation of cardiac dysfunction because of a clinical suspicion of heart failure fulfil the diagnosis…..Although high quality diagnosis is absolutely essential for adequate management, many doctors, especially in primary care, currently rely on diagnosing heart failure on clinical grounds alone, without further investigation, disregarding the available guidelines” (Fonseca 2006: 96).

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¹⁶ The same claim has frequently been made in the medical literature since the new millennium. For instance, over a decade ago doctors claimed that “[h]eart failure is difficult to define and to diagnose…..Diagnosis by clinical assessment is difficult and correct in less than half of cases confirmed by echocardiography” (Fuat 2003: 1). More recently, other doctors claimed that “the current diagnosis of heart failure in primary care is often inaccurate. In one recent UK study, only 34% of patients with an existing clinical label of heart failure had their diagnosis confirmed at echocardiography and review” (Hobbs et al 2010: 1774).
Overdiagnosis is the making of a diagnosis where it is not warranted, and underdiagnosis is the failure to make a diagnosis when it is. These doctors argue that the failure to confirm the clinical suspicion of heart failure by referring the patient for echocardiography leads to both these forms of diagnostic error, and therefore to the mismanagement of patients.

Educating general practitioners about how unreliable purely clinical diagnoses are appears to be the appropriate action to remedy this worrying state of affairs. Again, things are not so simple. Since the new millennium, several doctors have expressed their concerns about the usefulness of echocardiography as a method for measuring cardiac dysfunction. This is particularly the case in the measurement of diastolic dysfunction and the diagnosis of heart failure with a preserved ejection fraction:

“Although there are recommendations for the diagnosis of left ventricular systolic dysfunction and valvular disease, the existing ESC Guidelines on the diagnosis of diastolic heart failure have not yet gained general acceptance” (Fonseca 2006).

Some doctors have been so concerned about the usefulness of echocardiographic measurements of diastolic dysfunction that they have challenged the ESC’s requirement that objective evidence of cardiac dysfunction must be demonstrated for a diagnosis of heart failure to be made. Instead, these doctors argue that the diagnosis of heart failure should be a clinical diagnosis, where echocardiography is used not to make the diagnosis, but rather to subclassify patients according to the type of heart failure that they have. According to Ramachandran Vasan and Daniel Levy17:

17 These two doctors were the leaders of the world renowned and prestigious Framingham Heart Study in the U.S.A.
“Recently, the European Society of Cardiology proposed guidelines for the diagnosis of CHF [congestive heart failure]. These guidelines require objective evidence of LV [left ventricular] dysfunction for a diagnosis of CHF.....We think that although the assessment of LV systolic function is critical in determining the optimal treatment for patients with CHF, the diagnosis of CHF is clinical and should not be made on the basis of LV ejection fraction (EF).....Requiring objective evidence of LV dysfunction to diagnose CHF would inevitably lead to an underestimation of the occurrence of DHF in the community. This is because although definitive evidence of LV systolic dysfunction is easily obtained from the LVEF, unequivocal evidence of LV diastolic dysfunction is difficult to obtain by noninvasive methods.....Legitimate concerns about false-positive diagnoses of CHF can be adequately addressed by requiring clinicians to consider carefully and to rule out alternative diagnoses that can masquerade as CHF before making a diagnosis of CHF” (Vasan and Levy 2000: 2118-2119).

According to the view expressed here, the diagnosis of heart failure is largely a diagnosis of exclusion. The patients’ symptoms and signs signal to the doctor that the patient may have heart failure, and if the doctor cannot find an alternative explanation for the patient’s presentation, a diagnosis of heart failure is deemed warranted. Those patients with a reduced LVEF are then sub-classified as cases of heart failure with reduced ejection fraction. Those patients without a reduced LVEF are sub-classified as cases of heart failure with preserved ejection fraction, regardless of whether or not echocardiographic evidence of cardiac dysfunction is found. This difference in diagnostic practice between these doctors and those that advocate the ESC’s approach reportedly select significantly different groups of patients as having heart failure (Di Bari 2004), making this difference of opinion about how to diagnose heart failure a significant problem for clinical practice, as well as for medical research into epidemiology and treatments for heart failure.

Controversy over how best to diagnose heart failure persists into the present day. Various combinations of clinical symptoms and signs, echocardiographic findings, natriuretic peptide measurements, radiographic findings and intra-cardiac blood pressure measurements taken at cardiac catheterization have
been suggested and used to diagnose heart failure for clinical and research purposes (Yturralde and Gaasch 2005). “Heart failure with preserved ejection fraction is difficult to define as illustrated by the various classifications proposed by experts and by disparate inclusion criteria of clinical trials” (Senni et al 2014: 2798). This controversy still centres on the requirement to provide measurement of diastolic dysfunction. “Some trials require evidence of diastolic dysfunction, whereas others do not” (Senni et al 2014: 2809). The controversy surrounding the diagnosis of heart failure with a preserved ejection fraction runs so deep that some researchers have called for a fundamental rethink about how these patients are classified. It has been suggested that it is actually kidney dysfunction that determines whether patients with diastolic dysfunction become symptomatic (Victor and Barron 2010; Sato 2013), and that treatment of kidney dysfunction might be a useful way of managing patients in heart failure (Damman et al 2007). “Perhaps heart failure as we traditionally think of it is the wrong paradigm to pursue as we try to understand ‘heart failure’ with a preserved ejection fraction” (Oghlakian et al 2011: 531-2, 538).

Philosophers cannot just defer to the views that medics hold about how to measure cardiac dysfunction and how to diagnose heart failure, because there is significant disagreement within the medical community about these matters. Perhaps philosophers would be able to come to some conclusion about which views are correct by looking at the arguments that medics put forward in support of their positions. In this way, philosophers might be able to choose the correct views about how to diagnose heart failure from all these different views suggested by medics. Yet again, things are not so simple, because the arguments deployed by medics to support their views about how to diagnose heart failure are highly problematic.
3.1 – Arguments used by medics to support their views about how to diagnose heart failure are unconvincing

Not so long ago, doctors did not routinely measure cardiac function in order to make the diagnosis of heart failure. It is only in the last few decades that many doctors (particularly cardiologists) have argued that the measurement of cardiac function is important for diagnostic purposes:

“The clinical diagnosis of heart failure has, for decades, been based on the medical history, physical examination, chest x-ray, and response to treatment. The widely accepted diagnostic criteria for congestive heart failure (CHF) use physical signs and symptoms; the criteria do not require an objective measure of left ventricular (LV) size or function. With the development of techniques that made assessment of the LV ejection fraction (EF) widely available, it became fashionable to incorporate this index of contractile function into our definition of heart failure” (Yturralde and Gaasch 2005: 314).

The researchers state that the decision to incorporate measures of cardiac function into the criteria for the diagnosis of heart failure was made because this had become the “fashionable” thing to do. This suggests that there were no arguments made in support of the view that echocardiographic evidence of cardiac dysfunction needed to be provided for a diagnosis of heart failure to be made. Other researchers, however, state that arguments were advanced to support this position:

“This requirement for evidence of LV [left ventricular] dysfunction stemmed from evidence of inaccuracies in the clinical diagnosis of CHF, especially in women, the elderly, and the obese” (Vasan and Levy 2000: 2118).

According to these researchers, doctors decided that it was important to measure cardiac function when diagnosing heart failure because of evidence that the clinical diagnosis of heart failure, made by relying on the patient’s symptoms and signs and without measuring cardiac function echocardiographically, was not accurate.
Discussions of the evaluation of diagnostic tests is often entirely focused on the empirical assessment of diagnostic accuracy (Hawkins 2005; Weinstein 2005; Smith 2006: 33-52). As I show below, this approach is only valuable when there is consensus over how to identify diseased patients, and distinguish them from patients with other diseases. When which patients have a disease is being debated, the evaluation of diagnostic tests cannot be made by measuring diagnostic accuracy. Under these circumstances other methods of evaluating diagnostic tests have been put forward, but none are uncontroversial. For instance, some researchers argue that when there is no consensus about how to identify patients with a disease, doctors should draw on their knowledge of the pathophysiology of disease in order to decide how to do this (Knottnerus and Muris 2009: 55). This is a controversial claim, because of the low regard many proponents of evidence based medicine have for pathophysiological knowledge. Adopting this view would mean that pathophysiological knowledge would provide the foundation for evidence-based medicine, because evidence of therapeutic efficacy depends upon accurate diagnoses being made, and accurate diagnoses would on this view depend on knowledge of pathophysiology. In any case, relying on pathophysiology would only beg the question of why the pathophysiological knowledge is accepted. As I show in chapter 2, knowledge of pathophysiology is as historically contingent as any other sort of medical knowledge. Reitsma et al (2009) have recently reviewed the methods used in medical literature to evaluate diagnostic tests when there is no consensus about how to identify diseased patients, and conclude that no method (including expert consensus and latent class analysis) is without significant problems. They recommend that under these circumstances “the diagnostic accuracy paradigm may be abandoned in favour of alternative methods for evaluating tests” (Reitsma 2009: 803). Along with other
researchers in recent years, Reitsma et al (2009: 803) advocate making assessments of diagnostic tests based on the outcomes achieved using these tests (Lijmer and Bossuyt 2009: 63-95; Gluud and Gluud 2005: 725). Diagnostic tests are sometimes evaluated based on their ability to predict useful therapeutic or prognostic outcomes. This practice, however, is not without its own unique difficulties. Shifting attention to predicting outcomes leads to a proliferation of classification schemes for patients, as a single classification scheme is unlikely to capture all interesting outcomes. “The trouble is that homogeneity with respect to one of these characteristics does not imply homogeneity with respect to the others. So if we are trying to decide how to diagnose a disease, we need to know why we want to make the diagnosis, because different purposes of diagnosis can lead to different disease classification schemes” (Newman and Kohn 2009: 2).

The nosological pluralism advanced here is not itself a problem, but it is in conflict with efforts to find the single absolutely correct way of diagnosing disease (Binney 2015). Advocates of this approach often advise that the accuracy of the diagnostic test under evaluation be assessed for accuracy in the early stages of its evaluation, and before the outcomes it can achieve are assessed. The ‘paradigm of diagnostic accuracy’ is not so much abandoned, as added to. In the case of heart failure, the UK’s National Institute for Health and Clinical Excellence’s (2010) report on the diagnosis of heart failure incorporates measures of diagnostic accuracy into its assessments of the cost-effectiveness of different diagnostic approaches. The measurement of diagnostic accuracy still plays an important role in the evaluation of outcomes18.

18 Reitsma et al (2009) also point out that the interpretation of results of studies that assess outcomes do not fully determine whether the diagnostic test is useful or not. “Whenever the index test results fail to show the hypothesized test results, more than one conclusion is possible – the index test has low validity, the theory about the target condition is not correct, or both” (Reitsma et al 2009: 804).
In medicine, the measurement of diagnostic accuracy means something specific. To measure the accuracy of a particular diagnostic practice or test, the results of the test under investigation (commonly referred to as the index test) are compared to the results of a test which is trusted by the researchers (commonly referred to as the reference standard\textsuperscript{19}). An accurate index test is one which always returns the same result as the reference standard, and an inaccurate index test does not.

Studies which measure the diagnostic accuracy of an index test all have a similar structure. They start by selecting a population of patients that contains both diseased and non-diseased patients. The patients with the disease are distinguished from the patients without the disease using the reference standard. The index test is then used to assess the patients in the population, to see if the results are the same as those given by the reference standard. This gives rise to two further important properties of the index test – its sensitivity and its specificity.

The sensitivity of an index test is the probability that it will give a positive test result in those patients who have the disease in question, and the specificity of an index test is the probability that it will return a negative result in patients that do not have the disease. A sensitive test will pick up all the cases that have the disease (it will return no false negatives), even if it may also indicate that some patients have the disease when they do not (and thereby return false positive results). A specific test will only return positive test results if the patient actually has the disease (no false positives), even if it may also fail to pick out patients who actually do have the disease (and thereby return false negative results). An

\textsuperscript{19} An older term for the test against which other tests are judged is the ‘gold standard’, but the term reference standard is currently in vogue as it better reflects the view that no test is perfect (Knottnerus and Muris 2009: 50).
index test that is both sensitive and specific will return neither false positive or false negative results, will therefore be accurate. An index test might be inaccurate because it is not sensitive, not specific, or neither sensitive nor specific.

Those medics who argue that measurements of cardiac dysfunction need to be made for a diagnosis of heart failure to be appropriate cite study after study that shows that the symptoms and signs associated with heart failure are neither sensitive nor specific for the disease, and use this finding to argue that the diagnosis of heart failure should only be made if cardiac dysfunction is measured and confirmed (Fonseca 2006; ESC 2012: 1792, 1794-1795; ESC 2016). This may at first appear to support the view that cardiac dysfunction needs to be measured to secure a diagnosis of heart failure, but a closer look at these papers reveals that the arguments deployed to support this view are highly problematic.

Many of the papers cited in this medical literature on heart failure use reference standards that are not trusted by the researchers that cite them. Take for instance papers by Remes et al (1991) and Davie et al (1997). These papers are widely cited to provide evidence that the clinical diagnosis of heart failure is inaccurate (Vasan and Levy 2000; Fonseca 2006; ESC 2012; ESC 2016). According to a recent review of the epidemiology of heart failure, Remes et al 1991 was the “first paper to address the limited value of symptoms and signs in diagnosing heart failure” (Mosterd and Hoes 2007: 1146; as is apparent from the next chapter this claim is not true but it does show the high regard in which this paper is held). Remes et al (1991) require that radiographic evidence of heart failure is present, and Davie et al (1997) require that reduced systolic function be present, for diagnoses to be made using their respective reference standards. Both studies find that a purely clinical diagnosis is inaccurate. However, the ESC (2012: 1797)
state that a “chest X-ray is of limited use in the diagnostic work up of patients with suspected heart failure”. The review cited by the ESC (2012, 2016) also argues that “radiographic variables, although quite specific, have poor sensitivity” (Fonseca 2006: 100). If the chest X-ray is inaccurate, then the finding that purely clinical diagnoses do not correlate with diagnoses made using a reference standard that requires radiographic abnormalities should not be taken as evidence that a purely clinical diagnosis is inaccurate. If the purely clinical diagnosis is actually accurate, then it should fail to give the same results as an inaccurate reference standard. Furthermore, if chest X-ray is insensitive for heart failure, then there will be many patients with heart failure that the chest X-ray fails to detect. If a purely clinical diagnosis was actually accurate, then it should appear to return lots of false positive results, and this is what Remes et al (1991) report. The same can be said for Davie et al (1997). As described above, the ESC at present believe that systolic dysfunction need not be present for a diagnosis of heart failure to be made, because up to half of patients with heart failure are thought to have diastolic dysfunction. Again, if a purely clinical diagnosis were accurate, then it should be expected to return positive results in patients that did not have heart failure with systolic dysfunction, and this is what Davie et al (1997) describe. As the reference standards used are not trusted, their results are perfectly consistent with a purely clinical diagnosis being either accurate or inaccurate, and therefore uninformative. It is incoherent of researchers to use studies like this as evidence for their views about the inaccuracy of a purely clinical diagnosis.

A purely clinical diagnosis, made using symptoms and signs alone, is assessed as an index test against a reference standard in these papers, and the purely clinical diagnosis is found to be inaccurate because this does not tally with a
diagnosis made using the reference standard. However, as the reference standard used is not trusted by the researchers citing these papers, then the conclusion that a purely clinical diagnosis is inaccurate should not be trusted either. Indeed, finding that a purely clinical diagnosis does not tally with an untrustworthy reference standard is just what would be expected if a purely clinical diagnosis was accurate. It makes little sense that such papers are routinely cited in support of the view a purely clinical diagnosis is inaccurate.

Researchers also cite a large number of papers that measure the diagnostic accuracy of a purely clinical diagnosis using a reference standard they do trust. The ESC cite a large number of studies that evaluate the accuracy of a purely clinical diagnosis against a diagnosis made using the criteria that they themselves recommend (i.e. the presence of symptoms signs and echocardiographic evidence of cardiac dysfunction). This practice is also problematic, because it produces circular arguments, if it produces arguments in favour of the ESC's view at all.

Recall that the reason the echocardiographic measurement of cardiac dysfunction was deemed to be important in the first place was because a purely clinical diagnosis was deemed to be inaccurate (Vasan and Levy 2000: 2118).

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20 According to a review by Candida Fonseca (2006; cited by the ESC 2012; 2016), the use of different reference standards in different studies means that "it is difficult for the general reader to draw any conclusions from those studies, except that none of the clinical features is consistently useful and that symptoms and signs have limited value in diagnosing heart failure when used as the only tool" (Fonseca 2006: 98). It is unclear why this conclusion should follow inevitably from these studies. From close inspection of the medical literature on the diagnosis of heart failure, it seems that doctors searching for answers about the accuracy of a purely clinical diagnosis have little choice but to look at papers that use a reference standard they do not trust. Reference standards might be chosen from a combination of certain symptoms, signs, echocardiographic measurements, radiographic observations, and measurements of intra-cardiac pressure. If the medical literature on the diagnosis of heart failure cited by the ESC is to be believed, none of these indicators of heart failure correlate strongly with any of the others (Stevenson and Perloff 1989; Chakko et al 1991; Di Bari et al 2004). Given this, if a doctor who accepts the ESC's view looks at a paper that uses any of these other measurements as a reference standard, this paper is bound to produce results that are considered untrustworthy.
The ESC cites papers which measure the accuracy of symptoms and signs in the
diagnosis of heart failure as empirical evidence to support this claim, giving the
appearance that an empirical argument is being presented. As we have just seen,
a large number of the papers cited use the ESC’s criteria as a standard against
which the diagnostic performance of symptoms and signs are judged. Therefore,
the measurement of cardiac dysfunction is required because symptoms and
signs are not accurate, and symptoms and signs are not accurate because the
measurement of cardiac dysfunction is required. Although this argument for the
need to measure cardiac dysfunction has the appearance of an empirical argument, it is not one. It is a circular argument. If researchers are using these
studies as empirical evidence that the ESC’s diagnostic criteria are the best and
most accurate available, then they have assumed what they claim to have shown21.

Of course, the ESC (and others) might not be using studies that assess the
accuracy of a purely clinical diagnosis against the ESC’s diagnostic criteria to
argue that the ESC’s diagnostic criteria are the best available way of making the
diagnosis of heart failure. They may simply be using these papers to argue that,
given the accuracy of the ESC’s diagnostic criteria, a purely clinical diagnosis is
not accurate22. If this is the case, they have not presented any argument in favour

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21 Circular arguments are actually quite commonly deployed in medical literature on diagnosis. Instances
of circular arguments can be found in medical literature on rickets (Slovis and Chapman 2008),
fibromyalgia (Yunus et al 1981; Wolfe et al 1990; see Cohen and Quinter 1993 for comments), thyroid
disease (Harvey 1971; see Götzsche 2007: 80-81 for comments), Cushing’s disease (Elamin et al 2008),
and abusive head trauma in infants (Ewing Cobbs et al 1998; Feldman et al 2001; Wells et al 2002;
Vinchon et al 2005; Matschke et al 2009; see Vinchon 2010: 637-638; Moran et al 2012: 274; Höberg et
al 2016 for comments). Indeed, according to some commentators, “[t]he primary defect is that virtually
all the SBS/AHT literature is circular” (Moran et al 2012: 274). Circular arguments are quite common in
the medical literature on diagnostics.

22 In my view this is not a reasonable reading of the ESC’s guidelines. If the ESC were simply asserting
that their diagnostic criteria were accurate, and assessing a purely clinical diagnosis given this
assumption, then there would be no reason to cite research that does not use their criteria as a
reference standard. That they do cite such research shows that they are not taking the accuracy of their
criteria for granted. Rather they are trying to support the view that their criteria are accurate by
of their own criteria being the most accurate. They have simply asserted that the diagnosis of heart failure is made more accurate if cardiac dysfunction is measured. As I have shown, this claim is controversial because debates about how to measure cardiac function are ongoing.

In summary, medical debate about how to diagnose heart failure is focused on establishing which diagnostic criteria are the most accurate of all. Arguments that try to establish which set of diagnostic practices are the most accurate of all have proved problematic. The medical literature on heart failure is replete with examples of incoherent arguments, circular arguments, and bare assertions. It is difficult to find anything in this literature to oppose the view that measurements of cardiac dysfunction were incorporated into the diagnostic criteria for heart failure because it became “fashionable” to do so (see Yturralde and Gaasch 2005: 314 above)\textsuperscript{23}.

Given that medical researchers have been trying to discover the best way to diagnose disease by trying to find the most accurate diagnostic practices, it is unsurprising that they have produced unsatisfactory arguments. To measure the accuracy of a diagnostic practice is to treat this practice as an index test. The accuracy of an index test is measured against a reference standard. The test or diagnostic practice deemed most accurate of all is the reference standard, and

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\textsuperscript{23} I should highlight that it is not just advocates of the requirement for measuring cardiac dysfunction that produce problematic arguments. Advocates of the “traditional” purely clinical diagnosis also produce problematic arguments. For one thing, it is not clear why this approach is good just because it is traditional. Advocates of the purely clinical diagnosis (see Jolobe 2006) have also cited research that measures the accuracy of the purely clinical diagnosis, as well as of diagnoses that require radiographic and echocardiographic evidence of cardiac dysfunction, against a reference standard that they designed (Di Bari 2004). This study found large discrepancies between diagnoses made in these different ways, and that the purely clinical diagnosis was the most accurate. This finding only begs the question of why this especially designed reference standard is trusted more than any of the ways of making a diagnosis being assessed.
not any of the index tests. Therefore, the test deemed most accurate of all can never have its accuracy measured, and the status of most accurate of all cannot be acquired by measuring diagnostic accuracy. Attempting to determine which test is most accurate of all by measuring and comparing the diagnostic accuracy of various tests is incoherent, and will inevitably lead to the production of unsatisfactory arguments. Trying to measure the accuracy of the reference standard against any other set of diagnostic practices simply begs the question of how it is known that these other sets of practices are more accurate than the reference standard, setting up an infinite regress. Assessing the accuracy of the reference standard against one of the index tests means that the reference standard is being evaluated against a set of diagnostic practices that is trusted less than the reference standard, producing incoherent arguments such as ones identified above. Trying to measure the accuracy of the reference standard could also lead to assessing the reference standard against itself, which would produce a circular argument such as the one identified earlier.

A little philosophical reflection reveals that there is an important epistemic problem that manifests when attempts are made to investigate how to diagnose disease. If it is not known how to identify the group of patients with a disease, then it will not be possible to simply inspect a population of patients to discover the characteristics that can be used to distinguish diseased from non-diseased patients. For, in order to discover which characteristics can be used to identify the group of patients with a disease, it is first necessary to sort patients into diseased and non-diseased groups. This cannot be done unless the characteristics that can be used to identify the group of patients with a disease are already known, and this is not known as this is precisely what is under investigation.
This is a medical manifestation of a very ancient epistemic problem, identified and discussed by Plato. It is most clearly articulated in Plato’s *Meno*, and is discussed today as “Meno’s paradox”, or perhaps more properly as “Meno’s challenge” (Fine 2014). The main subject of this dialogue is the nature of virtue and whether virtue can be taught, but at one point during the discussion Meno reaches a point of despair, and argues that it is not possible to investigate the nature of anything at all: “And how will you inquire into this, Socrates, when you don’t know at all what it is? For what sort of thing, from among those you don’t know, will you put forward as the thing you are inquiring into? And even if you really encounter it, how will you know if this is the thing that you did not know?” (Fine 2014). Medics in the present day face this problem when trying to discover the characteristics that can be used to identify patients with a disease. As it is not known how to identify these patients, researchers cannot just inspect these patients and compare them to not diseased patients to see what these characteristics are. More recently, Hasok Chang has identified this problem (although he does not refer to it as such) in the philosophy of measurement (Chang 2004: 59, 89-90; Chang 2012b: 113). He calls it the “problem of nomic measurement”, and he formulates it as follows: “(i) We want to measure quantity X; (ii) If quantity X is not directly observable, we infer it from another quantity Y, which is directly observable. (iii) For this inference we need a law that expresses X as a function of Y. (iv) but the form of the function cannot be discovered or tested empirically, because that would involve knowing the values of both X and Y, but X is the unknown variable that we are trying to measure” (Chang 2012b: 113). Chang also comments that “[T]he precise formulation of the problem made me realize two things: first it must be a general problem pertaining to nearly every attempt to justify a method of measurement; second, there was no apparent
solution to it!” (Chang 2012b: 113). If we take $X$ to be the diseased group of patients that we don’t know how to detect, and $Y$ to be the observations by which patients with the disease can be identified, this formulation of the problem can be applied to the measurement/diagnosis of disease. Chang also points out that if we attempt to justify the belief that the function which tells us the relation between $X$ and $Y$ has the particular form “$f$” empirically, then we cannot help but argue in a circle (Chang 2004: 89). “$f$ cannot be determined without knowing $X$ values, but $X$ cannot be determined without knowing $f$” (2004: 89). The circular arguments seen in the medical literature on the diagnosis of heart failure and other disease are thus explained. Even though this problem is a very ancient one, medics in the present day have not come to terms with it, and persist in trying to discover how to distinguish different sorts of patient by a form of naïve empiricism. This thesis suggests an alternative epistemology, one which gives up the search for universal truths about the characteristics that can be used to distinguish different sorts of patient.

Researchers’ today try to present their views about how best to diagnose heart failure as the result of simple empirical correlations between patients with heart failure and the results of diagnostic testing. Despite their efforts, the production of knowledge of how to diagnose disease is not as simple as it may first appear\(^\text{24}\). It is not possible to learn how to identify patients with heart failure by making such

\(^{24}\text{I have concentrated on the medical literature on the diagnosis of heart failure in this chapter, but it is not hard to find examples of a similar attitude that is present in many areas of medical practice. For instance, it is quite commonplace to find medics arguing that very often there are no perfect tests for the presence of a disease, and that the reference or gold standard is only the test that is observed to be the most accurate (Versi 1992; Cardoso et al 2014). “Thus, in practice, there is no perfect gold standard. Instead we have a method with the greatest sensitivity and the highest specificity” (Cardoso et al 2014: 29). Nevertheless, it is not possible to determine which test is the most accurate by measuring diagnostic accuracy, because the test deemed to be the most accurate will not be amongst the tests evaluated (i.e. amongst the index tests), but rather will be the reference standard.}
simple empirical correlations. However researchers have actually learned to distinguish diseased and non-diseased patients, it is not by making simple empirical correlations between diseased patients and their observed characteristics. Attempts to present their views on how to diagnose heart failure as the outcome of such empirical correlations has resulted in researchers, like those in the ESC, deploying unconvincing arguments in support of their views.

Conclusion
Some historians claim that medical history can be used to inform medical decision making, because medical knowledge is historically contingent. Many philosophers of medicine tend to disagree with this view, and hold that medical knowledge about heart disease is particularly unproblematic and straightforward to establish. I have argued that these philosophers have been too quick to assume this, because their views are not in agreement with the views held by medics. Furthermore, medics themselves disagree about what heart failure is, and how to diagnose it. The attempts made by medical researchers to discover universal, timeless, ahistorical truths about which characteristics can be used to distinguish patients with and without heart failure have failed. I have argued that this is because trying to learn how to identify diseased patients by correlating the results of diagnostic observations with the disease status of the patient is impossible, as it is the disease status of the patient that is under investigation. If medical knowledge was historically contingent, then the difficulties encountered by researchers trying to discover timeless truths about how to diagnose heart failure would be unsurprising. This provides a reason to suspect that the historians discussed at the beginning of the chapter are correct to argue that medical knowledge is historically contingent. This question will be investigated more fully in the next chapter.
Chapter 2 – A longue durée history of heart failure

Introduction
In the last chapter I raised the question of whether medical knowledge, and specifically that of heart failure, is historically contingent. Might it be the case that knowledge of how heart failure can be diagnosed is not fully determined and inevitable given the empirical observations that doctors have made? In this chapter I present a history of the development of medical knowledge of heart failure over the last two centuries, and use this history to argue that medical knowledge of heart failure is historically contingent.

Even though I argue for the historical contingency of medical knowledge, this does not imply that doctors have just invented their knowledge of heart failure. Although empirical observation has not fully determined what doctor’s knowledge of heart failure is, it still has played an important role in constraining what doctors can take knowledge of heart failure to be. This history is not consistent with an extreme form of relativism, where doctor’s views about heart failure are unconstrained by empirical observation, and where doctors are free to make heart failure into anything they want it to be. An epistemology that can account for how knowledge of heart failure has developed needs to show how knowledge can at once be invented and discovered.

As discussed in the introduction, this history has been produced by starting in the present day and working backwards through time, linking different ways of understanding sick people together. However, for ease of narration I present this history chronologically, running from the early nineteenth century to the present day. This history is organized into a series of eight stages, each of which reflects
a different way of understanding sick people and heart disease. A change of stage represents a change to how heart disease or failure is diagnosed, and/or to the group of patients who would be diagnosed with the disease, and/or a change to the pathophysiology used to explain patient’s symptoms. Sometimes, the transition between stages is recognised by the historical actors themselves. So for instance, James Mackenzie (discussed in stage 2) explicitly rejected the way of understanding heart disease that emerged in the early nineteenth century (discussed in stage 1). Tinsley Randolph Harrison (discussed in stage 3) explicitly rejected aspects of Mackenzie’s way of understanding heart failure. However, this is not always the case, and sometimes I draw boundaries where historical actors did not. For instance, I draw attention to the contrast between the purely clinical diagnosis of heart failure made by Soufer and colleagues (1985) (discussed in stage 7) and the requirement of echocardiographic evidence of cardiac dysfunction made by the ESC (discussed in stage 8). Ultimately, I draw boundaries between stages that I think are helpful to understand the development of knowledge of heart failure. Often I do this because my historical actors recognise these boundaries, but this is not always so. I explain my decisions about why I have drawn the boundaries I have, and why I have connected the stages I have together, in footnotes to the main text of this chapter.

By focusing on the evidence and arguments presented by historical actors in support of their decisions to modify how heart failure was understood, I argue (often against the views of these historical actors) that their experiences did not

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25 I should highlight that I am not arguing that each successive stage entirely replaces the one before it. Indeed, I draw attention to how stages 6 and 7 coexisted, and how stages 7 and 8 currently coexist, because I cannot explain the changes to how heart failure was understood in these periods without discussing two parallel traditions for understanding heart failure, each of which feeds off the other. Nevertheless, in many periods my historical actors indicate that a particular way of understanding heart failure was dominant, and this is reflected in how I present this history.
force them to modify their understanding in the way that they did. A recurring motif in this history is that the transition between stages is precipitated by observations which conflict with the expectations of historical actors. The exception to this is the transition between stage 4 and stage 5, for which there was no discernible conflict with experience. Other than this, change precipitated by conflict is the rule. I describe how the transition from one stage to another involves the rejection (and very often the explicit and self-conscious rejection) of aspects of how heart failure was understood in the preceding stage, whilst at the same time conserving aspects of how heart failure was understood in the preceding stage. I argue that researchers always had options about what is rejected and what is conserved during the transition from one stage to another, even if they did not recognise that this was the case. I argue that the transition from one stage to another is historically, socially and culturally contingent.

Stage 1 - The nineteenth century
I begin in the Paris hospital system of early nineteenth century France. The I begin my longue durée by looking at the Paris hospital system of the early nineteenth century because later historical actors in this history reacted to the work done in this period. Mackenzie (see stage 2 of this history) was highly critical of the conclusions reached by doctors shortly after the development of the stethoscope (see chapter 5), who worked in hospitals and sought to learn how to detect morbid anatomy using the ante-mortem physical examination. Jean-Nicholas Corvisart (a prominent Parisian doctor in this period) was a champion of this approach to medical research, and his student Laennec invented the stethoscope (see chapter 4). Even though Mackenzie did not identify these actors by name, he does criticize the research programme that they exemplify. James Hope (an important early nineteenth century English doctor in my history) went to Paris specifically to learn how to use the stethoscope, before returning to Britain to champion its use in the diagnosis of heart disease (see chapter 4). Hope was an important actor in the development of the back-pressure theory of heart disease, and of the view that patients with heart murmurs should have their activities restricted. Mackenzie objected strongly to both of these doctrines. So although Mackenzie did not mention any of these actors by name, I argue it is reasonable to claim that he was reacting to the results of their work. Furthermore, T.R. Harrison (1935: 5, 9) did cite Hope as an important figure in the development of the back-pressure theory (see stage 3 of this history). I could continue my process of chasing citations back in time, and look at the work of eighteenth century researchers referenced by Parisian nineteenth century researchers. These Parisian researchers do draw on the work of other earlier researchers. Hope cites Sézéac, and Corvisart refers to Morgani (Fleming 1997: 7; Corvisart 1813: iii). Both Sézéac and Morgani associate breathlessness, orthopnoea, and oedema with lesions of the heart (Fleming 1997: 7, 10). I do not continue searching for further references because these researchers argue that they make the correlation between lesions of the heart and the syndrome of breathlessness and fluid accumulation for themselves. This makes the early nineteenth century a good place to stop my back-tracking, and start my historical account of the development of knowledge of heart failure.

26 I begin my longue durée by looking at the Paris hospital system of the early nineteenth century because later historical actors in this history reacted to the work done in this period. Mackenzie (see stage 2 of this history) was highly critical of the conclusions reached by doctors shortly after the development of the stethoscope (see chapter 5), who worked in hospitals and sought to learn how to detect morbid anatomy using the ante-mortem physical examination. Jean-Nicholas Corvisart (a prominent Parisian doctor in this period) was a champion of this approach to medical research, and his student Laennec invented the stethoscope (see chapter 4). Even though Mackenzie did not identify these actors by name, he does criticize the research programme that they exemplify. James Hope (an important early nineteenth century English doctor in my history) went to Paris specifically to learn how to use the stethoscope, before returning to Britain to champion its use in the diagnosis of heart disease (see chapter 4). Hope was an important actor in the development of the back-pressure theory of heart disease, and of the view that patients with heart murmurs should have their activities restricted. Mackenzie objected strongly to both of these doctrines. So although Mackenzie did not mention any of these actors by name, I argue it is reasonable to claim that he was reacting to the results of their work. Furthermore, T.R. Harrison (1935: 5, 9) did cite Hope as an important figure in the development of the back-pressure theory (see stage 3 of this history). I could continue my process of chasing citations back in time, and look at the work of eighteenth century researchers referenced by Parisian nineteenth century researchers. These Parisian researchers do draw on the work of other earlier researchers. Hope cites Sézéac, and Corvisart refers to Morgani (Fleming 1997: 7; Corvisart 1813: iii). Both Sézéac and Morgani associate breathlessness, orthopnoea, and oedema with lesions of the heart (Fleming 1997: 7, 10). I do not continue searching for further references because these researchers argue that they make the correlation between lesions of the heart and the syndrome of breathlessness and fluid accumulation for themselves. This makes the early nineteenth century a good place to stop my back-tracking, and start my historical account of the development of knowledge of heart failure.
profound changes to medical research and practice that took place in the Paris hospital system around the turn of the nineteenth century have been well documented (Foucault 1973; Jewson 1976; Lawrence 1985; Porter 1997: 306-320; Weiner and Sauter 2003; Jacyna 2006). Instead of focusing on detecting patterns in the symptoms reported by patients, physicians in the Paris hospital system focused on the detection of lesions to organs. According to Jean-Nicholas Corvisart (1755-1812) (Napoleon’s personal physician and a prominent advocate of this approach) “organic lesions……are far more prevalent than most physicians have yet thought” (1812: 15). Physicians in the Paris hospital system looked to the post-mortem examination as the best method of detecting lesions to organs, as this method allowed physicians to see for themselves whether or not their ante-mortem diagnoses had been correct. The post-mortem examination allowed physicians to find “in the dead body, the mistakes which their ignorance of anatomy had caused them to commit” (Corvisart 1812: 17).

These Parisian physicians also sought to learn how to use the results of a physical examination made ante-mortem (before the patient died) to diagnose the organic lesions that would be found post-mortem. To this end, new techniques of physical examination were developed and/or came into widespread use. Corvisart was a strong advocate of the value of the physical examination, and is credited with popularising Auenbrugger’s method of percussion (the use of tapping on the body wall to determine the size and contents of body cavities and organs) (Lawrence 1985: 5-6). Corvisart’s student, René Laennec, invented the stethoscope and the practice of mediate auscultation (Lawrence 1985: 5-6). The ability to use observation, palpation (feeling), auscultation (listening), and percussion to detect the organic lesions a patient had inside them were key
diagnostic skills for Corvisart and his colleagues. This passage from Corvisart’s *An Essay on the Organic Diseases and Lesions of the Heart and Great Vessels* (1812) captures the pride a physician from this period might take in diagnosing ante-mortem the lesions of the heart to be found post-mortem, rather than (for instance) being able to treat the patient:

“Although it were impossible to obtain more accurate information of this disease, the appearance of the patient, the state of respiration, and of the pulse, the thorax, when struck, giving no sound, the peculiar rushing noise of the region of the heart convinced me of the existence of dilatation of the heart and of the constriction of the aortic orifice, changes which I announced would be found on dissection” (Corvisart 1812: 97; see also 1813: 88).

Using the results of physical and post mortem examinations of patients, Corvisart made an empirical association between patients who struggled to breath (particularly during exertion or while recumbent) and the enlargement of the heart and lesions which obstruct the circulation (1812: 97, 121; 1813: 119-129, 178-179). Corvisart, in his description of the typical presentation of patients with “aneurisms” (enlargement) of the heart, describes a syndrome of progressively worsening breathlessness and fluid accumulation. In the early stages of the disease “the patient, in order to breath more easily, is obliged, from time to time, to stop when walking, particularly when going up stairs (Corvisart 1813: 119-120). As the disease progresses, fluid starts to accumulate in the patient’s body, and “[t]he feet and lower part of the legs swell on standing; but this tumefaction generally goes down at night” (Corvisart 1813: 122). The breathlessness continues to get worse, and the patient “cannot go up three of four steps at a time, without extreme shortness of breath obliging him to stop” (Corvisart 1813: 124). The patient has trouble breathing in certain postures, specifically “[h]e cannot breath in a horizontal position, but is obliged, in order to render his breathing somewhat less difficult, to sit, bending his body forward and supporting,
as it were, his chest upon his knees” (Corvisart 1813: 124). As the patient nears death, “[t]he sense of suffocation is every moment more threatening”, and the patient lacks the strength to hold himself in the position which makes breathing less uncomfortable (Corvisart 1813: 127). The face is oedematous (full of water under the skin), and the veins of the neck are swollen. The body can be “generally oedematous” (Corvisart 1813: 179), so much so that “[t]he skin is then flabby, and shakes on the slightest motion of the head or the gentlest touch” (Corvisart 1813: 126). “Death always terminates the distressing spectacle which the union of all these symptoms presents” (Corvisart 1813: 128).

Corvisart offered an explanation for how lesions that obstructed the circulation on the left side of the heart (which is just in front of the pulmonary circulation), could produce swelling of blood vessels and organs supplied by the systemic circulation (which is just behind the right side of the heart):

“This may easily be explained; the left cavities of the heart; not properly emptying themselves, the right could not become unloaded; the blood, therefore, from the venæ cavae accumulated; hence arouse the sanguinious distension of the liver, so frequent in diseases of the heart, as also the hypogastric venous plethora, and the intestinal haemorrhage” (Corvisart 1813: 180).

In the same way as an obstruction on a road can cause a traffic jam that extends for several miles behind the obstruction, Corvisart argued that an obstruction on the left side of the heart (such as that caused by narrowed blood vessels of valvular lesions) could cause blood to collect in the pulmonary circulation, and then in the right side of the heart, and then in the vena cava, and then in the blood vessels of bodily organs, like the liver and gut (Corvisart 1812: 95-97; 1813: 60, 83). Corvisart also reported that the enlarged pulmonary vessels and heart could lead to breathlessness because “the engorged and compressed lungs can admit
but a small quantity of air” (Corvisart 1812: 121; 1813: 123).

The work of English physician James Hope both corroborated and extended Corvisart’s conclusions. Hope had travelled to Paris in the late 1820s to learn the art of auscultation (Bluth 1970; Bynum 1994: 49). Hope's work was influential amongst the London medical community in the 1830s, and he worked hard to popularise the practice of auscultation in England. Hope thought that auscultation was a particularly important skill for a physician to develop, because it allowed the ante-mortem detection of anatomical lesions as they produced abnormal heart sounds or 'murmurs' (Bluth 1970). However, Hope still used the post-mortem examination to determine definitively whether heart disease was present or not (Bluth 1970). In the same way as Corvisart, Hope correlated the results of ante-mortem clinical observations with the results of the post-mortem examination, and gave this as his general description of the presentation of a patient with valvular disease of the heart:

“Briefly to recapitulate these symptoms – they are, cough, copious watery expectoration, orthopnoea [difficulty breathing when lying down], frightful dreams and starting from sleep, œdema [fluid accumulation] of the lungs, pulmonary apoplexy, passive hæmoptysis, (i.e. sputa stained with dark or grumous blood,) turgessence of the jugular veins [in the neck], lividity of the face, anasarca [generalised oedema], injection of almost all the mucous membranes, passive haemorrhages, especially of the mucous membranes, engorgement of the liver, spleen &c. and congestion of the brain with symptoms of oppression sometimes amounting to apoplexy” (Hope 1833: 330).

Hope also used the results of physical and post-mortem examinations in the same way as Corvisart (1813: 60-61, 83-86) to infer how hearts became enlarged and patients became breathless. He reached very similar conclusions to Corvisart, in that obstructions to the circulation caused sequential congestions and enlargement of chambers behind the obstruction, ultimately affecting both
the pulmonary and systemic circulation (Hope 1839: 198; 1833: 195-200). However, Hope did develop this theory. He proposed that the increased pressure behind an obstruction to the left side of the heart would not only cause the lung to become congested, but would force fluid out from the vessels into the air spaces in the lung (1839: 198). According to Hope, similar events occurred following a build-up of pressure behind the right side of the heart, which led to the transudation of fluid from the systemic circulation resulting in distension of the jugular, enlargement of the liver and the accumulation of fluid in the abdominal cavity (known as ascites) (Hope 1833: 215, 269). Hope also proposed that an incompetent valve which allowed the flow of blood in a retrograde direction would cause the chamber behind it to become distended and thus constituted an obstruction to the circulation (Hope 1839: 198), setting off the chain of events that would affect the whole circulation behind the blockage. Although Hope did not refer to it as such, this theory of heart disease came to be known as the back-pressure theory (Mackenzie 1913: 6-7). Consequently, Hope recommended that patients with valvular disease should have their exercise restricted, to prevent this chain of events getting started in the first place (Hope 1833: 326).

Stage 2 - James Mackenzie (1853-1925)
Hope’s efforts to popularise auscultation were successful, and it is reported that by the end of the nineteenth century auscultation was widespread in general practice in Britain (Mackenzie 1913: 7). However, this skill was being applied to a different group of patients to that which it had been by Hope. Physicians in general practice examined patients from general practice, not patients near death in hospitals as Corvisart and Hope had done. These physicians therefore encountered patients with murmurs at physical examination (and therefore judged to have incompetent heart valves or other lesions of the heart), but who
were not breathless or showing any other signs or symptoms. Many such patients were classified as having heart disease (Mackenzie 1913: 7, 18-19). This act of classification had consequences, such as patients being advised not to have children, patients being prevented from enlisting in military service, and patients being denied medical insurance (Hay 1930: 1036). The reverse situation was also reported, where people who were very ill and close to death were treated as if they were not because no lesion of their heart could be identified at physical examination (Mackenzie 1913: 18). Unlike Hope and Corvisart, these physicians in general practice were using the detection of lesions of the heart to try to prognosticate in the *early* stages of disease.

Around the turn of the twentieth century a number of physicians objected to these practices in the strongest terms. Some thought their rejection of these nineteenth century practices was so complete that they considered their new diagnostic practices as part of a “new cardiology” (Lawrence 1985: 1, 16). A Scottish physician, James Mackenzie (1853-1925), was very influential amongst this group of physicians. Like Hope, Mackenzie had graduated from the University of Edinburgh, and in 1879 he went into general practice in Burnley, an industrial town a few miles to the North of Manchester, England (Mair 1973: 34-39). He spent the next twenty-eight years in general practice in Burnley, where he did most of his original research. Using the clinical polygraph, an instrument of his own invention that could record tracings of several pulses at once, he studies the arrhythmias with which his own patients in general practice presented. In 1902, Mackenzie published his first book (*The Study of the Pulse*), in which he

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27 I connect Mackenzie with Harrison because Harrison explicitly cites Mackenzie’s views as the dominant views in Anglo-American medicine of the heart in the 1930s, and the research Harrison presented in *Failure of the Circulation* (1935) is framed as a test of Mackenzie’s ideas.
presented the results of his research into various forms of arrhythmia. This book was well received, and in 1907 Mackenzie moved to London to work as a physician with a special interest in diseases of the heart (Mair 1973: 236-237).

In general practice, Mackenzie had encountered significant problems with the traditional ways of diagnosing heart disease. This passage from his article *The Recruit's Heart* (1915) is revealing of the problems he encountered in practice and his research program:

“When I entered practice in 1879, I did so with the airy confidence of the young man fresh from medical school imagining that I was well equipped for my work. In a very short time I realised I was ignorant of the very essentials of my work. In heart work, like other young men, I flattered myself on my cleverness with the stethoscope in detecting and naming all sorts of murmurs, but when it came to telling their significance, I was completely at fault. I turned to my books for instruction, to find that this very essential knowledge had been ignored. Realising my ignorance of the meaning of the simplest symptoms, I set myself the task of finding them out.

To do this, I took note of my patients who presented any abnormal sign. I carefully differentiated each phenomenon from allied conditions. I noted all associated phenomena. I kept a large number of people under observation, watching them for a great many years, noting how they bore themselves through the strain and stress of life, in their labours and in their illnesses. As years went on, I found it necessary to seek other methods of investigation, and had to devise them. In this work I followed a large number of individuals for ten, fifteen, twenty or even thirty years to find out the significance of some sign” (Mackenzie 1915: 807).

Mackenzie found that many of the signs traditionally held to be indicative of a poor prognosis for a patient in fact did not carry a poor prognosis at all. For example, he found that many patients with heart murmurs and valvular lesions could lead very vigorous lives, and engage in strenuous occupations, without complications of any sort (Mackenzie 1913: 327). Mackenzie therefore concluded that the traditional view that the presence of a murmur meant the heart was diseased, and that a person with a diseased heart would soon die if they did not restrict their activity, was incorrect. Mackenzie also concluded that the ‘back-
pressure theory’ of heart disease must also be incorrect (Mackenzie 1913: 7).

To replace the back-pressure theory, Mackenzie proposed a different theory of heart disease. He employed what he saw as the common sense notion that the job of the heart was to ‘maintain an efficient circulation when called upon to meet the efforts necessary to the daily life of the individual’ (Mackenzie 1913: 5). If the heart could pump a sufficient volume of blood forwards to meet the metabolic requirements of the patient then the heart was not failing, regardless of whether a murmur was present or not (Mackenzie 1913: 10-11). Mackenzie also developed a concept of the ‘reserve force’ of the heart (Mackenzie 1913: 11), which was a heart’s ability to increase its output as the patient exercised. He argued that a failing heart was one which had a diminished reserve force, and so had a reduced capacity to increase the amount of blood pumped forwards in the circulation in response to effort. He called this state of reduced reserve force of the heart “heart failure” (Mackenzie 1913: 11). This theory became known as the “forward failure theory” (Bedford 1939: 1303).

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28 As I will discuss in chapter 4, Mackenzie operationalised this concept of reserve force by assessing the patient’s response to effort.

29 The term ‘heart failure’ first came into widespread usage in this period (Fleming 1997: 145). Mackenzie did not coin the phrase ‘heart failure’, but he did champion it aggressively. “The term “heart failure” will be employed in preference to heart disease, inasmuch as the symptoms are sometimes evoked in cases where there is no demonstrable cardiac lesion. The failure of heart power may be, however, the undoubted cause of their appearance, this failure and its accompanying symptoms frequently being evanescent” (Mackenzie 1895: 16). Physicians did comment on the term’s increasing use in the early twentieth century (Fleming 1997: 145). This change in thinking about heart disease is reflected in changes to how many textbooks were laid out. As was noted for Hope and Corvisart in the last chapter, textbooks on heart disease were laid out according to the anatomical site of the lesion. Lawrence makes the same observation for or texts on heart disease (Lawrence 1985: 5-6). Mackenzie’s text book Diseases of the heart, by contrast, begins with a discussion of what heart failure is, continues to a discussion of the physiology of the exhaustion of heart muscle, and goes on to discuss the symptoms indicative of heart failure (Mackenzie 1908). Mackenzie did have a chapter on valvular lesions, which he did think could cause heart failure sometimes, but warned that “[v]alvular defects will not be studied as a specific affection to be considered in themselves, but rather as a source of embarrassment to the heart muscle in its work” (Mackenzie 1908: 3; see Lawrence 1985: 20-21).
The notion of heart failure was very important to Mackenzie. As he did not think that patients who either were or were going to become sick could be diagnosed by identifying organic lesions of their hearts, he needed some other means of identifying these patients. By the late nineteenth century, the syndrome of breathlessness and fluid accumulation associated with heart disease in the early nineteenth century was being referred to as the syndrome of “heart failure”. He argued that these symptoms provided a better indication of how the heart was working, and could be used to detect patients who were or who were going to become ill. Instead of diagnosing organic heart disease, Mackenzie argued that doctors should focus on the symptoms that characterized the syndrome of heart failure (Mackenzie 1895: 16). Mackenzie was so confident that heart failure could be recognised from its characteristic symptoms that he argued that heart trouble could be diagnosed even when there was no detectable organic lesion of the heart:

“Even when we get a series of well-defined symptoms, such as those shown by orthopnoea, dyspnoea, dropsy, enlargement of the liver, or those shown by attacks of angina pectoris, we shall find pathological changes in the heart so varied that it is impossible to attribute the symptoms with assurance to any given pathological condition. This becomes all the more impressive when we find fatal heart failure associated with no perceptible lesions of the heart sufficient to account for the heart failure. No doubt a pathological process may play a considerable part in impairing the functional efficiency of the heart, but the fact that the heart may fail to do its work when there is an abundance of seemingly healthy muscle present forces us to the conclusion that the heart failure is really the outcome of the impairment of the functions of the heart muscle itself, while the seemingly healthy muscle is the seat of changes too subtle for our present methods to detect” (Mackenzie 1913: 7-8).

Even though Mackenzie rejected so much of the nineteenth century way of understanding heart disease, he still had some things in common with these earlier views. The association of heart trouble with the syndrome of breathlessness and fluid accumulation had been made in the early nineteenth
century, and Mackenzie kept this association. So although Mackenzie rejected some aspects of the nineteenth century approach to heart disease, such as their focus on the diagnosis of organic lesions, and the back-pressure explanation for the syndrome of breathlessness and fluid retention that he called heart failure, he still retained other aspects of it.

It should also be noted that Mackenzie was not “forced” to reject the detection of morbid anatomy as a reference standard by the evidence he produced. All he discovered was that lots of patients with breathlessness and fluid retention did not have certain pathological changes to their hearts, and vice versa. He could have interpreted this to mean that patients with breathlessness and fluid retention did not always have heart disease (if heart disease is defined as the presence of morbid anatomy). However, as discussed above, this approach to heart disease did not permit accurate prognostication, and prognostication was central to the practice of a physician as far as Mackenzie was concerned. Mackenzie therefore made the historically contingent choice to change how heart disease was understood, and to champion the notion of ‘heart failure’.

Mackenzie’s work became very influential in British medical circles (Lawrence 1985; Howell 1985), and served as an inspiration for clinicians who went on to form the new speciality of cardiology around new diagnostic tools like the electrocardiograph. In 1930, after Mackenzie’s death, John Hay (Mackenzie’s friend and professor of medicine at the University of Liverpool) gave an address summing up Mackenzie’s achievements:

“He blew the old theory of “back-pressure” into thin air, though it is true that one still hears of it from the mouths of those blind to the facts and deaf to the truth. But what is of still greater importance, he taught the real significance of cardiac failure and how to recognise its earliest
manifestations, for it is cardiac failure that the doctor fears and must foresee” (Hay 1930: 1036).

Mackenzie’s views even became dominant in America, as is apparent from the work of American clinician and physiologist Tinsley Randolph Harrison (1935). “A decade after his death his concepts still exert a dominant influence upon the current ideas of British and American cardiologists” (Harrison 1935: 8). Both physicians who were staunch supporters of Mackenzie’s work (Hay 1930) and physicians who came to disagree with him (Bedford 1939) were of the opinion that Mackenzie’s views and practices about heart disease became dominant in the first third of the twentieth century. In British and American cardiology, Mackenzie’s work came to represent the received view.

Stage 3 - Tinsley Randolph Harrison (1900-1978)
In the 1930s Mackenzie’s theories about heart failure were challenged by the American physician Tinsley Randolph Harrison. Throughout his career, Harrison worked as a physician, a medical investigator and pathophysiologist, and as an educator of medical students (Eddleman 1989:169). It was in his capacity as a pathophysiologist that he wrote “one of the most classic books on the subject of heart failure”, Failure of the Circulation, which was published in 1935 (Eddleman 1989: 169). In this book he argued against Mackenzie’s theory of heart failure, which he calls the “forward failure hypothesis” (Harrison 1935: 50).

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30 Harrison’s work again informs the work of later actors in my history, which is why I include him as an actor in this history. Harrison informs Merrill’s work (see stage 4 below), who frames his own (1946) paper as a modification of Harrison’s views in the light of unexpected observations. Harrison is also the original editor and namesake of Harrison’s Principles of Internal Medicine, in which Harrison and Braunwald (see stage 5 below) have a disagreement over how to approach the diagnosis of heart failure.
Harrison referred to Mackenzie’s views as an “hypothesis” because in his view Mackenzie had assumed that patients in heart failure did not maintain an adequate circulation; or as Harrison put it that the “cardiac output” was low in patients with heart failure (Harrison 1935: 51). Harrison reviewed a substantial body of medical literature (including his own research) which tested this hypothesis (1935: 52-69). He found that patient with heart failure did not necessarily have a reduced cardiac output in comparison to patients without heart failure. He even found that patients with heart failure could have raised cardiac outputs. Harrison interpreted these findings to mean that Mackenzie’s forward failure hypothesis was incorrect:

“It seems logical then for us to conclude that persons with congestive heart failure may and often do have diminished circulatory minute volume, but that this is not the essential cause of their symptoms. The ‘forward failure’ theory is not acceptable. In the following chapter an alternative hypothesis will be discussed” (Harrison 1935: 69).

Again, this was not the only “logical” conclusion which Harrison could draw from his data. To see this, it is important to note that Harrison is using the same method of diagnosing heart failure as Mackenzie. Both these physicians diagnosed heart failure by detecting the appropriate symptoms and signs. Harrison discovered that patients with the appropriate symptoms and signs did not necessarily have a reduced cardiac output, and interpreted this to mean that patients with heart failure did not necessarily have a reduced cardiac output. He could have interpreted this finding to mean that patients with the appropriate symptoms and signs did not necessarily have heart failure, if he had made the diagnosis only in patients with a reduced cardiac output.

Given the context in which Harrison was working, it is understandable that he did not interpret his findings in this way. For instance, the methods to measure
cardiac output available to Harrison were technically difficult to carry out. I will not describe them in detail here, but they involve the administration of gaseous and intravenous tracer substances, multiple blood samples being taken from patients in quick succession, the training of patients to use re-breathing apparatus, and numerous assumptions about how tracer substances recirculated (Harrison 1935: 52-69). Today, the measurement of cardiac output is much easier, as techniques to sample blood directly from the heart have developed since Harrison's time. In the 1930s, however, to use the measurement of cardiac output to diagnose heart failure would have been impractical at the time. Nevertheless, such considerations should show that Harrison's continued use of the appropriate symptoms and signs to diagnose heart failure was historically contingent, at least upon the technology available at the time.

Harrison (1935) therefore rejected Mackenzie's “forward failure hypothesis”, whilst at the same time preserving the use of the appropriate symptoms and signs as the reference standard for the diagnosis of heart failure. In addition to this, he proposed a new explanation for how heart failure could produce these symptoms. Harrison proposed an alternative hypothesis which he saw as very similar to Hope's (1839) back pressure theory (Harrison 1935: 5; 70-71). He referred to this hypothesis as the 'backward-failure theory' (Harrison 1935: 70), and used it to argue that many of the symptoms and signs of patients in heart failure are the result of a build-up in pressure behind the chambers of the heart (Harrison 1935: 71). However, this theory was not identical to Hope’s (1839) theory\(^\text{31}\), as it did not require an obstruction to the circulation to begin the sequence of enlargement of

\(^{31}\) Or at least how Hope’s theory was understood by general physicians at the end of the nineteenth century according to Mackenzie (1913).
heart chambers backwards in the circulation (Harrison 1935: 86). Instead Harrison used some research done by Ernst Henry Starling (1866-1927) and colleagues to argue that a heart chamber, should it lose its strength for any reason (not just as the result of an obstruction), could still maintain the cardiac output as a consequence of the increased pressure in the ventricle stretching the muscle before contraction (Harrison 1935: 82).

Starling and his colleagues had been doing physiological research in the 1910s using what is known as the 'heart lung preparation' (Patterson et al 1914). This apparatus allowed them to explore the relationship between the pressure and volume of the blood in the chambers of the heart at the end of filling, and the work the heart did when it squeezed to empty. Starling and his colleagues (Patterson et al 1914) found that the greater the volume of a heart chamber at the end of filling the harder it squeezed to eject the blood (Harrison 1935: 83). This relationship between filling volume and stroke work of the heart was referred to as ‘Starling’s law of the heart’ (Harrison 1935: 82). Additionally, Starling (Patterson et al 1914) found large filling pressures stretched the chamber more and increased its volume, and that there was a similar relationship between filling pressure and stroke work as filling volume and stroke work (Harrison 1935: 83).

Starling (Patterson et al 1914) also found that at low pressures a relatively slight increase in pressure was accompanied by a relatively great rise in the stroke work, but at higher initial pressures a greater increase in pressure is necessary to cause similar rise in stroke work (Harrison 1935: 83). Represented graphically the relationship between filling pressure and stroke work makes a curve which becomes progressively less steep as filling pressure rises.
These researchers also drew attention to the phenomenon of cardiac ‘fatigue’. Hearts in their apparatus which were working harder than normal would eventually become unable to squeeze as hard as they had previously for a given amount of filling pressure (Patterson et al 1914). A ‘strong’ heart would gradually become a ‘weak’ heart. This property would come to be referred to as a reduction in ‘contractility’ (Harrison 1950), and was often represented graphically as a ‘family’ of progressively less steep Starling curves (see figure 3).

Figure 3: Family of starling curves. As the heart fatigues, the curve which represents how stroke work changes with filling pressure becomes less steep. Later on this property becomes synonymous with heart failure (From Harrison and Resnik 1958: 108).

Harrison used these findings to provide a new explanation for the symptoms and signs of patients with heart failure (Harrison 1935: 245). A heart which for any
reason lost its ability to squeeze hard enough to maintain an adequate circulation at a given filling pressure could either fail to pump sufficient blood forward in the circulation, or fail to do so without increasing its filling pressures. These increased filling pressures were deemed the cause of the retention of fluid in the lungs and elsewhere in the body which had been associated with problems of the heart for so long (Harrison 1935: 246-247). Harrison argued that:

“Congestive heart failure may therefore be defined as a failure of the heart to maintain a normal output in the presence of a normal venous pressure, and it becomes manifest when the degree of dilatation required to maintain the cardiac output at a relatively normal level produces a rise in venous pressure sufficiently marked to lead to detectable congestive phenomena either in the greater or lesser circulation” (Harrison 1935: 246-247).

Stage 4 - Forward failure comes back
According to some sources Harrison's was the theory of heart failure which most students of medicine were taught in the early 1940s America (Warren and Stead 1944: 138). However, following Harrison’s publication of Failure of the Circulation (1935), several researchers had made observations which they argued were at odds with this received view (Little 1949: 207).

For instance, Starr (1940) observed that in many patients who died of heart failure central venous pressures remained high after death. This was interpreted to mean that the inefficiency of the heart’s action could not be the cause of this raised pressure which persisted even after the heart had stopped beating (Little 1949: 207). Warren and Stead (1944) found that in the early stages of heart failure many patients’ central venous pressure only became raised after the patient had begun to put on weight as a result of the fluid accumulation, and after the patients’ plasma volume had started to rise (Little 1949: 207). This was interpreted to mean that a rise in central venous pressure could not be the cause of fluid accumulation as it only started to rise after fluid had started to accumulate (Little 1949: 207).
A.J. Merrill (1946) drew attention to the observation that patients in heart failure tended to have lower than normal concentration of red blood cells in their blood, and argued that if fluid was being forced out of blood vessels this would increase to the concentration of red blood cells (Merrill 1946: 389). Merrill also suggested that these observations “point to a renal factor in heart failure”, as the kidneys were considered an important organ with respect to the regulation of total body water (Merrill 1946: 389).

Merrill (1946) did research which investigated the “renal factor in heart failure”. He measured the central venous pressure and cardiac output of heart failure patients, and compared his results with measurements of renal plasma flow in the same patients. He found that patients in heart failure often had markedly reduced renal plasma flow, and interpreted this as further evidence of a renal factor in the retention of fluid in the patients’ bodies (Merrill 1946: 397). He also found that low renal plasma flow did not correlate with high central venous pressures in patients with heart failure, but it did correlate with low cardiac output in these patients (Merrill 1946: 396). Furthermore, this latter relationship was not linear, with small reductions in cardiac output producing large reductions in renal blood flow (Merrill 1946: 396). Merrill interpreted this to mean that high venous pressures (and therefore the backwards theory of heart failure) could not be the cause of the fluid retention, and that low cardiac output (and therefore the

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32 It is this argument that there may be a renal factor in heart failure that makes Merrill and the work he reviews relevant to the history I have produced. By time Harrison published the first edition of *Harrison’s Principles of Internal Medicine*, his explanation for heart failure had been changed slightly in comparison to that which he presented in *Failure of the Circulation* (1935). In 1935, Harrison’s focus was on right heart failure as much as it was on left heart failure. In 1950, left heart failure had become the main focus of attention, and all the symptoms and signs of heart failure (including systemic fluid accumulation) were being explained by appealing to left heart failure alone. This change needs explaining, and Harrison (1950) cited Merrill’s (1946) paper as a review of the renal factor in heart failure which explains this change. Merrill and Harrison were also brothers-in-law.
forwards theory of heart failure) could be the cause of this phenomenon (Merrill 1946: 396).

Merrill was aware of previous work done by other researchers (Dole et al. 1946) which found that reductions in renal plasma flow did not produce a reduction in the clearance of marker substances, and that dogs in circulatory shock (with poor perfusion of their tissues with blood) also had very low renal plasma flow in comparison to their cardiac output (Merrill 1946: 395-396). These findings were interpreted to mean that the blood flow to the kidney would be decreased in times of low cardiac output in order to preserve blood flow to other organs which did not function so well when under-perfused (Merrill 1946: 396). Merrill suggested that something similar happened in heart failure in human patients when cardiac output dropped, and that the under-perfusion of the kidney resulted in the retention of fluid (Merrill 1946: 396).

Even though Merrill argued that the backward failure theory could not explain the build-up of fluid in the body, he was keen to emphasise that this did not invalidate the backward failure theory with respect to the build-up of fluid in the lungs:

“The demonstration that salt and water are retained in patients with chronic cardiac decompensation on the basis of ‘forward failure’ in no way invalidates the ‘backward failure’ theory of pulmonary congestion. All the phenomena observed on the ward are in accord with the concept that the left ventricle usually fails before the right. The assumption that this causes a rise in venous pressure in the lungs seems acceptable” (Merrill 1946: 398-399).

So instead of rejecting the backward failure theory entirely, Merrill (1946) just rejected it as important in producing symptoms and signs which Harrison (1935) associated with the failure of the right ventricle. Merrill (1946) focused instead on the left ventricle, the failure of which was now used to explain both systemic and
pulmonary congestion. It should be noted, however, that heart failure in this period was still diagnosed from the presence of the appropriate symptoms and signs. These decisions represent yet more historically contingent choices. Merrill could have rejected the back pressure theory entirely. Alternatively, he could have argued that patients with the appropriate symptoms and signs did not always have heart failure, if he defined and diagnosed the disease using increased filling pressures. It should also be noted that by this time the 'signs' of heart failure have come to include radiographic abnormalities, such as radiographic pulmonary oedema, or radiographic cardiomegaly (see figure 4).

<table>
<thead>
<tr>
<th>Major criteria</th>
</tr>
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<tbody>
<tr>
<td>Paroxysmal nocturnal dyspnea or orthopnea</td>
</tr>
<tr>
<td>Neck-vein distention</td>
</tr>
<tr>
<td>Rales</td>
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<tr>
<td>Cardiomegaly</td>
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<tr>
<td>Acute pulmonary edema</td>
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<tr>
<td>S3 gallop</td>
</tr>
<tr>
<td>Increased venous pressure &gt;16 cm of water</td>
</tr>
<tr>
<td>Circulation time ≥25 s</td>
</tr>
<tr>
<td>Hepatoljugular reflux</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Minor criteria</td>
</tr>
<tr>
<td>Ankle edema</td>
</tr>
<tr>
<td>Night cough</td>
</tr>
<tr>
<td>Dyspnea on exertion</td>
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<tr>
<td>Hepatomegaly</td>
</tr>
<tr>
<td>Pleural effusion</td>
</tr>
<tr>
<td>Vital capacity ≤1/3 from maximum</td>
</tr>
<tr>
<td>Tachycardia (range of ≥120/min)</td>
</tr>
<tr>
<td>Major or minor criteria</td>
</tr>
<tr>
<td>Weight loss ≥4.5 kg in 5 days in response to treatment</td>
</tr>
</tbody>
</table>

For establishing a definite diagnosis of CHF, 2 major criteria or 1 major and 2 minor criteria must be present.

Figure 4: The Framingham criteria for the diagnosis of heart failure (Yturralde and Gaasch 2005: 315). Developed in the late 1940s in an effort to standardize the diagnosis of heart failure for research purposes. They show that in this period the diagnosis of heart failure was made largely clinically, with some supplementary information from radiographic examination. The Framingham Heart Study is a large prospective cohort study, that follows thousands of patients forwards in time to try to uncover the causes and risk-factors for heart failure. The study was begun in the 1940s, began reporting results in the 1970s (McKee 1971) and is still running today. (CHF stands for congestive heart failure).

Merrill also suggested that a substance known as 'renin' was probably involved
in the maintenance of clearance of substances presented to the kidney. He speculated that renin did this by constricting the blood vessels leaving the tissue which filtered the blood to make primitive urine, so that the pressure in this tissue remained high (despite the low flow rates of plasma into the kidney) and substances in the blood were still forced from the blood vessels into the primitive urine (Merrill 1946: 396). Merrill measured the concentration of renin in the blood leaving the kidney and found that it was indeed high (Merrill 1946: 396-397). He interpreted this finding as supporting his suggestion that renin caused the maintenance of clearance of substances presented to the kidney (Merrill 1946: 397). Research into renin has a history dating back to 1898 (Basso and Norberto 2001), particularly in connection with the development of animal models of hypertension in man (Van Epps 2005), but this is the first reference to its causal role in heart failure I can find. This history continues with the development of knowledge out of endocrine systems which are considered important to the aetiology of heart failure today (the renin-angiotensin-aldosterone system) (Basso and Norberto 2001).

Stage 5 - Eugene Braunwald (1929-present)
I have described how Harrison (1935) associated heart failure with an inability of the heart to squeeze hard and throw blood forwards in the circulation without increased filling pressures, and how Merrill (1946) focused attention on the left side of the heart. It is worth emphasising that Merrill focused attention on the whole left side of the heart, and not just on the heart muscle itself. On this view, a defect of any tissue which meant that the heart could not pump an adequate volume of blood forwards unless filling pressures behind the left side of the heart were raised was deemed to be heart failure. This included conditions that meant larger filling pressures were required just to get blood into the heart chambers,
even if the heart muscle itself was healthy. Examples of such conditions include mitral valve stenosis (where the opening between the left atrium and the left ventricle is narrowed), or pericardial effusion (where the sac which surrounds the heart fills with fluid and compresses the heart chambers).

By the 1960s this version of heart failure was coming under pressure from some physicians who, for various reasons, wanted to focus more closely on the contractile properties of the left ventricular muscle itself. In the 1962 edition of the textbook which he edited, Harrison argued that focusing entirely on the left ventricular muscle would be a mistake, as this move would mean that some patients with the appropriate symptoms and signs would no longer be classified as having heart failure:

“Some believe that this term [heart failure] should be applied only to myocardial failure and should even be still further limited to failure of the ventricular myocardium. This concept holds that that the congestive syndromes resulting from stenosis of the atrioventricular valves or from pericardial disease are not heart failure. In our opinion ‘heart failure’ should include all disorders that occur as a result of the inability of the heart to receive and propel blood which is offered to it. The principle involved is the same whether heart failure is due to aortic stenosis or to mitral stenosis, even though in the one case the inadequacy of the thick left ventricle, and in the other the incompetence of the thin atrium. In these patients and in patients with endocardial fibrosis or with constrictive pericarditis, congestive phenomena due to structural disease of the heart are observed, and the restriction of the term ‘heart failure’ to the disorders in which the ventricular myocardium is at fault can lead only to confusion” (Harrison 1962: 1379).

Despite these concerns, in the 1970 edition of Harrison’s Principles of Internal Medicine heart failure was “defined as the condition in which an abnormality of myocardial function is responsible for the inability of the ventricles to deliver adequate quantities of blood to the tissues at rest or during normal activity” (Braunwald 1970: 1117). The author of the chapters on cardiology in this edition has changed the definition of heart failure from the one advocated by Harrison in
earlier editions. Now, instead of focusing on the whole left side of the heart, focus had shifted onto the contractile properties of the ventricular muscle alone.

The author of these chapters on cardiology in this 1970 edition of *Harrison’s Principles of Internal Medicine* was Eugene Braunwald. Braunwald is “probably the most famous academic cardiologist of his generation” and has been an important figure in cardiology and medical education from the 1960s to the present day (Ross 1994: 161). In 1968 he co-authored *mechanisms of contraction in the normal and failing heart*, which made a review of the literature which had investigated the contractile properties of the muscle of failing hearts. This book is revealing of the reasons Braunwald and other physicians felt that the definition of heart failure should focus on the contractile properties of the ventricular myocardium:

“Since William Harvey’s discovery of the pumping action of the heart, physiologists have asked two fundamental questions concerning the contraction of the heart: what are the events that constitute a normal cardiac contraction, and how are these events controlled so that the normal heart can adjust its performance almost instantly to meet the rapidly varying requirements of the peripheral tissues? The clinician has added a third question: how are these events disturbed when the heart fails? The clinician’s interest in the first two questions has been stimulated, in large measure, by the expectation that elucidation of the fundamental cause of heart failure must await their answers” (Braunwald et al 1968: ix).

Braunwald (1968) argued that if the “fundamental cause” of the disease was to be understood, heart failure must be reduced to a malfunction of the ability of the

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33 Braunwald’s work on left ventricular contractility, as well as that of his colleagues like J. Ross, E.H. Sonnenblick and R. Folse, is widely cited by the researchers I refer to in stages 6, 7, and 8. Perhaps more importantly, the researchers in these stages all make use of some measure of left ventricular ejection fraction, and Braunwald is an important actor in the story of the development and acceptance of the measurement of ejection fraction in the diagnosis of heart failure. It was Braunwald and Folse (1962) who developed the first techniques, radionuclide ventriculography, that could do this before echocardiography became widespread. Much of the early literature on systolic function uses this technique to measure contractility, and not echocardiography (e.g. Mattleman et al 1983; Dougherty et al 1984; Soufer et al 1985; Marantz et al 1988; Gadsbøll et al 1989).
heart muscle to contract. This move meant that patients with defects affecting the left side of the heart, but not of the ventricular muscle itself, were no longer considered to have heart failure. For Braunwald, “a defect of myocardial contraction always exists in heart failure” (1968: 139). Consequently, he argued it was “important to distinguish heart failure from states of circulatory insufficiency in which the myocardial function is not primarily impaired (such as cardiac tamponade, hemorrhagic shock or tricuspid stenosis)” (Braunwald et al 1968: 139).

Braunwald (1968) also developed a number of new ways in which heart failure might be measured. He argued that a very ‘sensitive’ way to detect decreased myocardial contractility is to measure ventricular filling pressures, cardiac output and oxygen consumption using intra-cardiac catheterisation and respirometry before and after exercise (Braunwald et al 1968: 153). A subject in heart failure may have normal ventricular filling pressures before exercise, but after exercise pressures were expected to be greatly raised relative to the change in cardiac output. A less sensitive (but still very good method according to Braunwald) was to measure the ratio of stroke volume to the end diastolic volume (Braunwald et al 1968: 153). If a failing ventricle needed to be stretched to a greater degree than a normal ventricle to produce the same stroke volume then it was expected that in failing ventricles this ratio would be reduced. Braunwald was not the first to reason that this would be the case, both Hope (1839) and Corvisart (1812) discussed this possibility, but he did suggest that this index should be measured to make a diagnosis. He also described how this index could be measured using the rate at which ventricular blood containing radioisotope marker was diluted by new blood being presented to the ventricle (Folse and Braunwald 1962). This
index came to be known as the left ventricular ejection fraction (LVEF).

So, Braunwald developed and adopted new definition of heart failure. If patients with pericardial tamponade or stenosis of the atrioventricular valves did not have heart failure even if they developed breathlessness and signs of fluid accumulation, then Braunwald must not have considered the appropriate symptoms and signs to be the most accurate way to diagnose heart failure. Instead, he argued that measurement of left ventricular contractility should be used to define and diagnose heart failure. Braunwald (1968; 1970) therefore chose to reject Harrison’s (1962) arguments that the appropriate symptoms and signs should be used to define and diagnose heart failure. At the same time however, Braunwald preserved connections with Harrison’s work by continuing to recognise the importance of contractility and Starling's law of the heart.

Stage 6 - Loss of confidence in symptoms and signs
However, despite Braunwald et al’s (1968) suggestion that heart failure could be measured in these new ways, the invasive nature of these procedures probably prevented them from becoming widespread diagnostic techniques (Sutton 2011). The application of the diagnostic practices developed by Braunwald and his colleagues seem limited to a research context. This would remain the case for techniques that made use of intra-cardiac catheterisation, but following the introduction of echocardiography (ultrasound of the heart) the measurement indices of contractility became more accessible to the practising physician.

Echocardiography was first developed by Swedish physician Inge Edler in the mid-1950s (Achierno and Worrell 2002: 197). Cardiologists found it difficult to interpret the one dimensional images which were produced at first, but the
imaging technique became more popular following the introduction of two-dimensional imaging in the late 1960s (Edler and Lindstrom 2004: 1603). It seems that echocardiography became widespread in amongst cardiologists in Europe and America in the early 1970s (Edler and Lindstrom 2004: 1599; Sutton 2011).

The early focus of cardiologists was on the echocardiographic evaluation of the mitral valve, but within a short space of time attention had shifted to the function of the left ventricular myocardium (Sutton 2011). Following the widespread introduction of echocardiography, it became possible for cardiologists to measure indices of left ventricular function without the need to resort to invasive procedures involving cardiac catheterisation (Mattleman et al 1983: 417). “The left ventricular myocardium was the thing that you really needed to get a handle on, and echocardiography did it” (Sutton 2011).

Amongst the echocardiographic indices measured by cardiologists in the 1970s to assess left ventricular function was the LVEF (Sutton: 2011). Other indices measured included chamber size and wall thickness (Sutton: 2011). Even though it was recognised that LVEF was not the very best method of assessing left ventricular contractility (Braunwald et al 1968: 153), the measurement of LVEF using echocardiography became a widely used method of determining cardiac function, particularly amongst American cardiologists (Sutton 2011). It is at this time that “ejection fraction was chosen by the cardiology community at large and remains the index overwhelmingly used to assess cardiac function in both clinical and experimental studies” (Carabello 2002: 2701).

In the 1980s, physicians used this new method of measuring contractility in
studies to investigate the relationship between patients with the symptoms and signs of “chronic heart failure”\(^{34}\) and various other abnormalities which were associated with heart failure (such as reduced contractility of the heart muscle, radiographic abnormalities, and reduced cardiac output and increased filling pressures). All of these studies found significant disagreement between these measurements associated with poor ventricular function and the signs and symptoms associated with heart failure (Cease and Nicklas 1986: 429; Mattleman et al 1983: 417; Stevenson and Perloff 1989: 884; Gadsbøll et al 1989: 1017; Marantz et al 1988: 607)\(^ {35}\). With respect to ejection fraction, all of these studies found a poor correlation between the level of decreased LVEF and the severity of clinical signs and symptoms. Many patients with low LVEF had no signs and symptoms of the disease, and vice versa (Mattleman et al 1983: 419; Gadsbøll et al 1989: 1027).

As there was only a poor correlation between the presence of a reduced LVEF and the appropriate signs and symptoms of heart failure, many physicians lost faith that symptoms and signs alone could accurately predict which patients were in heart failure (Gadsbøll et al 1989; Stevenson and Perloff 1989; Cease and Nicklas 1986; Mattleman et al 1983). Many physicians argued that symptoms and

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\(^{34}\) Chronic heart failure is a syndrome in which the symptoms and signs of heart failure develop slowly over a period of weeks and months. This contrasts with acute heart failure, where symptoms and signs develop quickly over a period of days. The relationship between symptoms and signs, radiographic abnormalities, reduced cardiac output and increased filling pressures had been established in patients with acute heart failure during the 1950s, 1960s and 1970s (Freis 1951, Gilbert 1954, Fluck 1967, Fowler 1968, Hamosh 1971, Rathshin 1972, Forrester 1977). However, it was not until the 1980s that these relationships were investigated in patients with chronic heart failure (Stevenson and Perloff 1989).

\(^{35}\) Studies like these were (and still are) frequently cited in the medical literature to support the view that a purely clinical diagnosis could not accurately detect patients with reduced systolic function. The ESC (2012; 2016) cite Fonseca (2006) as a review of the medical literature that showed that a purely clinical diagnosis is not accurate. Fonseca (2006) cite Stevenson and Perloff (1989) as a study that shows this to be the case. Stevenson and Perloff (1989) cite Mattleman et al (1983) and Cease and Nicklas (1986) as research that produces similar findings. Gadsbøll et al (1989) is cited directly by the ESC (2005, Remme and Swedberg 2001).
signs should not be used alone to make diagnostic decisions:

“The fact that most physical signs of heart failure have an unpredictable relationship to radiographic and hemodynamic results and the finding that the absence of these signs by no means excludes the existence of a severely reduced ventricular function further questions the validity of physical examination as the sole basis for therapeutic decisions” (Gadsbøll et al 1989: 1027).

“Although several attempts have been made to define uniform criteria for the diagnosis of heart failure, one may ask whether clinical manifestations of heart failure are sufficiently reliable to constitute the basis for diagnosis and management of this condition, or whether our clinical decisions should rather depend on one of the several modern methods of investigation, offering quantitative assessment of cardiac function” (Gadsbøll et al 1989: 1017).

However, it was not common for physicians to argue that the diagnosis of heart failure should be based solely on a reduced LVEF either. Rather, most physicians seem to have argued that both the appropriate symptoms and signs, and evidence of cardiac dysfunction should be present for an accurate diagnosis to be made (Gadsbøll et al 1989; Stevenson and Perloff 1989; Cease and Nicklas 1986; Mattleman et al 1983; Marantz et al 1988). Nevertheless, many physicians argued that the use of symptoms and signs alone could not be trusted as an accurate method of diagnosis any more. In most instances, this lack of faith in the signs associated with heart failure extended to radiographic abnormalities (such as pulmonary oedema and cardiomegaly), which had been found to be absent in many patients who were breathless and had a reduced LVEF (Gadsbøll et al 1989; Stevenson and Perloff 1989).

It was also found that physicians frequently disagreed over the presence and absence of the symptoms and signs associated with heart failure when they examined the same patient, a phenomenon referred to as “interobserver variation” (Gadsbøll et al 1989: 1019). This only contributed to the loss of
confidence in the diagnostic value of symptoms and signs associated with heart failure when used alone.

So, in response to the finding that patients with the appropriate symptoms and signs frequently did not have evidence of cardiac dysfunction some physicians rejected the use of symptoms and signs alone as the most accurate method of making a diagnosis. Instead, many physicians adopted the combination of both the appropriate symptoms and signs, and a reduced LVEF as the preferred method for the diagnosis of heart failure.

Stage 7 - Systolic and diastolic heart failure
It should be noted that this interpretation of how best to diagnose heart failure was again not the only one available to physicians in this period. They could, for instance, have chosen to reject the use of symptoms and signs entirely, and make a diagnosis based entirely on a reduced LVEF. Alternatively, physicians could have chosen to reject the measurement of LVEF as diagnostically valuable. In addition to these choices, physicians could have argued that a reduced LVEF did indeed cause heart failure, but that there must also be another form of cardiac dysfunction in addition to reduced contractility of heart muscle. Many physicians adopted this latter position.

In accordance with this position, some physicians continued to use the appropriate symptoms and signs as the reference standard for the diagnosis of heart failure, and evaluated the diagnostic accuracy of LVEF against this standard (Dougherty et al 1984; Soufer et al 1985; Marantz et al 1988). These investigators also found a lack of correlation between symptoms and signs associated with heart failure and LVEF, but did not interpret this as evidence of the limited
diagnostic value of symptoms and signs. Instead these investigators argued that it was LVEF that had the limited diagnostic value:

“To determine the relationship between LVEF and clinically diagnosed CHF [congestive heart failure] we compared resting LVEF determined by radionuclide angiography with diagnosis of CHF by clinical criteria in 407 patients, based on clinical data collected by a cardiology fellow. Of 153 patients with a low LVEF (<0.40), 30 (20%) met none of the criteria for CHF. Conversely, of 204 patients with normal LVEF (>0.50), 105 (51%) met at least one of the criteria” (Marantz et al 1988: 607).

“This implies that clinical judgement provides information about congestive heart failure that cannot be obtained from nuclear scan” (Marantz et al 1988: 610).

However, the finding that LVEF had limited diagnostic value did not lead to its rejection as a diagnostic tool. Instead, the finding that the symptoms and signs associated with heart failure could occur in patients with a normal LVEF was interpreted as evidence of the existence of a large group of patients who were in heart failure despite having hearts which could eject blood adequately:

“The objective of this study was to test the hypothesis that among patients with clinical heart failure, normal global systolic function is common, and that pulmonary congestive symptoms are associated with abnormal left ventricular compliance. To this end, we prospectively reviewed patients with documented CHF by Framingham criteria for prevalence of normal global EF, clinical characteristics that distinguish patients with normal EF from others, and diastolic performance” (Dougherty et al 1984: 778).

Thus patients with the symptoms and signs associated with heart failure were

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36 These papers are frequently cited in the medical literature in support of the view that diastolic heart failure should be recognised. The ESC (2012; 2016) cite Paulus et al (2007) as a review of the diagnosis of diastolic heart failure, and Paulus et al (2007) cites Soufer et al (1985) and Dougherty et al (1984) as seminal studies that identified this form of heart failure. Marantz et al is cited directly by the ESC (2005, see also Remme and Swedberg 2001), by Yturralde and Gaasch (2005), and by Vasan and Levy (2000). I have presented Gadsbøll et al (1989) as arguing that a purely clinical diagnosis is not accurate, and Marantz et al (1988) as highlighting the limitations of LVEF. Care is required here, as I do not want to misrepresent these investigators. Both Marantz and Gadsbøll and colleagues argue for using a combination of clinical signs and ‘objective’ measurements, and they argue for plural approaches depending on circumstances. They frame their work in opposition to each other, but when they find that abnormal clinical findings accompany normal LVEF (Gadsbøll et al 1989) and that abnormal LVEF accompanies normal clinical findings (Marantz et al 1988), they both swap allegiance and say that the standard they have discredited has value sometimes.
split into two groups by these investigators; those with reduced LVEF and those with preserved LVEF (Sutton 2011). The phase of the heart's action when it squeezes and ejects blood is called 'systole', and patients with the appropriate symptoms and signs and a reduced LVEF were referred to as having 'systolic heart failure'. LVEF was thus now understood to only be a measurement of systolic function, and not of cardiac function in general. Furthermore, it was suspected that many of the patients with preserved LVEF had hearts with reduced compliance, or “stiff heart syndrome” (Dougherty et al 1984: 778), and that the signs and symptoms of heart failure were the result of a difficulty during filling. The filling phase of the heart's action is called 'diastole', and many patients with the appropriate symptoms and signs and a preserved LVEF were said to have 'diastolic heart failure'.

Investigators began to search for ways of measuring diastolic dysfunction. A large number of different indices have been proposed since the 1980s (ESC 2012: 1803). These are too numerous and complex to describe here, but is still useful to consider how many of these indices came to be accepted. In many cases a proposed method of measuring diastolic function was evaluated by determining whether they were abnormal in patients with heart failure and preserved LVEF, but normal in patients without heart failure (Soufer et al 1985: 1035). So measures of diastolic dysfunction evaluated using symptoms and signs alone were used to make the diagnosis of heart failure.

Despite the development of many methods of measuring diastolic function, many physicians did not trust in them to provide valid measures of diastolic function. Many physicians made the diagnosis of heart failure using the symptoms and
signs associated with heart failure, and then used indices of systolic and diastolic dysfunction to sub-classify the patients so selected (Dougherty et al 1984; Soufer et al 1985; Marantz et al 1988; Aguirre 1989):

“For these and other reasons, many if not most clinicians make the clinical diagnosis of diastolic dysfunction or failure if a patient simply has heart failure in the presence of a normal ejection fraction” (Zile 2001: 781).

Stage 8 - Both symptoms/signs and cardiac dysfunction become required Despite the lack of clear consensus over which measurements constituted the best way to diagnose diastolic heart failure (Brutsaert and Stanislas 1997: 225), and the common practice of using symptoms and signs associated with heart failure to evaluate those measurements, some physicians still considered symptoms and signs to be unreliable way of making a diagnosis of heart failure, particularly when made by physicians working in primary care (Remes et al 1991; Wheeldon et al 1993; Task force on heart failure of the European society of cardiology 1995; Mosterd et al 1999; ESC 2008; Maestre et al 2009). Such physicians were keen to promote the use of “objective” measurement of cardiac dysfunction in the assessment of diastolic heart failure to complement the measurement of LVEF to diagnose systolic heart failure, and the use of echocardiography in particular (Wheeldon et al 1993: 17). To accomplish this, various measures of diastolic dysfunction were incorporated into guidelines for the diagnosis of heart failure (ESC 2012: 1803).

According to these physicians, “echocardiography should be used routinely for the optimal diagnosis of heart failure. The test is widely available, simple and safe. The functional integrity of the valves, chamber dimensions, ventricular hypertrophy, and systolic and diastolic function can all be assessed” (Task force on heart failure of the European Society of Cardiology 1995: 745). From 1995 the
European Society of Cardiology included objective measurement of cardiac dysfunction in their definition of heart failure, and echocardiography was their recommended method of making such measurements (Task force on heart failure of the European Society of Cardiology 1995: 742; ESC 2008: 2391)\textsuperscript{37}. Some investigators even evaluated the diagnostic accuracy of the symptoms and signs associated with heart failure using echocardiography as a standard (Maestre et al 2009: 55). To such physicians echocardiography was an “essential tool in the evaluation of persons suspected of heart failure” (Mosterd et al 1999: 448).

As my story reaches the twenty-first century, the different approaches to the diagnosis of heart failure discussed in the last chapter are apparent. Doctors who agree with the ESC (Such as Hobbs et al 2010 and Fuat 2003), and require both symptoms and signs and objective evidence of cardiac dysfunction for a diagnosis to be made, are working in what I have called stage 8. Those doctors that disagree with the ESC (such as Jolobe 2006; and Vasan and Levy 2000), and make no such requirement (particularly for the diagnosis of diastolic heart failure), are working in what I have called stage 7.

Conclusion
I have presented a history of the development of knowledge of heart failure from the early 1800s to the present day, which is organized into eight stages. Each stage is characterised by a different set of diagnostic practices and/or a different pathophysiological understanding of the disease. In all but one case (the transition from stage 4 to stage 5) historical actors argued for the change from one stage to another because of some empirical experience that conflicted with

\textsuperscript{37} This is the position of the ESC, which is the starting point for my historical investigations.
their expectations. The transition from one stage to the next always involved the (often explicit) rejection of some aspect of how heart failure was understood in an earlier stage by the historical actors the subsequent stage. However, aspects of the earlier stages were also always conserved in the later stages. It should also be apparent that exactly what it was about an earlier stage that was rejected or conserved at a later stage was not fully determined by the observations made by my actors. Historical actors always had alternative ways to resolve their conflicts with experience, even if they themselves did not recognise this.

In summary, Mackenzie rejected the view held by many nineteenth century doctors that valvular lesions and heart murmurs were sure signs of heart disease. He did this because he observed that many of his patients with valvular lesions and heart murmurs did not become ill despite their alleged disease. He also rejected the back-pressure theory as an explanation for the symptoms of patients with heart disease. Even though Mackenzie rejected all this, he still conserved the connection made by early nineteenth century doctors between the syndrome of breathlessness and fluid accumulation and heart disease. Mackenzie was not forced to resolve his conflict with experience in this way. He could have preserved the view that valvular lesions of the heart and heart murmurs were sure signs of disease by accepting that patients with heart disease did not necessarily have a poor prognosis.

Harrison rejected Mackenzie’s forward failure theory, because he observed that many patients who were in heart failure did not have reduced cardiac outputs. In making this observation, he preserved Mackenzie’s view that patients with the syndrome of breathlessness and fluid accumulation had heart disease, whether
they had lesions of the hearts or not. Even though Harrison argued he had falsified Mackenzie’s forward failure theory, Harrison need not have resolved this conflict with experience in this way. The view that patients with heart failure had a reduced cardiac output could have been preserved if Harrison had concluded that some patients with the syndrome of breathlessness and fluid retention did not have heart failure.

Merrill rejected Harrison's backwards failure theory for right heart failure, instead suggesting that reduced cardiac output from the left side of the heart is what produced systemic fluid accumulation. He did this because of observations like the finding that patients in heart failure tended to have dilute blood, rather than concentrated blood as he expected. As he did this he conserved Harrison’s view that pulmonary congestion was caused by a build-up of pressure behind the left side of the heart. Again, this was not the only way Merrill could have resolved this problem. Merrill could have concluded that the patients with systemic fluid accumulation and dilute blood did not have heart failure. Alternatively, he could also have concluded that Harrison’s backwards failure theory was entirely wrong, instead of just partly wrong.

Braunwald rejected Harrison's (and Merrill's) view that heart failure was not caused solely by a reduction in contractility of the left ventricle, but conserved the notion that contractility is a very important aetiological factor in heart failure. In making this change, Braunwald was not responding to a conflict with experience, but rather argued that it was only by focusing on this aspect of the pathophysiology of heart failure that a deeper and more useful understanding of the disease could be achieved. Alternatively, Braunwald could instead have
focused on a different aspect of the pathophysiology of the disease as it was then understood, such as a reduction in renal blood flow causing salt and water retention. As there was no conflict with experience to resolve, Braunwald also could have continued to research into heart failure in the same way that Harrison and Merrill had before him, without changing how heart failure was understood at all.

On finding that patients with the syndrome of breathlessness and fluid retention did not always have reduced contractility of their left ventricles, doctors in the 1980s rejected the assumption of earlier doctors that the vast majority of patients with heart failure did have reduced contractility of their left ventricles. However, they conserved the notion that patients in heart failure should have some detectable reduction in cardiac function. Alternatively they could have done the reverse, and conserved the notion that patients with the syndrome of breathlessness and fluid retention had heart failure, and rejected the notion that patients in heart failure had some measurable reduction of cardiac function.

Indeed, some doctors (in stage 7) did just this. These doctors set about developing measurements for the sort of cardiac dysfunction which they believed they could not yet measure: diastolic dysfunction. Alternatively, they could have rejected the notion that reduced contractility was a proximal cause of the syndrome of breathlessness and fluid retention, as it was absent in many patients with the syndrome.

After many measurements for diastolic dysfunction had been developed, some doctors (in stage 8) incorporated these indices into their measurements for
cardiac dysfunction, and claimed that some measurement of systolic or diastolic dysfunction was required as “objective” evidence of cardiac dysfunction for a diagnosis of heart failure to be made. These doctors did this largely in response to evidence that patients with the syndrome of breathlessness and fluid accumulation did not always have the radiographic, haemodynamic, and echocardiographic characteristics that they were expected to have. This contributed to the suspicion that a purely clinical diagnosis was not accurate, first raised by doctors in stage 6. As a result, these doctors (in stage 8) rejected the notion that a purely clinical diagnosis of heart failure was accurate. These doctors did not have to respond in this way to this conflict with experience. Instead, they could have responded to it in the same way as doctors in stage 7, by remaining suspicious of the accuracy of their measurements of diastolic dysfunction. Even though doctors in stage 8 disagree with doctors in stage 7, they still conserved the practices of measuring diastolic dysfunction developed by doctors in stage 7.

The transition from one stage to another always involves the rejection of some aspects of the earlier stage by doctors in the later stage, as well as the conservation of some aspects of the earlier stage by doctors in the later stage. I have argued that, over and over again, historical actors did not need to respond to the observations they made in the way that they did. There were always alternative ways of resolving the conflicts with experience they encountered. It follows from this that present day knowledge of heart failure has not been fully determined by the observations that have been made concerning heart failure in the past. This history is not consistent with the view that medics have discovered timeless truths about the diagnosis and physiology of heart failure. And yet, this history is perfectly consistent with the view that medical knowledge is historically
contingent, and need not have developed in the way that it has. I argue that this is a good reason to suspect that medical knowledge is actually historically contingent, and to wonder what an epistemology that can cope with this would be like. Even so, it is also clear from this history that historical actors were not free to adopt any understanding of heart failure that they liked. Empirical observations did play a very important role in constraining how heart failure could be understood. The epistemology that I am looking for does not reduce to knowledge of heart failure being entirely made up and invented. Rather, I need an epistemology that can show how medical knowledge can at once be invented and discovered.
Introduction

In the last chapter I argued that there was good reason to suspect that the knowledge of heart failure that has been produced by medics is historically, socially and culturally contingent. I argued that the knowledge of heart failure that has been produced is not the inevitable result of the collective empirical experience of researchers over the last two centuries. Researchers have not managed to discover timeless facts about what heart failure is, what its pathophysiology is, which patients have it, or how to diagnose it. Knowledge of heart failure could have developed in other ways. I argued that, because of this, it was reasonable to suspect that all knowledge of heart failure is at least partly of our own making. I want to consider the possibility that researchers get to decide, to choose, what heart failure is. Knowledge of heart failure may well be invented.

At the same time, however, I emphasised that empirical experience did constrain the knowledge that researchers produced, and that researchers were consequently not free to make heart failure into anything they wanted. Even though researchers did get (in some sense) to choose what heart failure was, they were not free to choose that heart failure was anything they wanted it to be. As researchers were not free to make heart failure into anything they liked, there is a sense in which knowledge of heart failure is also discovered. It appears as though knowledge of heart failure is at once invented and discovered. In this chapter I search for an epistemology presented by contemporary philosophers of medicine that can cope with these demands. Despite a thorough search, I do not find such an epistemology.
Focusing on choice provides a useful framework for discussing the invention and discovery of knowledge. On the one hand, if medical knowledge is completely discovered, then it is fully determined by the way the ‘world is in itself’. If our knowledge is entirely discovered, and we intend to represent the ‘world in itself’ faithfully, we have no choice in this matter, as this knowledge is fully determined by the way nature is. For the purposes of this thesis, I have identified the condition of being ‘discovered’ with a limitation of our freedom to choose how things are. On the other hand, if knowledge of disease is completely invented or made up, then it is the choices made by people that constrain this knowledge, not the natural world. I have identified the condition of being ‘invented’ with having the freedom to choose how things are. I will use this framework to review the contemporary philosophical literature on the epistemology of diagnosis, and consider which aspects of knowledge of disease several different contemporary philosophers of medicine take to be invented and/or discovered.

In the introduction, I referred to some scholars of medicine who hold that at least some aspects of knowledge are discovered (Thagard 1999; Harrison 2015). Issues of the invention and/or discovery of medical knowledge are discussed in the philosophical literature on health and disease. Philosophical discussions of health and disease have for many years been focused on a single question: “are the concepts of health and disease value-free?” (Kingma 2013: 37). It is commonplace to divide the participants in this debate into two camps: the naturalists and the normativists. (Marcum 2008: 63; Kingma 2013: 37). According

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38 This is closely related to Ludwik Fleck’s (1979: 95) arguments that facts are what resist the researcher’s will, and Hasok Chang’s (2012a: 217) arguments that what is real is that which resists our will. At the moment I am equating discovery with finding something that resists our will because this is what the mind independent ‘world in itself’ is supposed to do. Later in this thesis I show (by following Fleck) how it is possible to retain the resistance to our will, and thus discovery, without retaining the problematic notion of the ‘world in itself’.
to the naturalists, at least some medical concepts “are descriptive concepts that can be used to define the objective or real state of a condition or person” (Marcum 2008: 63). Normativists disagree, and argue that all medical concepts “depend upon personal and social values” (Marcum 2008: 63).

From this it may appear that naturalists believe that all aspects medical knowledge are entirely discovered, and fully determined by the way the natural world is. It may also appear that normativists believe that all aspects of medical knowledge are entirely invented, and fully determined by the choices made by researchers. This, however, is not the case. Naturalism and normativism together cover a wide spectrum of philosophical positions, which represent different views about which aspects of medical knowledge are discovered and which are invented.

Naturalists hold that being diseased is to have a biological dysfunction of a bodily organ. They argue that the functional status of an organ is an objective matter, and therefore that disease status is also an objective matter (e.g. Christopher Boorse). Some normativists agree that dysfunction can be assessed objectively, but disagree that this is sufficient to determine disease status, arguing instead that some value laden judgement must also be made to determine if the dysfunction counts as a disease (e.g. Jerome Wakefield). Other normativists argue that the function of an organ is not an objective matter, even though biological facts about its role in keeping the patient alive are (e.g. Lennart Nordenfelt). Some normativist argue that it is not necessary that a condition have a biological cause for it to be counted as a disease (e.g. Rachel Cooper); and others hold that diseases are whatever doctors say that they are (e.g. Tristram Engelhardt).
Given the great variety of positions on health and disease that are available in the philosophical literature, one might expect that at least one of these positions might be able to show me how the development of knowledge of heart failure might at once be invented and discovered. In this chapter I review the contemporary philosophical literature on health and disease and argue that none of these positions is suitable for my purposes.

Elselijn Kingma (2013) has made a useful analysis of the philosophical literature on health and disease, which I will use to explain why this literature is not quite suited to my purposes. Kingma argues that prominent naturalist and normativist positons are “two-layered” (2013). Above I suggested that it might appear that naturalists believe that all medical knowledge is discovered. Kingma argues (correctly, in my view) that this is not so, because naturalists like Christopher Boorse (1975; 1977) adopt a two-layer view of medical knowledge. Boorse’s first layer is knowledge of the biological dysfunction of an organ, like the heart. Knowledge in this layer is supposed to be a purely biological and value free description of the patient (Kingma 2013: 39). It is here that the “objective or real state” (Marcum 2008: 63) of a patient is supposedly described. This layer corresponds to Boorse’s “theoretical health”, which is an unapplied set of concepts said to be used by pathologists (Boorse 1977). On this two-layered view, it is only in the second layer that decisions about whether or not a condition is disvalued are supposed to be made (Kingma 2013: 39). Here it is decided whether or not a condition is serious, or whether or not it should be treated, or whether or not it should excuse the patient from legal responsibility (Kingma 2013: 39). This corresponds to Boorse’s “practical health”, which is an applied set of concepts said to be the set of concepts with which doctors actually work (Boorse 1977). So, for example, doctors in the clinic may choose not to
distinguish patients with different pathologies if the treatment for these patients is the same. For all practical purposes these patients are placed in the same group, but are still theoretically distinguishable.

It may seem as though the two-layered approach to disease can show how knowledge of disease is at once invented and discovered. First layer knowledge is knowledge of the biological aspects of disease, which for naturalists like Boorse is knowledge of the functional status of a patient’s organs. This is fully determined by the way that the world is in itself, and is therefore discovered (if it is known at all). Second layer concepts of practical health depend upon the choices made by people about whether the biological states described in the first layer are good or bad, and is thus at least partly invented. Thus, knowledge in this second layer is both invented and discovered.

However, for my purposes this integration of invention and discovery will not do. I am looking for an epistemology in which all aspects of medical knowledge are at once invented and discovered – including of biological aspects of disease. As Kingma (2013: 53) argues, in the two-layered approach, the biological and the evaluative are kept strictly separate, and the biological aspects of disease are supposedly entirely discovered. In the epistemology that I am looking for, the biological aspects of disease would also be invented and discovered.

39 Kingma objects to this, and follows others (including Engelhardt 1985b: 158; Margolis 1976: 249; Stempsey 2000: 3) in arguing that human value judgements enter into the supposedly value-free first layer concepts as well (Kingma 2007; 2013: 40). For Kingma, the account of health and disease presented in the first layer is not the inevitable result of objective investigation, but rather is the “contingent result of social and historical processes” (2013: 37). So instead of simply adding purely factual, biological considerations to social, value-laden considerations, Kingma calls for naturalism and normativism to be integrated in a way that preserves the strengths of each position whilst addressing their weaknesses (2013: 37; see also van der Steen and Thung (1988: 117) who also call for naturalism and normativism to be integrated). Kingma suggests we do this by showing how it is possible to make naturalistic descriptions of disease that are the contingent result of social and historical processes, without having to say that any part of the concept of health and disease is value-free, and without having to define disease in an explicitly value-laden language (2013: 51-53). Rather than doing this, I suggest that philosophers should explore how knowledge of the biological aspects of disease can at
Kingma also argues at least one normativist (Jerome Wakefield) also endorses the two-layered view of health and disease (Kingma 2013: 53). I argue below that this is correct, and that some form of two-layered view is even more widespread than this. This widespread adoption of two-layered views is problematic for me, as it does not meet the epistemological demands that I have made.

I should emphasize that my claim that philosophers tend to treat biological facts relevant to disease classifications as entirely discovered does not entail that philosophers tend to treat disease classifications as entirely discovered. Discussions of nosology (the study of the classification of diseases and the diseased) are abundant in philosophical reflections on medicine, and have been for many years. Nevertheless, these discussions often leave some aspect of the biology of disease unproblematicized. For example, Victor McKusick (1969) argues that many contemporary disease classifications will have to change, as patients with different clinical presentations are found to have the same genetic cause, and many patients with similar clinical presentations are found to have different genetic conditions. “In genetic nosology both lumping and splitting have a place: lumping in connection with pleiotropism; splitting in connection with genetic heterogeneity” (McKusick 1969: 30). Even though disease classification is problematized here, basic biological facts about the role certain genetic anomalies play in disease causation are not.

According to McKusick (1969: 24), “the principal, almost the only, question the nosologist asks is whether syndromes A and B are one and the same entity or separate ones”. He says this contrasts with taxonomists, who are concerned with
how different groups of individuals (species) are related to each other (1969: 24). Some philosophers, however, are concerned with how diseases themselves are classified. Focusing on the example of Langerhans cell histiocytosis (LCH), Mathias Brochhausen (2017) argues that diseases can be classified in different ways for different purposes. So LCH might be seen as a neoplastic disease (a cancer) or as a reactive disease (a disease of the immune system). Such considerations are important, not least because they affect the direction of research and treatment (Brochhausen 2017: 180). However, the biological reality of the entities being classified, like LCH, is not seriously questioned by Brochhausen (2017). Focusing on the philosophical literature on health and disease allows me to focus on questions about how historically contingent human choices influence knowledge of which people are diseased, and how these patients should be classified.

The philosophical literature concerning the opposition of naturalist and normativist positions is extensive, and therefore impossible to review completely here. For the purposes of this discussion, the taxonomy of available positions used is one introduced by Christopher Boorse (1975: 51) and extended by George Khushf (2007)40. Boorse describes three different sorts of philosophical

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40 Elselijn Kingma (2014) has recently proposed a different taxonomy of naturalist and normativist positions. I have not used Kingma’s taxonomy because, as Kingma admits, “important versions of and distinctions within normativism are overlooked” in it (2014: 591). As such, most of the normativists who I discuss here would be classified as “domain 2” normativists by Kingma (2014), and the differences that I want to highlight between (for instance) Nordenfelt (1995) and Cooper (2002) on the one hand and Engelhardt (1974) and Cunningham (2002) on the other would go unrecognised. Marc Ereshefsky has also suggested an alternative taxonomy of positions in this debate, dividing positions into naturalist, normativist, and hybrid positions. This taxonomy is problematic for several reasons (Dominic Murphy’s (2015) taxonomy of objectivism and constructivism has similar problems to Ereshefsky’s taxonomy). Hybrid positions are two-layered in that they require both dysfunction and disvalue to be present for disease to be present. Naturalist positions are thus distinct from hybrid positions, even though naturalists also adopt a two-layered view. Ereshefsky only refers to Reznek (1987), Wakefield (1992) and Caplan (1992) as examples of hybrid theorists. These philosophers hold that value free accounts of dysfunction can be produced, so it seems that philosophers who argue that there can be no value free account of bodily function (like Nordenfelt and Fulford, see section 2 below) should not count as hybrid theorists. However, these philosophers cannot be normativists according to Ereshefsky’s taxonomy, as
position: naturalist (section 1), weak normativist (section 2), and strong normativist (section 3). Khushf further divides the strong normativist into two, recognising strong normativists of the first (section 3.1) and second sort (section 3.2) (2007: 24). Only a few strong normativist philosophers reject any form of two layered account. These include a subgroup of the first sort of strong normativists (section 3.3), but these philosophers do this by arguing that knowledge of disease is entirely invented, and therefore not at once invented and discovered.

Some strong normativists of the second sort (including Kingma’s own views) do argue that all aspects of medical knowledge are at once invented and discovered, but I argue that none of these views are suited to my purposes. I discuss each of these positions in turn.

1 – Naturalist
The most influential naturalist position is Boorse’s “bio-statistical theory” (1977; 1997). As discussed above, the account is explicitly two-layered, and contrasts value-free “theoretical health” with value-laden “practical health” (Boorse 1977: 542). For Boorse to argue that theoretical health is value-free, is also to argue that this concept is entirely discovered and fully determined by the way the world is. Boorse holds that “the classification of human states as healthy or diseased is an objective matter, to be read off the biological facts of nature without need for value judgements” (1997: 4). If disease status can be “read off the biological facts

\[\text{they see some role for biology in defining disease states, and in Ereshefsky’s taxonomy “[f]or normativists, if a state is disvalued it is a disease state” (Ereshefsky 2009: 226). Ereshefsky would also draw no distinction between philosophers who accept that there are absolute biological facts that describe reality, even if disease include biological and non-biological conditions (like Cooper, see section 3), and philosophers who do not accept that there are absolute biological facts (like Engelhardt). Furthermore, Ereshefsky argues that the problem of finding the correct definition of health and disease would dissolve if philosophers concentrated on separating state descriptions and normative claims, and this advice preserves the two layered view that troubles me.}\]
of nature” then we have no choice in the matter as it is fully determined by the way the world is. Disease status is entirely discovered, and not at all invented.

It is worth unpacking this claim a little, to show how important the question of choice has been to naturalist arguments. Boorse understands theoretical health in an organism as “the readiness of each internal part to perform all its normal functions with at least typical efficiency” (1977: 555). Normal functioning is thus central to Boorse’s account. His argument that theoretical health is value-free (and therefore discovered) depends on his being able to show that normal functioning is value-free.

Boorse holds that functions are contributions to goals, and that the goals of any living organism are survival and reproduction (1976: 77; 1977: 556). Several authors (for instance Engelhardt 1976: 263; Agich 1983: 30-32; Brown 1985: 315-316; DeVito 2000: 542; Ereshefsky 2009: 223; Kingma 2014: 601; see Boorse 1997: 24-25) have accused Boorse of simply asserting that these are the goals of living organisms, and thus covertly introducing value judgements (and therefore choices) into his discussion of function. Boorse certainly disagrees with this criticism, and argues that biologists have no choice but to adopt survival and reproduction as the goals of individual living organisms (specifically people) (1976: 84-85; 2002: 76). Following Sommerhoff (1950: 6), Boorse argues that all living systems show goal directed behaviour. According to Sommerhoff “a beast is only distinguishable from its dung” by virtue of its parts being organised in such a way that promotes the continuation of life (Boorse 2002: 76). “I cannot here discuss the problem of defining life. But, if Sommerhoff was right that life just is a kind of natural goal directedness, then it is not arbitrary to take life’s continuation, individual survival and reproduction, as the apical goal” (Boorse 2002: 76). Consequently, Boorse thinks that the selection of survival and reproduction as
goals pertinent to physiology does not involve any “arbitrary, evaluative, or non-naturalistic choice” (Boorse 2002: 77). These, then, are the goals which living organisms do in fact have, and therefore are the goals the physiologist and the medic must use to study function. Furthermore, once these goals are accepted, naturalists like Boorse argue that there is no longer any freedom to choose what the function of a living body should be. This matter is fully determined by the way the natural world is. For the naturalist, biological function is “not a human evaluative choice; it is nature’s “choice”” (Hausman 2012: 520).

Perhaps more tricky for Boorse is the concept of normal functioning. Boorse’s understanding of normality is statistical. Consequently, to tell if an individual is functioning normally, it is necessary to compare that individual to some standard of functioning that is deemed typical – which Boorse refers to as the “species design” (1977). Boorse argues that it is possible to produce this standard in a value neutral way by making a statistical abstraction of typical functioning from a population of organisms who should function in the same way as the individual in question (1997: 32). Boorse refers to this population of organisms as a “reference class”, specifically referring to organisms of the same species, age and sex as the individual in question (1977).

Several philosophers have argued that, because of the tremendous variability found amongst members of a species, the designation of one set of characteristics rather than another as the species design is an evaluative decision that people make (Engelhardt 1976: 263-264; van der Steen and Thung 1988: 86; see also Boorse 1997: 32; 2014: 697-699). In other words, such philosophers argue that the notion of species design is invented, not discovered. Boorse disagrees, arguing that species (and in particular humans) are sufficiently similar to support his notion of species design. “On all but evolutionary time scales,
biological designs have a massive constancy vigorously maintained by normalizing selection. It is this short-term constancy on which theory and practice of medicine rely” (Boorse 1977: 557). Boorse argues that his position “better fits contemporary biology, which regards species (at least as defined by Mayr’s “biological species concept”) as real and crucial units with describable characteristics” (1997: 39). Boorse thus argues that his notion of species design is discovered – we have no choice in the matter. If it was invented, and we could choose what species designs were, Boorse holds that the claims made in textbooks of human physiology would necessarily be “tissues of lies” (1997: 33, 38).

Others have argued that the subdivision of species groups by age and sex to produce particular reference classes is similarly evaluative (Kingma 2007; DeVito 2000). For instance, Kingma (2007: 128) argues that Boorse is wrong to claim that the designation of an appropriate reference class is “determined solely by empirical facts and does not depend on evaluative judgement”. Kingma therefore argues that we have some choice over which reference classes to use, and that reference classes are not entirely discovered. Kingma says that “although facts determine both that I am a woman and I am short-sighted, there are no empirical facts that determine that ‘woman’ is an appropriate reference class, and ‘short-sighted people’ is not” (2007: 131). As Kingma believes there are other possible candidate reference classes (such as short sighted people), she believes there are alternative accounts of function and dysfunction (in short sighted people, short sightedness is the normal function of the eye), and alternative accounts of health and disease (short sightedness is not a disease).

Boorse has recently denied this. “I [Boorse] continue to maintain that value-laden choice of a health concept by anyone, me or medicine, is a mythical process”
(2014: 694). He provides two grounds for this claim. Firstly, he claims that he has no choice but to adopt this theory of health because this is the theory of health that medics in fact employ (Boorse 2014: 693). Even if this were accepted as true, this is not an adequate defence of the naturalism to which Boorse is committed. As I have shown, Boorse is committed to a naturalism in which disease status is an “objective” matter, which can be read directly from the “biological facts of nature” (1997: 4). As such, Boorse is claiming that medics have no choice but to adopt the theory of health that he describes if they wish to represent the natural world faithfully. Claiming that philosophers doing descriptive work have no choice but to accept his theory of health does nothing to support this latter claim.

Secondly, Boorse (2014) denies that any of the terms he uses to define his concept of health (i.e. “concepts of statistical normality, species, age, sex, and causation”) are value-laden. He thus maintains that someone’s age and sex etc. can be determined without claiming that the person in question is good or bad in any way; and he claims that none of his critics (including Kingma) say otherwise (2014: 693). According to Boorse, “[a] correct definition of concept H in terms of concepts C₁, C₂, … Cₙ is value-laden precisely if one of the Cᵢ is value-laden: that is if a judgement of the form “x is C₁” is a value judgement” (2014: 693). As none of the terms he uses to define his concept of health are value-laden, Boorse argues that his concept of health is not value-laden. Contrary to this, Kingma has claimed that even if a concept of health can be described in value-free terms this does not show that the concept is value-free (2013: 40-41). Regardless of whether it is Kingma or Boorse who gives the correct analysis of what it means to be value-free, Boorse’s defence does not support the claim that medics have no choice but to adopt his notion of health. Even if a patient’s sex or age can be
determined without saying the patient is good or bad, this does not show that medics have no choice but to employ the concepts of age and sex in their concept of health and disease. Kingma’s (2007) reference class objection remains unanswered precisely because Boorse has failed to address the question of whether or not medics have a choice in this matter. The claim that medics have no choice but to accept certain biological facts if they are to faithfully represent the natural world is central to this philosophical naturalism.

It is not only philosophers like Boorse who adopt a naturalist view of health and disease. Naturalism has also been identified amongst historians of medicine. Historians of medicine sometimes make philosophical arguments about what diseases are when reflecting on how to write the history of medicine. Historiographical positions have been identified that have a lot in common with the naturalism put forward by philosophers of medicine. For instance, Adrian Wilson has identified what he calls the “naturalist-realist” approach to history, which “excludes disease concepts from historical investigation, because it takes modern disease concepts as the mirror of natural reality” (Wilson 2000: 276). As faithful representations of the natural world, diseases are natural things the existence of which we have no choice but to accept. On this view, historians have the right to interpret the actions of doctors and patients in the past using these modern disease categories. When approached like this, the history of disease becomes the history of how different societies have reacted to a transhistorical and stable disease entity (Wilson 2000: 282). The two-layer view of disease is preserved in the naturalist-realist approach to historiography, as there is a timeless biological fact about what disease any patient in history had (the first layer), which different societies choose to react to differently (the second layer).
Wilson (2000) argues against the naturalist-realist approach to history, which he says is surprisingly\(^{41}\) common. Claudia Stein has also claimed that there is an increasing tendency amongst historians to treat diseases as “transhistorical and stable” entities (2014: 56), and that historians may be “unwitting” culprits in this historiographical crime (2014: 54). Indeed, the only explicit endorsement of this way of writing history I can find comes from David Wootton (2007: 2-3, 289-297), who has argued that it is ridiculous to maintain that medicine is not making progress, and that this progress is the result of our knowledge of disease becoming an ever more precise mirror of nature. On this view we have no choice but to accept modern disease concepts as the truth (or at least closer to the truth) than those of our predecessors, if we wish to avoid self-delusion. Even if it is not frequently endorsed explicitly, the naturalist-realist approach to history is reportedly common, and increasing in popularity.

So naturalist arguments depend on the ability to show that we have no choice but to accept first-layer concepts of health and disease as entirely determined by the way the world is. There are no choices to be made when it comes to the biological aspects of disease. However, naturalists are not alone in making this commitment. Many weak normativists also accept that there are no choices to be made when it comes to biological knowledge, and endorse some form of two-layered account of health and disease.

2 – Weak normativist
According to Khushf (2007) weak normativists, while arguing that value judgements play a central role in medical concepts, still think that it is possible to

\(^{41}\) Wilson claims that this is surprising because a common narrative in contemporary historiography of medicine is that historians have moved away from telling linear, whiggish stories of the development of medical knowledge that culminate in our present day understanding as an accurate mirror of nature (see Gabbay 1989; Webster 1976; Waddington 2011). As discussed in the introduction, this perhaps does not reflect current trends in medical historiography.
distinguish the factual from the evaluative components of these concepts (2007: 24). “While they see values as integral to health concepts, weak normativists share with naturalists a confidence in our ability to tease out facts from values” (Khushf 2007: 24). Prominent examples of weak normativists include Jerome Wakefield (1992); Robert Brown (1977); Lawrie Reznek (1987); Lennart Nordenfelt (1995); Bill Fulford (2004). Of these, Wakefield (1992) and Nordenfelt (1995) have been identified as the most influential (Lemoine 2013: 11; Schramme 2007: 11. See Boorse 1997: 5 for the claim that Wakefield and Brown are normativists).

Wakefield defines mental disorder as “harmful dysfunction, wherein harmful is a value term based on social norms, and dysfunction is a scientific term referring to the failure of a mental mechanism to perform a natural function for which it was designed by evolution” (1992: 373). Wakefield thus strictly separates a discovered, value-free first-layer medical concept (dysfunction) from an invented, value-laden second layer concept (harmful). Wakefield therefore explicitly endorses the two-layer model, and is in close agreement with Boorse in this respect (Boorse 1997: 5) 42.

Nordenfelt (1995) and Fulford (2004) are slightly different to Wakefield in that they adopt the “Reverse Theory of Disease and Illness” (Nordenfelt 2007: 8). This holds that our concept of disease is derived from the concept of illness. “The primary focus of attention is thus the illness – the problem as perceived normally by the patient” (Nordenfelt 2007: 8). On this view, people consider themselves to be “ill” if they find themselves unable to realize all of their “vital goals”, and thus not to be fully “healthy” (Nordenfelt 2013: 24). This illness is then investigated,

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42 Brown (1977: 37) also endorses the harmful dysfunction view of disease, and therefore the two-layered model.
and if it turns out that “at least one organ or other bodily part” is involved in producing the illness, the patient is said to have a disease. The disease is identical with the state or process involved in the illness (Nordenfelt 2013: 24). This is the “reverse” of Boorse’s “disease-plus” view, which holds that we can determine what disease is in the biological sciences without thinking about values, and then determine what illness is by considering whether or not we dislike the disease.

This has important implications for who we consider diseased. Notice, that by making illness primary, the goals which are deemed to be pursued by the living organism are expanded beyond survival and reproduction. Consequently, more patients are recognised as diseased in Nordenfelt’s “holistic theory of health” than in Boorse’s account (Nordenfelt 2013: 23). “[S]ome diseases picked out by the holistic criteria will not be counted as diseases by the Boorsian ones. A person may be in pain and disabled by internal bodily causes without this condition lowering the probability of the person’s survival” (Nordenfelt 2007: 8-9). Consequently, there are significant differences in how some naturalists and normativists apply their concepts of health and disease.43

Even so, both Nordenfelt (2013) and Fulford (2004) still adopt a two-layer account of medical concepts. As identified by Khushf, Nordenfelt does not question the possibility of purely empirical investigation of whether or not “at least one organ or other bodily part” is involved in producing a patient’s illness, and he does not deeply challenge the fact/value distinction (Khushf 2007: 23; see Nordenfelt

43 Kingma (2013: 37) has argued that there are no important differences between most naturalist and normativist accounts because these all employ a two-layered view. Even though this is true, I cannot agree with Kingma that naturalists and normativists have been arguing over nothing, because the differences between naturalists and normativists do make a difference in practice to who is considered diseased.
1995: 147). Choices need to be made to determine which problems are going to become the subject of biological investigation, but those choices have no effect on the results of that investigation. Nordenfelt will admit to a sociology of biology, but not to a sociology of biological knowledge.

In Fulford’s case, a two-layer account of medical concepts is endorsed in the first principle of “values-based medicine” (Fulford’s value oriented counterpart to evidence-based medicine): the “two feet” principle (Fulford 2004: 206-212). This principle is that “[all] decisions stand on two feet, on values as well as facts, including decisions about diagnosis” (Fulford 2004: 208). Here Fulford explicitly endorses a “fact + value conceptual framework for diagnosis” (2004: 211). Each foot of this principle represents a layer of a two-layered account of medical concepts, in which discovered facts and invented values are separable.44

So, proponents of the reverse view hold that there are first layer medical concepts that depend upon choices made by people about what counts as a problem, and are therefore invented. Biological knowledge is then added in a second layer to determine which of these problems involves the human body. As there is no choice about which problems qualify as biological in the second layer, these concepts are entirely discovered. So a two-layer model of medical concepts is found amongst a wide range of influential naturalists and normativists. But, this does not just apply to weak normativists. The two-layered model can be found amongst strong normativists as well.

44 Lawrie Reznek (1987: 212) also claims that for a condition to be a disease it must first be judged harmful, and then found to have some bodily or physiological basis. Reznek therefore endorses the reverse view of disease concepts, like Nordenfelt and Fulford.
3 – Strong normativist
As stated at the beginning of this section, Khushf (2007) augments Boorse’s (1975) category of strong normativist. Khushf (2007) argues that there are at least two different sorts of strong normativist: strong normativists of the first and second type. What these positions have in common is that they believe factual and value-laden elements of disease concepts cannot be separated from each other. Consequently, at first sight it would seem that strong normativists reject the two-layered account of medical concepts, and see the invented and the discovered as indistinguishable. I argue here that this is not always the case.

Strong normativists of the first type “give priority to the socially determined features of health concepts, and downplay the descriptive, scientific aspects of medicine” (Khushf 2007: 24). On this view, medical concepts are understood as invented things, and the role of nature in limiting our choice of medical concepts is dismissed or ignored. Strong normativists of the second type are less radical than those of the first type. “While they think we cannot neatly tease out the conventional and invariant aspects of disease, and they also are attentive to how evaluative considerations configure health concepts, they also recognise that health phenomena are not fully plastic” (Khushf 2007: 24). As they are the less radical of the two sorts of strong normativist, I will consider them first.

3.1 – Strong normativist of the second type
Khushf (2007) does not specify which philosophers he would consider as strong normativists of the second sort. I suggest Joseph Margolis (1976) should certainly be considered a philosopher of this sort, as he argues that it is impossible to “tease out the conventional and invariant aspects” of medical concepts. According to Margolis (1976):

“[T]he ascription of “natural” functions to organisms…..cannot be straightforwardly made on the basis of some empirical inspection of the
essential nature of such creatures. This is not to lose the notion of natural function but only to question in an important way the sense in which natural functions may be said to be discovered by an exercise of medical science or any other relevant science” (Margolis 1976: 249. Original emphasis).

If the natural functions belonging to first layer medical concepts “cannot be straightforwardly made on the basis of some empirical inspection”, Margolis (1976) surely must reject any two-layered account of those concepts. Whilst this is certainly true in principle, Margolis believes this insight has very little significance in practice. Even though Margoliss does argue that natural functions are not simply “discovered”, he does still maintain that it is reasonable to speak of “natural functions”, and even that what these functions are is often quite obvious. This is because Margolis holds that the values which affect our notion of natural function are prudential values, or values shared by the great majority of people in society (1976: 251). For Margolis, these prudential values include the “avoidance of death, prolongation of life, restriction of pain, gratification of desires, insuring the security of person and property and associates, and the like” (1976: 251). Medicine, like law, has “provided an institutionally determinate rule for managing a portion of our prudential interests” (Margolis 1976: 252). Once these values are in place however, what the natural functions are becomes reasonably obvious:

“The apparent obviousness of the natural functions of the organs and the bodily processes can only be understood in terms of the conservatism of our prudential interests, such that the shifting of ideologies leaves relatively intact – but only relatively – the detailed schedules of bodily disease and illness. What we see is that it is entirely fair to insist on the natural functions of the various parts of our system” (Margolis 1976: 253).

Although Margolis disagrees with philosophers who argue that natural functions are objectively determined, it is still fair to talk about natural functions as if they had been discovered, as opposed to invented. This is because the values that
could affect “the detailed schedules of bodily disease and illness” are prudential and agreed upon. Consequently, even though human choices and value judgements are involved in the production of knowledge about natural function, we do not need to consider these choices closely, as people will never make alternative choices. Even though the distinction between the invented and the discovered has in principle collapsed, in practice the two-layer model is preserved.

Other philosophers also hold that value judgements and human choices affect first layer medical concepts, and Elselijn Kingma has been a very vocal advocate of such positions in recent years (2013: 54). Drawing on philosophical work on the mineral “jade”, Kingma argues in favour of pluralism with respect to the biological aspects of the concepts of health and disease (Kingma 2013: 47-48; Hacking 2007a; LaPorte 2004; Engelhardt 1985a). According to Kingma, “our concept of “jade” was not inevitable, but the result of social and historical processes” (2013: 48). Kingma (2013: 50) argues that just as it is possible to classify minerals in different ways and produce different geologies, it is possible to classify people in different ways and produce different biologies.

Kingma (2013) argues against the view that disease concepts come in two distinct layers, which strictly separate that which is biological and fixed from that which is value laden and historically contingent. Instead, she argues that naturalist and normativist considerations could interact (2013: 54) to produce an account of biological knowledge that is historically contingent, but which does not “reductively define health and disease in evaluative terms and tend to lack any formal appeal to the biological sciences” (Kingma 2013: 41). Kingma contrasts this potential account with a reductive and unidirectional normativism which accepts biological facts as fixed and given, but allows value laden choices to
dictate which aspects of biology should count as relevant for the definition of disease:

“Importantly, and in contrast to the reductive normativist picture that suggests that a change in value judgement unidirectionally changes what is a disease, these values, attitudes, social developments and the concept of disease interact, change and develop in conjunction. All of these processes have affected many aspects of our world, and only one of these is the naturalistic account of biology, sexuality, disease and/or dysfunction that we have come to adopt” (Kingma 2013: 53-54).

I should point out that these positions are not special because they are pluralist with respect to how patients are classified. Most of the normativists discussed thus far advocate some form of classificatory pluralism, because they argue that people have to make choices about what biological states to value and disvalue. What makes these stronger normativist positions different is the view that they hold that researchers have choices to make about the basic facts of physiology, such as how the heart’s action contributes to the patient’s survival.

At first glance, then, normativists like Kingma are advocates of the sort of epistemological position that I am seeking; as they argue that even the basic biological facts about disease are affected by the choices of researchers, and that this has the potential to lead to significant classificatory pluralism. The devil is, however, in the detail. The accounts of how choices affect the study of human biology that are offered by these philosophers are not applicable to my case study of heart failure.

Kingma suggests that naturalism and normativism can be integrated in a particular way, so that the strengths of each position can be preserved whilst addressing their weaknesses (2013: 37). She suggests this can be done by recognising that it is possible to offer a naturalistic description of the biological condition of the patient without using evaluative language, whilst at the same time
recognising that these descriptions are the contingent result of social and historical processes (2013: 51-53). As the only ‘strength’ of naturalism that is conserved by Kingma is the absence of evaluative terminology (such as ‘good’ and ‘bad’), this attempt at integrating naturalism and normativism is unsuitable for my purposes, as it does not show how knowledge of disease is constrained by something other than the historically contingent decisions of researchers. When described in the abstract, Kingma’s attempt to integrate naturalism and normativism results in a form of strong normativism of the first type, which I discuss below.

In more recent work, Kingma expands in more concrete terms on how descriptions of a patient’s disease status can reflect evaluative choices made by researchers without using evaluative language in these descriptions. Kingma (2014: 599-602) summarises the choices that allegedly have to be made when investigating human biology. These include choices about an appropriate reference class, the goals of human beings, and about what counts as a standard environment. If the reference class is not taken to be representative of people of the same age and sex at the patient, as Boorse claims, then all sorts of physiological states can be rendered normal by comparing the patient to a reference class in the same physiological state (see the short sightedness example from earlier). If the goals of an individual are not survival and reproduction, as Boorse says they are, then the presence a condition that will kill this patient in a short space of time need not be seen as the presence of biological malfunction. If a condition reduces life expectancy in some environments, but extends it in others (e.g. sickle cell anaemia), and biological function is associated with survival, then researchers have to choose the environment that will serve as the “reference environment” (Kingma 2014: 600).
As discussed earlier, Kingma (2007) has argued forcefully that there is no value free way to determine what should serve as an appropriate reference class in the assessment of the functional status of a patient's organs. Using Kingma’s example of homosexuality, it may be correct to point out that it is possible to argue that homosexuality is not a disease because homosexuals make up their own reference class. However, as Boorse points out using Kingma’s example, “in the 1973 debate over homosexuality, APA members did not divide into those who opposed and those who defended sexual preference as a basic factor in the reference class” (Boorse 2014: 695). The same could be said for the examples of short sightedness (Kingma 2007), Down’s Syndrome, or long term abusers of alcohol (Kingma 2014: 601). Crucially, the same can be said for my case study of heart failure. Researchers investigating heart failure certainly did compare groups of patients they took to be diseased with a reference class of patients they took to be healthy. However, this reference class was not taken to represent an arbitrarily chosen sub-population of humans, but rather to represent all healthy humans as a whole. This is the case in every stage of my longue durée history. Consequently, I cannot explain the historical contingency I see in my case study by appealing to changes to what researchers considered to be an appropriate reference class.

Neither can I explain the contingency I see in my longue durée history by appealing to changing environments or to the apical goals of individual patients. At no point in my history does a patient’s becoming breathless on exertion and accumulating fluid promote their survival. In every stage doctors were focused on explaining and preventing the suffering and death of patients. Even though Kingma argues that the basic biological facts about disease should be
problematised, she does not provide the epistemic account for which I am searching.

Kingma (2013) says that she draws from Ian Hacking’s work in her account of social construction. Hacking himself offers an account of how something can at once be socially constructed and yet real (1999: 119). Hacking’s views on this topic are expressed as part of his discussion of “making up people”, and of “looping effects” between the classifications of people and the people who are classified (Hacking 2007b). To discuss Hacking’s views, I will focus on two different sorts of case study that Hacking makes use of; which are represented by his discussions of homosexuality and multiple personality disorder on the one hand, and autism on the other.

Hacking argues that homosexuality and multiple personality disorder did not exist before the twentieth century:

“The homosexual’ is a kind of person that exists only in particular historical and social setting, for example now, but not in ancient Athens. The homosexual ‘as a kind of person’ did not exist then, although there were plenty of same-sex acts with complex codes about which acts were right and which were wrong” (Hacking 2007b: 295).

Although behaviours that people now would recognise as homosexual were expressed well before the twentieth century, the people who expressed those behaviours were not put in a group together before the twentieth century. Homosexuality was not “a way of being” a person until quite recently (Hacking 2007b: 295). Consequently, homosexuals literally did not exist before the twentieth century, when this kind of person was made up for the first time.

Hacking makes a similar analysis of patients with multiple personality disorder, and argues that:
"In 1955 this was not a way to be a person, people did not experience themselves in this way, they did not interact with their friends, their families, their employers, their counsellors, in this way; but in 1985 this was a way to be a person, to live in society" (Hacking 2007b: 299).

Hacking also makes the stronger claim about whether multiple personality disorder existed before it was recognised, which he refers to as claim “A”. “There were no multiple personalities in 1955; there were many in 1985” (Hacking 2007b: 299).

Hacking argues that this is not the case for autism. Referring to claim A about multiple personality, Hacking says:

“[I]n my opinion, A is true for multiple personalities: it is a transient mental illness after all. Multiple personality advocates will have disagreed with me. My opinion about A for high-functioning autism is quite different: it is absolutely false. It is almost as absurd as saying that infantile autism did not exist before 1943, when Kanner introduced the name” (Hacking 2007b: 303).

According to Hacking, autism “is almost certainly some combination of neurological, biological and genetic abnormality” that did exist before it was recognised (2007: 301). He designates this pathology “P” (1999: 119). As P exists whether researchers recognise that it exists or not, Hacking argues that autism existed before it was recognised as such. P is real, whereas ideas about autism are constructed. Autism, Hacking says, can be at once socially constructed and real, so long as different senses of autism are recognised:

“Now for the bottom line. Someone writes a paper titled “The Social Construction of Childhood Autism.” The author could perfectly well maintain (a) there is probably a definite unknown neuropathology P that is the cause of prototypical and most other examples of what we now call childhood autism; (b) the idea of childhood autism is a social construct that interacts not only with therapists and psychiatrists in their treatments, but also interacts with autistic children themselves, who find the current mode of being autistic a way for themselves to be” (Hacking 1999: 121).
Hacking adds a further layer to his thesis about the possibility of being constructed and real by discussing “looping effects” (1999: 123). The act of classifying affects the people so classified, because these people know that they have been classified, react, and thus change their behaviour. In multiple personality disorder, the explanations and expectations of classifiers changed the characteristics of the patients they classified:

“The psychiatrists cast around for causes, and created a primitive, easily understood pseudo-Freudian aetiology of early sexual abuse, coupled with repressed memories. Knowing this was the cause, the patients obligingly retrieved the memories. More than that: this became a way to be a person” (Hacking 2007b: 296).

So the characteristics of this kind of person were affected by act of identification. Hacking argues that the same sort of thing happened in autism, and one of the results of this process was the production of the “high-functioning autistic” as a kind of person (2007b: 304). Hacking even suggests that looping effects can alter the biology of the people so classified, which he calls “biolooping” (1999: 123). Thus, the kind under investigation is a “moving target”, a dynamic rather than a static kind (2007b: 312). The social act of classification can affect the kind of person under investigation, and possibly even biological characteristics like P. Hacking argues than none of this affects the objectivity of P:

“Or we might find that most obesity and all autism is linked to a certain organisation of genetic anomalies. It is important to know. We try to find out by using all seven listed scientific engines. I observe that we tend to think of them as directed at fixed targets. I suggest that the engines modify the targets. This in no way queries their objectivity” (Hacking 2007b: 312).

From my point of view, this analysis of how something can be objective and real at once is disappointing. What makes autism real is P, and knowledge of P is timelessly true, regardless of whether researchers know it or not. According to
Hacking, P is entirely discovered, and not at all invented. The kind may change over time for a variety of reasons, including looping effects (in the case of human kinds), or evolutionary change (in the case of species of animal or microbe). Even so, there are timeless facts about these moving targets, which are entirely discovered. Hacking’s views on autism are a form of naturalism.

Hacking does not apply his analysis of how something can be both constructed and real to homosexuality or multiple personality disorder, because these lack something timeless that is equivalent to P. This disappoints me, as it is precisely “transient” ways of being a person (perhaps like homosexuality and multiple personality disorder) that I want to be able to say are both invented and discovered. I want to be able to do this without appealing to timeless things like P. Hacking’s discussion of looping effects does not provide a way for me to do this. It is not clear what distinguishes Hacking’s views of homosexuality and multiple personality disorder from accounts that explicitly endorse the view that disease categories are entirely invented (see Engelhardt’s (1974) views on masturbation below). Rather than showing how knowledge of disease is invented and discovered, Hacking argues that knowledge of some diseases (multiple personality disorder) is invented, and that knowledge of others (autism) is discovered.

Furthermore, I do not see looping effects as important driver of change in my longue durée history. I suspect looping effects do play a role to which I have not been sufficiently sensitive. For instance, James Mackenzie (one of the historical actors I discuss) did report that telling patients that they might have a heart problem affected patient’s health even in cases where no such problem existed. In one case he recalled that “[i]t was evident that the whole train of symptoms was the outcome of fear produced by the vague prognosis” (Mackenzie 1919: 147).
Nevertheless, the historical story I tell about changes to a human kind (patients in heart failure) could also be told about changes to kinds that are indifferent to how they are classified by people (e.g. horses in heart failure). Hacking argues that looping affects will not take place in these indifferent cases, as these patients are not aware of their classification, and are not affected in the same way as people are (1999: 107). I do not see that Hacking’s analysis of looping effects will assist my efforts to explain how the development of medical knowledge about heart failure is at one invented and discovered. I now turn to strong normativists of the first type to see if their epistemologies fare any better.

3.3 – Strong normativist of the first type
According to Khushf, strong normativists of the first type “downplay the descriptive, scientific aspects of medicine” (2007: 24). These are the normativists who, according to Kingma, define medical concepts in terms “that lack any form of appeal to the biological sciences” (2013: 41). In Boorse’s original classification, strong normativism was the view that health judgements are pure evaluations without descriptive meaning” (Boorse 1975: 51). On this view, “to call a condition unhealthy is only to express disapproval of it. In other words – to collapse a few ethical distinctions – for a condition to be unhealthy it is necessary and sufficient that it be bad” (Boorse 1975: 51-52). On this view, nothing about disease is discovered. The appropriate application of the term “disease” is whatever people decide it should be.

Kingma (2013) cites Rachel Cooper (2002) as the clearest example of such a philosopher (Kingma 2013: 41, footnote 5), and Cooper (2002) does argue that the concept of disease does not have a biological basis. According to Cooper “[n]o biological account of disease can be provided because this class of conditions is by its nature anthropocentric and corresponds to no natural class of
conditions in the world” (2002: 271). Instead, Cooper (2002: 263) argues that for a condition to be a disease, it has to be bad for the patient, the patient should be unlucky to have it, and it has to be potentially medically treatable. Nevertheless, it is still possible that a two-layered account is used in Cooper’s (2002) work, and it is possible to see this in the horticultural analogy she uses:

“A plant is only a weed if it is not wanted. Thus a daisy can be a weed in one garden but a flower in another, depending on whether or not it is a good thing in a particular garden” (Cooper 2002: 274).

In this analogy, a daisy (which stands for a condition) is evaluated to determine whether or not it is a weed (which stands for a disease). But how the daisy in question acquired the status of being a daisy is not problematized. Cooper just treats conditions as givens. For instance, Cooper says things like “whether a condition is a disease depends upon whether it is a bad thing for the ‘sufferer’ I mean that disease-status depends on how the condition in and of itself [my emphasis] is evaluated”, and “the same biological condition may produce different experiences in different people” (Cooper 2002: 274-275). Such statements can be read to mean that it is possible to know what a condition is without attending to their social ramifications, and to know objectively which patients have the “same biological condition”. Furthermore, Cooper appeals to expertise in “human physiology and biology and other sciences” to determine which patients count as “potentially medically treatable”; and she distinguishes between conditions that it is “technically feasible” to treat and those that are “socially feasible” to treat (2002: 278). Cooper treats “conditions” as unproblematic, discovered entities; many of which we have no choice but to adopt because of the results of objective biological investigation.
The main point of difference from other positions described above is that Cooper (2002) does not require a condition to be biological, as “there might be some mental diseases where there is nothing wrong with the patient's brain” (Cooper 2002: 277). To extend Cooper’s metaphor, non-biological things found in gardens, such as water features or garden gnomes, are candidate “weeds” as well. If such things are judged to be bad, and if the homeowners are unlucky to have them in their garden, and if they are things which gardeners typically deal with, then these things are considered weeds on Cooper’s view. But none of these candidate weeds are problematized either. If this is the case, Cooper (2002) still uses a two-layered account of medical concepts. The first layer is comprised of unproblematic conditions, which may or may not be biological. As the biological conditions treated as givens, it seems that we have no choice but to accept them as part of the natural world. The first layer conditions are therefore discovered. In the second layer we make some evaluation of these conditions, and therefore introduce an invented element. Even for allegedly very strong normativists like Cooper (2002), the discovered and the invented are kept separate.

Many of the various naturalist and normativist positions we have looked at thus far have preserved some form of two-layered account of disease concepts, and have kept the discovered and invented separate. There are some strong normativist positions, however, that reject the two-layered account; but they do so by embracing the invented alone. This is particularly the case for authors who make use of historical case studies in their work.

3.3 – Strong normativism and the appeal to history
In addition to making conceptual analyses of how the concepts of health and disease are applied in the present day, some philosophers have appealed to
historical case studies to show how human value judgements have shaped these concepts. The presentation of historical case studies often reveals the very strongest forms of normativism available. Such historical case studies are designed to show that in the past, where values were different, people made different choices and developed different disease categories and biologies to those in use today (Boorse 1997: 72). This is not to say that values act to introduce biases and promote the acceptance of erroneous disease categories. Rather, philosophers who make use of such historical examples see a perfectly legitimate (or even necessary) role for value judgements and choices in producing different concepts of disease. “The variations are not due to mere fallacies of scientific method, but involve a basic dependence of the logic of scientific discovery and explanation upon prior evaluations of reality” (Engelhardt 1974: 247).

Prominent amongst such philosophers is Tristram Engelhardt, who makes use of examples such as masturbation, drapetomania and dysaesthesia aethiopis (Engelhardt 1974: 247; 1976: 262 See also Boorse 1997). Engelhardt’s (1974) case study of masturbation is his best developed example of “the value laden nature of science in general and of medicine in particular” (1974: 234). Engelhardt argues that in 18th and 19th century America masturbation was widely believed to cause a wide variety of very serious conditions; including epilepsy, blindness, deafness, insanity, and rickets (1974: 236-237). The symptoms and signs of these conditions were linked aetio logically to masturbation in a variety of ways, including “disordered nerve tone due to excess and/or unnatural sexual excitation” (Engelhardt 1974: 243). Patients were treated with procedures as radical as vasectomy, castration, clitoroidectomy, and burning the genitals with acid to make masturbation painful (Engelhardt 1974: 244). In many instances
these treatments were reportedly successful. Engelhardt argues that the nineteenth century saw the development of an “authentic disease of masturbation” (1974: 243).

The view that past societies could have used different disease categories to the ones in use in more modern times, and yet for these still to be “authentic” diseases, is in-keeping with recent historiographical trends. In the latter decades of the twentieth century, historians have raised concerns about the telling of linear, or “whiggish”, stories of scientific and medical progress (Webster 1976; Gabbay 1989; Waddington 2011). Such historians have argued that scientific and medical knowledge is not steadily developing from a past position of near total ignorance to a modern one of near perfect knowledge. On this view, modern scientific and medical knowledge is not timelessly correct, but is instead seen as a “human construct” (Wilson 2000: 282). In the history of medicine, this view amounts to a rejection of the naturalist-realist view described above (Wilson 2000: 276; see also Cunningham 2002: 20). Inspired by the work of Ludwick Fleck (1979), historians of medicine such as Andrew Cunningham (2002) and Adrian Wilson (2000) have argued that “disease concepts are always social products”, and are not simply “‘out there’ in Nature” (Cunningham 2002: 14-15). Even though present day doctors and philosophers may believe that they have captured trans-historical truths about disease, “in fact this is just our society’s way of thinking: true for us and our world, but not necessarily true for other societies and other times”45 (Cunningham 2002: 15).

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45 Wilson (2000) describes this approach to writing history, where diseases are treated as produced in a particular social and historical context, as the “historicist-conceptualist” approach, whereas Stein refers to such an approach as “constructionist” (Stein 2014: 54). Wootton (2007: 15) refers to it as the “worm’s eye view”, as in this style of writing history there is a tremendous focus on the detailed exploration of a particular social and historical context. Wootton contrasts this with “bird’s eye view” longue durée history, which Wootton argues is necessarily anachronistic.
Cunningham (2002) in particular has gone even further than this. Addressing the knotty problem of how to write the history of disease in past societies and cultures, Cunningham (2002) argues that the only way to avoid the pitfalls of whiggish historiography is to move away from studying disease concepts and instead focus on historical activities and practices. Cunningham advocates turning towards “how diagnosis happens”, by which he means looking at “people thinking and acting in particular cultures” (Cunningham 2002: 16). According to Cunningham:

“By making how diagnosis happens central to our historical investigations, we are using the only sure thing we have, the only thing which we can rely on. For it is by the act of diagnosis that disease identity is given or established. The operations that humans perform in making diagnosis are not just the key to disease identity, but the source of disease identity. The only identity that disease has is the operational identity” (Cunningham 2002: 16).

By making disease identity identical with how diagnosis happens (i.e. with how people think and act), Cunningham (2002) is claiming that there is nothing else to disease beyond what historical actors think and do (2002: 20). Cunningham is quite clear about what he sees as the far reaching ontological consequences of this view. He emphasises one such consequence in particular, which is that:

“[Y]ou die of what your doctor says you die of. Your cause-of-death certificate is not negotiable. While this might seem a reasonable thing to say about people dying today, I want to argue that this also applies to everyone in the past. They died of what their doctors said they died of. Their cause-of-death certificates (as it were, for of course such certificates are very modern and very western) are equally not negotiable, neither by the modern medic, whether clinician, pathologist, epidemiologist or psychiatrist, nor by the modern historian” (Cunningham 2002: 17).
This view represents a very strong form of normativism indeed. If you die of what your doctor says you die of, disease is an entirely invented thing. The identity of disease is only constrained by what people choose to say and do. How the natural world constrains our choice of disease identities is treated as an insoluble problem, the “Gordian knot of disease history” (Cunningham 2002: 16), through which we must cut if we are to study disease in different times and cultures. As an entirely invented thing, disease concepts (or as Cunningham would have it disease identities) only have one layer. This, then, is the only position in the naturalist versus normativist debate that does not use a two-layered account that we have managed to find as yet. But even here, the discovered and the invented are not blended together. Instead, disease is understood as an entirely invented thing.

This sort of extreme relativist epistemology makes it impossible for doctors to be wrong, or to make mistakes, or to improve their practice. Adopting such a position is to concede that any method of managing (including surgically removing parts of a patient’s genitals in the hope that this would cure their rickets) is as good as any other. To people concerned about the welfare of patients, such epistemic views ought to be unacceptable.

Many authors have rejected this notion that some conditions and behaviours (like masturbation and drapetomania) were “authentic diseases” (Daniels 1981: 156; Reznek 1987, 4-6; Boorse 1997: 74). Reznek has argued that doctors who believed that masturbation was a disease were simply mistaken (1987: 4-6). Boorse agrees. “19th-century masturbation science consists of spectacularly

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46 Normativists who hold positions as strong as this are often referred to as “constructivists” (Murphy 2015). As this chapter reviews various naturalist and normativist positions, I will continue to use the term normativism, even in such extreme cases.
false statements. Masturbation does not damage nerve-tone, much less the spinal cord. It does not cause rickets, blindness, insanity, acne, or even moist palms. Theorists were wrong in their theories, wrong in their data, wrong about virtually everything; almost no statement listed in [Engelhardt's] essay contains even a grain of truth" (Boorse 1997: 74).

For Reznek and Boorse, historical examples like the case of masturbation not only fail to show that there are various, alternative, “authentic” versions of disease, but also reveal the danger in allowing scientific work to be biased by social interests and values (Reznek 1987: 6; Boorse 1997: 72-78). I agree that cases like Engelhardt's are not good examples of historical contingency. Even in the event that some scholars continue to maintain that these Victorian views on masturbation are medically valuable, it is still important to be able to show how it is possible for doctors to be mistaken. Engelhardt and Cunningham sometimes fail to distinguish the historically contingent from the entirely fictional, and this failure is unacceptable from an epistemic point of view. The history of heart failure provides several much better examples of historical contingency.

These concerns about the legitimacy of the Victorian diagnosis of masturbation highlights a difficulty with reducing discussions of disease categories to the discussion of what doctors say and do, as Cunningham (2002) advises. Anne-Marie Mol (2002) and Bruno Latour (2000) have made similar arguments with respect to disease ontology and human activity. Mol argues that diseases are activities, things done by people. Latour argues that a middle way between “radical whiggish and anti-whiggish metaphysics” (2000: 251) can be found through attending to the work that historical actors had to do in order to produce the knowledge they did. Sadly, these views are epistemologically unsatisfying for the same reasons Cunningham’s is. It is not enough to attend to whether
something was done. It is also important to consider whether this was a good thing to do. Attending to what is done does not allow us to distinguish good practice from bad. The nineteenth century doctors actually did diagnose patients with the disease of masturbation, and they did actually cut these patients’ genitals off in an attempt to treat these patients’ disease. This took work and effort to do. Even so, these doctors were not treating their patients, they were mutilating their patients, whether they recognised this or not. Neither Mol (2002) nor Latour (2000) provide an epistemology that tries to distinguish good practice from poor practice; and Cunningham (2002) does not either.

To be fair I must add that historians need not follow Cunningham (2002) in believing that people die of what their doctors say they die of. Despite claims to the contrary, historians of science are not often radical epistemological relativists, who believe that we completely make up our own reality (Daston 2009: 813). Rather, according to Lorraine Daston:

“Historians of science, for their part, rarely reflect on such matters. Probably most historians of science these days, if asked about an episode like the refinement of precision measuring techniques or the formulation of statistical correlations, would answer that such scientific practices are both socially constructed and real. That is, they depend crucially on the cultural resources at hand in a given context…..and they capture some aspect of the world; they work. But they are neither historically inevitable nor metaphysically true. Rather, they are contingent to a certain time and place and yet valid for certain purposes” (Daston 2009: 813).

If Daston is correct about this, historians of science by and large think that scientific knowledge is at the same time a cultural product and the result of the way the natural world is. But, historians do not care sufficiently about the way the natural world is to “reflect on such matters”. What is important to the historian is how their actors lived, and how they understood the world, which makes considering whether and how these actors might “capture some aspect of the
world” unimportant. Although she does not phrase it this way, Daston (2009) argues that this lack of interest in the natural world can be mistaken for an extreme form of relativism.

It is easy to see how historians of medicine might also align themselves with this view. Since the 1970s the range of historiographic approaches referred to as the “social history of medicine” have become prominent in the subject (Waddington 2011: 3). This new social history of medicine involved “relegating to a subordinate place any linear account of medical progress in favour of an approach which is primarily concerned with contributing to an understanding of the dynamics of any particular society” (Webster 1976; see Waddington 2011: 4). When medicine is understood as a part of a society, medicine can be studied to learn about that society. It should be noted that, on this view, the object of study is society, and not the natural world. It is not only whiggish historiography that is side-lined, the study of the natural world is as well. If the subject of interest is how the actors that make up a society lived and understood things, consideration of whether or not these actors were correct to do so becomes unimportant.

A similar tendency to diminish the importance of epistemic questions about whether historical actors were correct to accept certain ways of understanding sick people is displayed by other prominent philosophers of medicine. Ann-Marie Jutel (2017), in a recent discussion of the formation of diagnostic categories, has sought “to de-bunk the idea that diagnostic categories simply mark natural boundaries” (Jutel 2017: 166). Using examples such as overweight and female sexual interest/arousal disorder, she argues that special interest groups, such as the fitness, diet, fashion, self-help and pharmaceutical industries, were prominent actors in establishing many diagnostic categories (2017: 161-164). Jutel claims that personal profit was a major motivating factor in these processes (2017: 161,
Jutel emphasises how it is researchers that force their diagnostic categories on the world, rather than the other way around:

“When we name things as belonging to one group as opposed to another, we are imposing a structure on our environment that reflects our values, knowledge and beliefs…How we categorize says as much about who we are as it does pathophysiology” (Jutel 2017: 157).

Jutel’s (2017) work offers a warning that diagnostic categories are often designed to medicalize, stigmatize and exploit people. Diagnostic categories are used to promote the view chubby people and women with low sexual libido have serious medical problems, which need treatment with expensive gym memberships and drugs. These diagnostic categories are presented as natural, timeless and real, which Jutel fears can lead people (both doctors and patients) to accept these classifications uncritically. “Considering overweight as a disease creates a situation in which scientists/doctors stop interrogating the data” (Jutel 2017: 163).

Jutel’s efforts to highlight the dangers of uncritically reifying diagnostic categories are welcome. Even so, she offers little to no epistemic discussion of how diagnostic categories should be decided upon. Jutel claims that knowledge of pathophysiology must be taken into account when deciding upon diagnostic categories (2017: 160), but says little about what role this aspect of medical knowledge should play. Is knowledge of pathophysiology fixed and timeless? If so, then this epistemology will not serve my needs. If not, then how is this knowledge not just made up? Jutel does not say. From Jutel we learn that “[a]lthough weight often co-varies with health, and on this basis is used as a proxy, it doesn’t always co-vary, slender people who have health risks get missed, in the same way as fat people without risks are presumed to be unwell” (Jutel 2017: 163). We also learn that “[s]ome data have shown that being overweight carries less risk than being normal weight” (Jutel 2017: 163). These
comments imply that Jutel has an account of health that she has used to decide that some people have been put in the wrong diagnostic category. And yet, she does not discuss it. Indeed, she says that it is not her goal to provide such an account of what good diagnostic classifications are and about how knowledge of them can be gained:

“The point here is not to debate whether they were right or wrong in their assessment that the disease label “might have broad effects for a large portion of society for the greater good”. Rather it is to underline the social (and often conflicted) ways disease categories come to be recognized as such” (Jutel 2017: 164).

The lack of attention paid to epistemology by Jutel (2017) makes it difficult to use this work to address concerns raised about relativism and strong normativist epistemology/social constructivist epistemology. Several scholars have sought to defend strong normativism and social constructivism from the charge of being insufficiently sensitive to epistemic concerns, and to the role that the natural world plays in constraining medical knowledge. Ludmilla Jordanova (1995) has defended social constructivism from what she describes as three common misapprehensions about it – that social constructivism is concerned primarily with undercutting medicalization, that it promotes the division between internalist and externalist approaches to the study of science by supporting externalist approaches, and that it “ignores the material dimensions of life” (1995: 367-368). Her discussion of this last misapprehension is relevant here. Jordanova claims that social constructivism “is often caricatured by its critics, who impute to it the claim that diseases are not real, and who associate it with a denial that science and medicine really work” (1995: 368). Jordanova argues that social constructivists are very much concerned with the material world. She claims that social constructionism “is not a form of idealism. But it does insist that there is
room for a variety of interpretations and meanings” (1995: 368). This rebuttal may technically address the concerns of these critics (I cannot tell as Jordanova does not cite them), but I doubt it would satisfy them, and it does not satisfy me. What needs addressing is not whether social constructivism deals with material stuff, but rather whether it recognises that the “room for a variety of interpretations” of which Jordanova speaks is limited by something other than the decisions people individually or collectively make. I have provided examples of scholars who explicitly deny that there is any such constraint on knowledge. If social constructionism is to be taken seriously, its proponents must distance it from these extreme relativist positions, and arguing that social constructivists are materialists does not do this47.

Charles Rosenberg (1992) has tried to distance himself from social constructivism by speaking about “framing” disease instead. Rosenberg claimed that the term social construction “tended to overemphasize functionalist ends and the degree of arbitrariness inherent in the negotiations that result in accepted disease pictures” (1992: xiv-xv). As Adrian Wilson (2000: 281) has argued, in distancing himself from the apparent arbitrariness of social constructivism, Rosenberg moved too far back towards a naturalist-realist understanding of disease. The metaphor of a frame refers to framing a picture, rather than framing a conversation, and the picture that is framed is the timeless biological reality of a disease: “Biology, significantly often shapes the variety of choices available to

47 I should note that there is a healthy literature on the social construction of medical knowledge that is produced and curated by sociologists of medicine and scientific knowledge. It is beyond the scope of this thesis to review this literature here, but see Jutel 2009 and Brown 1995 for an overview. Many diverse ways of understanding disease are used in this literature (Brown 1995: 34). Some take knowledge of disease entities to be universal, but others do not. Even in the sociological literature, however, “[t]he main debate about constructionism centres on its relativist claims about knowledge” (McDonnell 2013: 118; see also Schilling 2005; Williams 2001; Brown 1995: 34-35; Nicolson and Mclaughlin 1988, 1987; Bury 1986). My thesis, by staking out a relativist epistemic position that does not collapse into extreme relativism, may be able to contribute to the literature in future work.
societies in framing conceptual and institutional responses to disease; tuberculosis and cholera, for example, offer different pictures to frame for society’s would-be framers” (Rosenberg 1992: xv). When disease is “framed” like this, biological aspects of disease are not problematized.

So, several apparently strong normativist positions are not necessarily the product of a denial of the possibility of studying the natural world, but rather the result of a lack of interest in the natural world. Nevertheless, if the study of disease is to be used to inform a discussion of how disease should be understood, and how disease should be diagnosed, then how attempts to “capture some aspect of the world” might be successful or unsuccessful becomes very important. In this context, the failure to “reflect on such matters” is a luxury that those who practise medicine cannot afford.

I want to emphasize that I am not arguing that all historians of science and medicine fail to attend to how the historical actors they study might be correct or incorrect. For example, Hasok Chang’s work on the history of thermometry and chemistry is very sensitive to how and whether actors were successful and unsuccessful in their work (2004; 2012a). In the history of medicine, Christopher Lawrence provides a sophisticated analysis of the relationship between coronary thrombosis and changes to the electrocardiogram, which he uses to distance himself from extreme relativist positions: “I do not wish to be read as implying that an entirely arbitrary relation exists between ECG changes and coronary thrombosis, that physicians could have picked any old change they liked and agreed that it represented infarction. When Smith and Herrick, or Pardee, or Parkinson and Bedford, or any other figures began their work, only certain selections or classifications were possible, just as it was with the clinicopathological entity itself” (Lawrence 1992: 72). Nor do I want to suggest
that historians of medicine as a monolithic block fail to problematize all the biological aspects of disease. Lawrence’s work on coronary thrombosis provides an excellent example of medical history that does just this, as does Steven Peitzman’s (2007) work on kidney disease, Keith Wailoo’s work on (1997) splenic anaemia, and Steven Epstein’s (1996) work on AIDS. I do claim, however, that finding a way for all aspects of medical knowledge to be historically at once invented and discovered still presents a significant philosophical and historiographical challenge. In this chapter I have emphasised opposing positions in this debate, as I believe this helps to clarify the historiographical and philosophical problem at hand.

Conclusion
I have reviewed several philosophical positions in the debate about concepts of health and disease. Following taxonomy of philosophical positions put forward by Boorse (1975) and extended by Khushf (2007), I have reviewed the naturalist position, weak normativist positions, and strong normativist positions of the first and second sort. The great majority of these philosophers have adopted a two-layer view of disease concepts, and hold that the first layer biological aspects of disease concepts are fully determined by the way the natural world is. On this view, we have no choice in the matter, as disease status is an entirely discovered thing.

Naturalists like Boorse (1997) explicitly adopt a two-layered view, as they distinguish between theoretical and practical medical concepts. Weak normativists like Wakefield (1992) preserve it by separating what it is to have a dysfunction, from what it is to be harmful. Even weak normativists that adopt a “reverse” theory of medical concepts recognise a role for “pure empirical research” (Nordenfelt 1995) as part of a “two feet” model that stands on one
value-laden leg and one value-free leg (Fulford 2004). Some of the second sort of strong normativist, like Margolis (1976), do argue that values are involved in first layer medical concepts. However, these values are prudential, and thus shared by all. Consequently, there is no need to attend to the value judgements and choices made in the production of first layer medical concepts, because everybody will always make the same choices. On this view, even if the first layer is not value-free in principle, it is value-free from a pragmatic point of view.

Even Cooper (2002), a strong normativists of the first sort, retains a two-layer model, because she does not problematize her notion of a “condition”. Many of these supposedly strongly opposed philosophical positions adopt a two-layered account of medical concepts. As such, they hold that at least some component part of applied medical concepts is fully determined by the way the natural world is, and that we have no choice but to accept this state of affairs. They hold that the biological aspects of disease are entirely discovered.

In contrast to this, some strong normativists (such as Engelhardt 1974) hold that diseases are entirely invented things, or at least ignore the role the natural world plays in constraining our choice of disease categories. On this view, “you die of what you doctor (or bystander) says you die of. And that’s that” (Cunningham 2002: 34). If disease status depends only on what people say and do, then disease status depends only upon the choices that people make, and not at all on the way the natural world is. As such, these strong normativists reject any two-layered account of medical concepts, and hold that medical knowledge is entirely invented. Other strong normativists of the first sort (such as Jutel 2017) do not take on the task of showing how knowledge of disease is not entirely invented.
The epistemological position that I am looking for is in-between these two poles. I agree with Kingma, who has called for the development of an epistemology that could show how discovery and invention interact in the production of medical knowledge, especially for the biological aspects of disease (2013: 53). This would require, as Daston (2009: 813) says, for medical knowledge to be “both socially constructed and real”. This review supports Kingma’s claim that “[a]mong philosophers, social constructivism is now hardly taken seriously as an approach to health and disease” (2013: 55). Kingma’s position comes closest to the one which I seek, but ultimately is not suitable for my purposes. Variation of reference class is not an important driver of change in my longue durée history.

Kingma’s (2013) suggestion, that naturalism and normativism can be integrated by recognising that value free descriptions of disease states can be socially constructed, does not show how these value free descriptions are constrained by anything other than the choices made by people in particular cultural contexts. It is not clear how this position can be distinguished from extreme relativist views. As I argued in the last chapter, the historical contingency of all aspects of medical knowledge should be taken seriously, but in doing so the pitfalls of extreme relativism must be avoided. Many historians and philosophers who advocate some form of social constructionism have slipped too easily into epistemic positions that fail to distinguish the historically contingent from the entirely fictional.

As I have not managed to find the sort of epistemology that I am looking for in contemporary philosophical discussions of health and disease, in the next two chapters I return to the history of heart failure, and use this history to develop an

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48 For Kingma a social construct is simply something that is “not fixed or inevitable, but is the contingent result of social and historical processes” (2013: 37). I understand social construction in this way.
epistemic position that can cope with medical knowledge being at once invented and discovered. From my *longue durée* history, there appears to be a recurring historical motif of unexpected observations which precipitate changes to how heart failure was understood. I explore one of these transitions between two stages in greater detail, and use insights from this history to develop my epistemological position in chapters 6 and 7. I now look in greater depth at the work of the principal actors in my first and second stages – that of James Hope and James Mackenzie.
Chapter 4 – James Hope and hospital medicine

Introduction
The purpose of this chapter is to explore in greater depth how knowledge of heart disease introduced in the first stage of my longue durée history was produced, so that the transition from the first to the second stage can be discussed from an epistemic point of view. All of the historical actors to which I refer in the first stage of this history had close connections to the Paris hospital system in early nineteenth century France. Fortunately, there is a useful and widely accepted framework for discussing the production of knowledge in the Paris hospital system. Each of these doctors has been described as a practitioner of a particular style of medical research, which developed in the early nineteenth century Paris hospital system following the French Revolution. This is referred to as “Paris medicine” (La Berge and Hannaway 1998: 3) or “hospital medicine” (Bynum 1994: 25). Corvisart is described as one of the “high priests of hospital medicine” (Bynum 1994: 26). Laennec, “the inventor of the stethoscope and probably the greatest clinician of the French school” (Bynum 1994: 37), is always listed as a prominent member of the Paris School (La Berge and Hannaway 1998: 4; Brockliss 1998: 98). Even though Hope was British, he was trained to use the stethoscope in Paris, and “Hope’s career does illustrate an English version of hospital medicine” (Bynum 1994: 50)49. Looking at these actors as practitioners of hospital medicine will help explain why they produced the knowledge of heart disease that they did in this period, and then to understand the legacy that was

49 Bynum also says that “[b]ut if hospitals in London were hardly arranged along Parisian lines, at least the doctrines of localism [i.e. disease as local lesion of an organ], clinicopathological correlation, and hospital based statistics were often embraced by London hospital élites. A good example of this tendency is that of James Hope himself, a medical graduate of Edinburgh, student of Paris, and physician of St. George’s Hospital in London” (Bynum 1994: 49).
handed down to historical actors like Mackenzie in the second stage of this history.

At the same time, looking at the historical development of knowledge of heart disease will inform a discussion of hospital medicine. Practitioners of hospital medicine were supposedly much less interested in determining what will happen to the patient in the future (prognostication), in the treatment of the patient, and in explaining the disease processes that made the patient ill. Instead, practitioners of hospital medicine were said to be focused on the description and detection of pathological anatomy (Jewson 1976; Jacyna 2006). Against this story, the history of heart disease presented here shows that practitioners of hospital medicine were very interested in prognostication and treatment (or at least management) of patients, and that they considered trying to explain how the patient became ill to be very important. And yet, the traditional account of hospital medicine is still very valuable, as this knowledge of prognosis, treatment and physiological explanation of disease emerged out of the practice of pathological anatomy.

After introducing hospital medicine as a mode of production of medical knowledge (section 1), I will describe how clinico-pathological anatomy formed the basis for what Corvisart called “pathological physiology” (section 2). I then discuss how physiological knowledge was necessary even for Hope to make correlations between lesions of the heart and heart murmurs (section 3), before going on to show that the pathological anatomy done to explain clinico-pathological correlations involved speculation and conjecture (section 4). I also argue that Hope developed prognostic and therapeutic knowledge from this physiological explanation, and that speculation and conjecture were again the main method he
used to do this (section 5). I close by discussing the reception of Hope’s ideas in the second half of the nineteenth century.

1 – Hospital medicine as a mode of production of medical knowledge

Hospital medicine is a central part of a widely employed and accepted narrative used to describe the development of western medicine in the eighteenth and nineteenth centuries, in which hospital medicine is contrasted with the earlier “bedside medicine” and the later “laboratory medicine”. This basic scheme was introduced by Erwin Ackerknecht (1967), and then adopted by many others (Waddington 1973; Jewson 1976; Hannaway and La Berge 1998; Waddington 2011). It links the mode of production of medical knowledge to the “source of patronage” of the doctor, and to the location where the knowledge was produced (Jewson 1976: 228).

This framework begins by describing the heterogeneous theories and practices of medicine in the eighteenth century as “bedside” medicine (Waddington 1973: 211-212; Jewson 227-231). Practitioners of bedside medicine are said to have been focused on the patients’ account of their symptoms, rather than on results of a physical examination or other more intrusive tests. Patients were grouped according to the syndrome (i.e. the constellation of symptoms) with which they presented, and doctors speculated about what was wrong with the patient, what treatment to offer. According to Jewson (1976: 227), two of the main characteristics of bedside medicine were “phenomenological nosology and speculative pathology”. In bedside medicine, the doctor is employed directly by the patient, often working in the patient’s own home. The patient therefore

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50 Whilst the patient was patron, the doctor was not empowered to offend the modest sensibilities of the wealthy with the intrusion of a thorough physical examination. "Notions of class, delicacy, and modesty reinforced a doctor-patient relationship in which (especially for those in the upper part of the social scale) undressing was not part of a visit from the doctor" (Bynum 1994: 33-34).
controlled the medical encounter, which was focused on the patient’s account of their symptoms. The doctor’s “occupational task”, dictated by the patient’s interests, was to provide an accurate prognosis and to treat the patient (Jewson 1976: 228).

In the Paris hospital system of the early nineteenth century, by contrast, the doctor was not employed by the patient, but rather by the state. The rise of hospital medicine is linked to the French Revolution. Following the political transformation of Parisian and French society in the late 1700s, the established system of medical education was abolished (Jacyna 2006: 39; La Berge and Hannaway 1998: 5). The need for a constant stream of competent doctors and surgeons to serve in the army led to massive investment by the fledgling state in military-medical infrastructure, and to the nationalization and secularization of French hospitals (Waddington 2011: 149; Jacyna 2006: 40-41). The state now employed doctors, and doctors now controlled the clinical encounter (Jacyna 2006: 58; Jewson 1976: 238). Patients who did not comply with doctors’ wishes would simply be asked to leave the hospital (Jacyna 2006: 55; Waddington 1973: 215-216). As doctors did not have to derive an income from their patients, the population of the hospitals of Paris was drawn from the urban and rural poor, who were less concerned about public nudity and physical contact than their more wealthy counterparts (Jacyna 2006: 56). In this hospital environment, it became possible to develop and employ new methods of physical examination (observation, palpation, percussion and auscultation) (Jacyna 2006: 55). Hospitals also gave doctors access to the unclaimed dead bodies, predominantly of the poor (Porter 2006b: 194). This in turn gave doctors and medical students access to the inside of recently deceased patients, and to the lesions to these patients’ organs. The detection of these lesions became the central diagnostic

In this period, the patient’s subjective account of their symptoms became less important, and doctors were empowered to make more intrusive physical examinations. Signs of disease (observations made by the doctor of the patient, made from the doctor’s perspective) become more important than symptoms\(^{51}\).

As this work was going on in hospitals, patients who died could routinely be examined post-mortem. “Actually, it was only in the hospital that the three pillars of the new medicine – physical examination, autopsy, and statistics – could be developed” (Ackerknecht 1967: 15). Clinico-pathological correlations between the physical signs of the patient before they died and the results of the post-mortem examination could now be made, and doctors could learn how to diagnose lesions of organs discovered post-mortem from the results of ante-mortem physical examinations. Disease became widely understood in terms of lesions of organs. “The major achievement of the Parisian School was the delineation of objective disease entities by means of correlating external symptoms with internal lesions” (Jewson 1976: 229). The efficacy of therapies was investigated by trying to detect statistical correlations between the results of therapy and groups of patients with the same lesions. Hospital medicine is famous for the widespread use of statistics to make clinical-pathological correlations (La Berge and Hannaway 1998: 4; Jacyna 2006: 41). According to Jewson (1976: 229) “[t]he four great innovations of Hospital Medicine were structural nosology, localized pathology, physical examination, and statistical

\(^{51}\) Pierre Louis, famous for his advocacy of numerical methods for evaluating traditionally accepted therapeutic claims, argued that signs were far more significant than symptoms to the diagnosis of disease (Porter 2006a: 154). As we shall see in the next chapter, Mackenzie held the opposite view.
analysis”. This use of statistics has been connected to the rise of the hospital system in Paris in this period (Waddington 2011: 149). Rather than doctors only having access to a few dozen patients from house visits to private clients or smaller hospital wards, the rise of the Paris hospital system gave doctors access to vast numbers of patients, which facilitated the systematic physical and post-mortem examinations on an unprecedented scale (Waddington 1973: 212; Waddington 2011: 112; Weisz 2001: 107). This allowed doctors to determine empirically whether they really could predict the post-mortem findings of patients from the physical examination, and bolstered the view that this new knowledge really was free of speculation (Bynum 1994: 43). “Pinel had stressed the greater objectivity of multiple observations, and the monographs of Corvisart, Bayle, Laennec were infused with confidence wrested from thousands of patients and hundreds of autopsies” (Bynum 1994: 43).

Many traditionally trusted therapies were found to be ineffective using these statistic techniques, and this encouraged a generalized scepticism about the efficacy of medical therapies amongst practitioners of hospital medicine (Waddington 1973: 217; Jewson 1976: 228; Bynum 1994: 44). As the patient no longer controlled the clinical encounter, prognostication and therapeutics are said to have become less important than they had been for practitioners of bedside medicine. In hospital medicine, doctors’ occupational task was diagnosis of these lesions and the classification of patients by the sort of lesion that they had (Jewson 1978: 228). “The patient’s interest in prognosis and therapy was eclipsed by the clinician’s concern with diagnosis and pathology” (Jewson 1976: 235).

Laboratory medicine is said to emerge in the latter half of the nineteenth century. Practitioners of laboratory medicine reportedly “asserted that observational anatomy would never explain the cause of disease”, and their attention turned to
experiment and to physiology (Jewson 1976: 230). In laboratory medicine, the person producing medical knowledge need not even come into contact with patients. Rather, these researchers experiment with animal subjects, or with tissue samples taken from a patient (Cunningham and Williams 1992: 1-13). In laboratory medicine, the patient need not be directly involved in the production of medical knowledge.

The relative lack of interest of practitioners of hospital medicine in theoretical explanation of disease has also been emphasized in recent years. Hospital medicine has been described as focused on observation, rather than theory, on the collection, description and cataloguing of cases, rather than on physiological extrapolation. “It was almost certainly the classificatory and morphological model of natural history that induced a significant part of the Paris School to adopt an essentially descriptive and morphological anatomical tissue pathology” (Keel 1998: 140). Practitioners of hospital medicine are also described as being suspicious of medical theorizing characteristic of bedside medicine, preferring instead the supposedly less theoretical and more practical mind-set of the surgeon. “The old medicine had been too much concerned with theory; the new medicine, like the old surgery, would be devoted to practice” (Bynum 1994: 28).

It is with the rise of hospital medicine that humoral theories of pathology were rejected and replaced by anatomical pathology (Cunningham and Williams 1992: 2). This framework places “Paris at the centre of a paradigm shift from theoretical

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52 Keel (1998: 148) contrasts the purely descriptive approach of the Paris School with the more physiologically interested “Hunterian School” of anatomical pathology that developed in Britain during the eighteenth century.
studies of disease to their practical application in the dissection room and wards” 
(Waddington 2011: 111).53

Scholars have also identified a strong impulse among practitioners of hospital 
medicine to present their research as free from the conjecture associated with 
medicine before their time, and as a certain science. By adopting the methods of 
clinico-pathological correlation, Corvisart and his colleagues argued that 
medicine had already begun to achieve its goal of diagnostic certainty, and 
against the view that medicine was a “conjectural art” (Albury 1998: 235; see also 
Corvisart). John Warner reports that American medical students looked to the 
perceived empiricism of Paris medicine as a way of shutting off the “gush of 
speculation” that many of them argued tarnished much of American medicine 
(Warner 1998: 354).54 This desire for certainty and abhorrence for conjecture and 
speculation is often emphasized in contemporary discussions of hospital 
medicine. So, according to W.F. Bynum, practitioners of hospital medicine 
thought that it “was fruitless to waste time speculating on the hidden causes of 
things. Rather, the mastery of nature, and the understanding and cure of disease 
would come through the careful observation, classification, and analysis of 
events, objects, or diseases” (Bynum 1994: 29).55 Speculative theorizing about 
disease is presented as antithetical to hospital medicine.

53 Antoine-François de Fourcroy (1755-1809), one of the principle architects of the new system of 
medical training, captured the new school of medicine’s suspicion of medical theories in the aphorism: 
“reading little, seeing and doing much: this will be the basis of the new teaching” (Jacyna 2006: 41).
54 Warner (1998) argues that this pessimistic attitude towards American medicine in the nineteenth 
century was not warranted, but it is nevertheless revealing of Paris medicine’s reputation for rejecting 
rationalistic systems of theoretical knowledge.
55 Bynum also emphasises that the numerical methods of Pierre Louis (another prominent member of 
the Paris School) explored whether treatments worked, but theoretical considerations of why 
treatments did or did not work were ignored. “It was consistent with his empiricism that he hardly 
speculated why these therapies might or might not work: he merely devised clinical trials to see 
whether they did. Only through accumulation of numerous instances could doctors hope to formulate 
general laws” (Bynum 1994: 44). This also emphasises the role that empirical correlation played in
Scholars do sometimes allude to practitioners of hospital medicine using clinico-pathological correlation to develop a distinctive form of physiological explanations for disease. “The consequences of organic heart disease were of course physiological: disturbances of respiration and circulation, pain, and loss of appetite. But Corvisart’s physiology was clinical, not experimental; human, not animal; bedside, not laboratory” (Bynum 1994: 37). “Hope subscribed perfectly to the Parisian notion that symptoms should be explained in terms of structure” (Bynum 1994: 49). Hints that clinico-pathological correlation can also provide the basis for prognostication and therapy are also found in the historical literature. “On the basis of such signs, the lesions of the pertinent organs could be determined, and they were the most definite guides to identifying disease, devising therapies, and making prognoses” (Porter 2006a: 154, discussing the views of Pierre Louis). Jewson says that the “raw materials of medical theorizing now became innumerable morbid events” (1976: 229). Even so, how clinico-pathological correlation is supposed to be used to produce a bedside physiology, to explain disease, to prognosticate or to devise therapies is not discussed. Jewson also refers to the development of machine-metaphors by practitioners of hospital medicine, suggesting that these researchers were interested in developing moving, functional, physiological accounts of disease. “The new conceptions of disease were accompanied by radical innovations in medical analogies and metaphors. Thus, for example, Rostan’s theory of ‘organicism’ compared the composition and disposition of organs of the body with the parts of a moving clock. Once in motion both clockwork and living organisms run out their cycles according to inexorable mechanical laws” (Jewson 1976: 230). This
contrasts with the view expressed in contemporary historical literature that “[t]he thrust of pathological anatomy was towards a static view of disease” (Bynum 1994: 45). In any case, how analogies and mechanical metaphors were used to produce physiological knowledge was not discussed by Jewson. This history contributes to a discussion of how clinico-pathological correlation was used to produce physiological knowledge of disease.

So hospital medicine is supposed to contrast with both bedside and laboratory medicine, in that practitioners of hospital medicine are supposed to reject the speculative methodology of bedside medicine, not to focus on prognosis and therapy, and to be much less interested in explaining disease than practitioners of laboratory medicine. This framework for discussing different modes of production of medical knowledge has proved successful, remains influential (Waddington 2011: 115; Nicolson 2009; Weisz 2001), and will be of great use to me in explaining both Hope’s and Mackenzie’s work. Even so, investigation into the historical development of knowledge of heart disease reveals that some aspects of this framework are a little problematic, and might benefit from reconsideration. In what follows I show how practitioners of hospital medicine did use research into clinico-pathological correlation to develop explanations for

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56 Scholars have argued that anatomical pathology did not originate in the Paris School, but rather had developed in the era supposedly dominated by bedside medicine (Keel 1998; Waddington 2011: 114-115). Hospital based training was not unique to Paris in the early nineteenth century (Waddington 2011: 150). Nevertheless, the damage these critiques have done to this framework, and particularly to the commonplace account of hospital medicine “is not crippling” (Weisz 2001: 112). “No one seriously disputes that from roughly 1815 to sometime in the 1840s, Paris was closely associated with the new medicine. If it was not the only city where sophisticated medical research utilizing the new clinical and anatomical techniques took place, the scale on which such work occurred was unprecedented. To my knowledge, no one seriously challenges the view that the hospital was the central location for research on this scale” (Weisz 2001: 107). Additionally, even though historians have shown that physicians in the era of bedside medicine did sometimes make use of the physical examination, the physical examination was still a rarity in this period (Nicolson 2009). Despite these counter examples “Jewson still holds sway, because the presence of departures from the norms of bedside medicine correlates very closely with variations in either the authority of the patient and/or the attachment of the practitioner to a humoral theory of disease. In other words, the explanatory importance of the factors upon which Jewson based his theories is enhanced, rather than diminished” (Nicolson 2009).
patients’ illnesses, and how they used these explanations to prognosticate and to develop strategies for managing patients. Although I agree that Hope’s work is usefully characterized as hospital medicine, I argue he nevertheless was very concerned with prognostication and the treatment (or at least the management) of patients. I also argue that speculation and conjecture were very important methods which both Hope and Corvisart used to explain how the lesions they observed made patients ill.

2 - Clinico-pathological correlation as the basis for “pathological physiology” in Corvisart’s and Hope’s research
In the early nineteenth century, the English physician James Hope (1801-1841) was a harbinger of significant changes to medical practice in Britain, particularly with respect to the diagnosis and management of diseases of the heart. Hope was an important advocate of the practice of auscultation in Britain, a diagnostic practice that used the stethoscope to listen to sounds produced by the heart’s action. The stethoscope had been invented in the early 1800s by the French physician René Laennec, and used in the Paris hospital system to detect lesions to the lungs and heart before the death of the patient. Hope had been introduced to auscultation by a fellow house physician during his medical training at the Edinburgh Infirmary. After completing his training in Edinburgh in 1825, Hope had gone to Paris to work and study at the Charité hospital, the hospital at which Laennec had trained with Corvisart (Bluth 1970: 204; Duffin 1998: 32). It was here that Hope developed his skill at auscultation, before returning to Britain in 1828 (Bluth 1970: 204). Hope took up a position at St. George’s Hospital in London, determined to popularise auscultation to the London medical community, and to

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57 See letter to Alexander Hannay, printed at the beginning of the third edition of Hope’s Treatise, dated 1839.
continue the research into the diagnosis of diseases of the heart to which he had been exposed in Paris.\textsuperscript{58}

Like other doctors considered as practitioners of hospital medicine, Hope focused his diagnostic attention on the detection of lesions to organs. However, it was not the ability to detect organic lesions post-mortem which was prized by these doctors, but rather the ability to use ante-mortem symptoms and signs to detect organic lesions. “The only end to be desired by the practitioner, is not to investigate, from vain curiosity, what singularities dead bodies exhibit, but to acquire the habit of distinguishing diseases by certain signs, and constant symptoms” (Corvisart 1812: 16). Thus, the focus of research into the diagnosis of disease in the Paris hospital system was the correlation of a patient’s ante-mortem symptoms and signs with the lesions to organs detected at post-mortem examination. The reorganisation of the medical profession in post-revolutionary France created an environment well suited to the pursuit of this project. This is particularly true of the Paris hospital system, where large numbers of patients became available for examination, and their cadavers available for dissection (Weiner and Sauter 2003: 25). In this environment it became possible to correlate the results of the physical examinations of large numbers of patients with the organic lesions of those same patients discovered post mortem, and thus learn how to use the former to predict the latter.

\textsuperscript{58} The recent historiography of James Hope is not extensive. Hope has been called “the first cardiologist in the modern sense” (East 1957: 38), but this perspective is not valuable here. The quarrel between Hope and Charles Williams about who should have priority in their research into the causes of the normal heart sounds has also been addressed (see Fleming (1997: 93) for discussion and bibliography). Hope’s work on the association of murmurs with valvular disease has also been discussed (Fleming 1997: 98), but not with reference to the contrast between Hope and Laennec’s approaches as I do here. Hope’s connection of breathlessness and fluid accumulation with heart disease is also mentioned (Fleming 1997: 77-78). That Hope was a student of the Paris School and a champion of the stethoscope in Britain has also been discussed (Bluth 1970; Nicolson 1993; Bynum 1994). How Hope produced his explanation of how heart disease could cause breathlessness and fluid accumulation has not been discussed in this literature, and it is on this that I focus here.
Hope’s own programme of research had much in common with his Parisian colleagues\textsuperscript{59}. In 1831 Hope published his \textit{Treatise on the diseases of the heart and great vessels} (Bluth 1970: 206), and claimed that in this work “it has constantly been my aim, by studying the symptoms in connection with the morbid anatomy, to trace the alliance of the two as cause and effect” (Hope 1833: xxv-xxvi). The great bulk of the \textit{Treatise} itself is organised anatomically, and is devoted to the description of lesions to and inflammation of various tissues of the heart, and the symptoms and signs these lesions produce (Hope 1833: 84-473, 502-519). In a book of some 600 pages, only 27 are devoted to non-organic disease\textsuperscript{60} (Hope 1833: 474-501).

The organic lesions to the heart described by Hope are many and various, but much of the text is devoted to different kinds of enlargement of the heart, aneurisms of the aorta, and valvular diseases (which produced obstructions to the circulation). These lesions were associated with many symptoms, but some symptoms were described again and again in the text. I want to draw particular attention to \textit{dyspnoea} (breathlessness) and \textit{dropsy} (the accumulation of fluid in some part of the body)\textsuperscript{61}. The dyspnoea was often described as progressively worsening (although it could also occur in fits of “asthma” (Hope 1833: 346)):

“While the enlargement of the heart is moderate, the patient, during a tranquil state of circulation, feels little or no difficulty of respiration; but he is incapable in making the same corporeal efforts as other persons without losing breath: to use a common phrase, he is “short winded”…..When the disease has proceeded so far as to occasion dropsy, more or less dyspnoea becomes habitual” (Hope 1833: 215).

\textsuperscript{59} Corvisart and Laennec had died before Hope’s arrival in Paris. Hope studied under Laennec’s successor (see Bluth 1970).

\textsuperscript{60} Part IV of the \textit{Treatise} is devoted to “nervous affectations of the heart”. This part of Hope’s book is organised symptomatically rather than anatomically, and is divided into chapters on angina pectoris (suffocating chest pain), palpitation (conscious awareness of the heart’s beating) and syncope (fainting).

\textsuperscript{61} Breathlessness and fluid accumulation are also associated with heart disease by Corvisart and Laennec. For more examples of Hope (1833) discussing breathlessness caused by lesions to the heart, see pages 267, 278, 293, 329-330, 346.
As mentioned in the passage above, Hope saw fluid accumulation as an indication of advanced heart disease, where fluid accumulation in the liver and abdomen was seen as particularly prevalent:

“There is scarcely a disease of the heart, accompanied with obstruction of the circulation for any considerable period, which is not productive of enlargement of the liver, and, sooner or later, of its ordinary consequence, abdominal dropsy” (Hope 1842: 24).

Hope was very much of the opinion that the hospital was the best site to conduct clinical research into how to diagnose disease. Working in hospitals gave Hope access to large numbers of patients “where living, literally, I may say, as well as figuratively, at the bed-side of the patient, I had the opportunity of closely watching every habitude and phasis of the disease – every operation and effect of remedies” (Hope 1833: xix). Hospitals also provided a public space, where his diagnosis could be announced (and recorded in writing) before death, and tested by the results of post-mortem examination:

“As the authenticity of cases and observations is of the first importance, I deem it necessary to present a short explanation of the manner in which I have conducted my investigations. Being persuaded that no evidence is so suspicious as that of the senses, because the magnitude of error is in proportion to the certitude which is supposed to attach to that mode of exploration, it has constantly been my endeavour to avail myself of the collective testimony of many. Accordingly, I have, for publication, preferred hospital cases, as being the best attested; I have invariably written the opinions or diagnoses before the death of the patient; have publicly tested them by the results of post mortem examination” (Hope 1833: xxvi-xxvii).

In-keeping with the method described above, Hope presented a series of cases in the final chapter of his Treatise (1833: 519-613), which he had collected from St George’s and St Bartholomew’s Hospital in 1829 and 1830. For the majority of these cases he described the presentation of the patient, his ante-mortem diagnosis, the deterioration of the patient to their eventual death, and the results
of a post-mortem examination, in that order\textsuperscript{62}. All of these cases had severe symptoms on admission to the hospital, and the majority had both symptoms of dyspnoea and dropsy of some form. No patient was reported to have survived more than five months from admission, most lasting just a few weeks, and some dying on the day of admission. Hope’s diagnosis was invariably correct when checked against the results of a post-mortem examination, and he presented these results in a table for ease of reference (Hope 1833: 613).

However, even if the diagnosis of organic lesions using ante-mortem symptoms and signs was the cornerstone of this approach to medicine, for many of these hospital physicians it was not sufficient for good medical practice. Contrary to the vision of hospital medicine presented by Jewson (1976) and sustained by others, many of these practitioners were determined to \textit{explain} how the organic lesions observed could produce the observed symptoms and signs. For instance Corvisart is quite clear that it is not enough to be a pure empiric, and argued that it was important to infer from the results of post-mortem examination how the body \textit{functioned during life} in order to avoid diagnostic error:

“What then is the source of such mistakes? I repeat it, it is the deficiency of the correct physiology. Without this, what avails correct anatomy? It is not enough to distinguish the various springs of the human machine by their names, forms, place, and relative situation, or by their principles, if it be possible; if one does not animate by thought, all the wheels of this astonishing machine” (Corvisart 1812: 18, my emphasis; see also Corvisart 1813: vi-vii).

Here Corvisart argued that \textit{physiology} is of vital importance to medical research. Description of the springs and wheels of the human machine, and of how they

\textsuperscript{62} Of the 31 cases presented, there are three exceptions to this scheme. Two (cases 27 and 29) are from patients from private practice (non-hospital cases), and another (case 28) is of a patient that was admitted to St Bartholomew’s hospital, but appears not to have died, and there are no results of an autopsy recorded (Hope 1833: 604-606).
break, was not adequate. Neither was the correlation of observations made in the living with observations made in the dead. The pathologist’s task was to *imagine* how the lesions observed in the dead would produce the symptoms observed during life. This is rather like an engineer inspecting a large and complex machine, and musing to herself about how the turning of a wheel would move a crank shaft, which would then move a connecting rod, which then would carry out some work. Like this, the engineer could try to work out how the machine works, and how the failure of some part of the mechanism would disrupt the machine’s work. This process is assisted by looking at broken machines, and observing how the failure of different parts of the machine affect the machine’s outputs in different ways. Corvisart distinguished this sort of physiology, which he calls “pathological physiology”, from “systematic physiology”, where the physician makes suppositions about how the human machine works (and fails to work) *without* opening the human machine up at post-mortem examination (Corvisart 1813: vi). Even though Corvisart warned that pathological physiology should always be done whilst “guarding against the too easy inductions of analogy” (1813: vi), I argue that pathological physiology still involved making speculations about how the human machine worked and failed to work, even if these speculations were made off a pathological base.

Hope shared the desire to explain the observed symptoms using the results of the post-mortem examination, or as he put it “trace the alliance of the two as cause and effect” (1833: xxv-xxvi). Indeed, Hope devoted the first part of his treatise to the “anatomy and physiology of the heart” (1833: 1-82). But Hope does not limit himself to animating by *thought*, “all the wheels of this astonishing machine”. Instead of drawing physiological insights from looking inside *dead* bodies, he also looked inside *living* bodies. To do this, Hope carried out a number
of experiments on living animal subjects, including frogs, rabbits and donkeys in 1830 and 1831 (1833: 14, 13, 22). Hope found that he had difficulty observing the action of the heart in small animals, “as, in them, the sounds are too indistinct, the motions too rapid, and the impulse too feeble, to afford satisfactory data” (1833: 12). Consequently he carried out experiments on donkeys, as the larger animal has a slower heart rate, making the action of the heart more easily observable. The animal subject was stunned by a blow to the head, and ventilated artificially with bellows. Its chest was cut open to expose the heart, and the movement of the heart was observed directly (1833: 22-39). Hope used this research to inform his discussions of the physiology of how the sounds of the heart heard at auscultation were produced, and to inform his discussions of how heart disease produced the associated clinical observations of breathlessness and fluid accumulation. The full title to Hope’s (1833) treatise on heart disease (A treatise on the diseases of the heart and great vessels comprising a new view of the physiology of the heart’s action according to which physical signs are explained) is revealing of the goals of his research. Hope’s goal was not simply to learn how to detect lesions using clinical observations before the patient dies. Rather he wanted to explain how those lesions produced those clinical observations in living patients, and he used clinico-pathological correlation and vivisection to do this.

Before I discuss how Hope and Corvisart explained the association between lesions of the heart and the physical signs of heart disease, it is prudent to discuss how he made an association between lesions of the heart and an important group of physical signs – heart murmurs. This association was not the result of simple correlations made between un-interpreted, raw, observations. Rather, Hope’s observations could only be made in conjunction with a sophisticated physiological
understanding how heart sounds were produced. This is particularly evident following comparison with Laennec’s attempts to correlate murmurs with lesions of the heart.

3 – Clinico-pathological correlation of heart lesions and murmurs required a sophisticated physiological understanding of how heart sounds are produced. The particular focus of Hope’s research was to continue the research of Paris physicians (particularly Laennec), and determine the origin and significance of the sounds that could be heard using the stethoscope (1833: xvi-xvii, 10-11).

Following his invention of the stethoscope, Laennec had identified the two main heart sounds by auscultating human patients, and had identified the first sound with the ventricular contraction, and the second sound with the auricular contraction (Hope 1833:10). Hope found otherwise. By observing the action of the heart, whilst simultaneously listening with a stethoscope applied directly to it, Hope was able to “hear the sounds at the same moment that the actions were inspected and felt; since thus alone could it be unequivocally ascertained with what motions the sounds respectively coincided” (Hope 1833: 12, 22-23). Like this, Hope concluded that the first sound was related to the contraction of the ventricles, and the second related to the filling of the ventricles (1833: 40).

Hope also suggested how these sounds were produced (1833: 48-49). Laennec had suggested that some of the sounds heard at auscultation were the direct result of the contraction of the heart muscle63 (1833: 47). Again, Hope disagreed, as the heart sounds were far “louder than the loudest “muscular sounds” that can be produced by any exertion of the most powerful muscles in the body” (Hope 1833: 47). Hope instead came to the conclusion that the heart sounds were not the direct result of the movement of the heart at all, but rather the result of the

63 According to Hope, Laennec only suggests this as an explanation for some murmurs. According to Hope, Laennec provides no explanation for the two main heart sounds (1833: 47).
blood “being thrown into an infinity of conflicting currents” by the movement of the heart (Hope 1833: 48). He argued that the first heart sound was the result of blood becoming turbulent as it was squeezed out of the ventricles, and the second was due to blood becoming turbulent as the ventricle filled (1833: 48-50)⁶⁴.

In addition to these two main heart sounds, Hope also investigated the causes of other sounds. In particular, he investigated the production of sounds he referred to as “murmurs”. These sounds were described as having a different character to the two main heart sounds. For example, murmurs were described as “filing”, “rasping”, “sawing”, “musical or whistling”, “hissing”, “whizzing” and “bellows” murmurs (Hope 1833: 337-338). Laennec had reported these murmurs (or “bruits”), and had thought of them as “preternatural” or “morbid” sounds (Hope 1833: 47, 336). By drawing an analogy with water flowing through a tube, which makes a similar murmuring noise when there is some obstruction in the pipe, Hope reasoned that any obstacle that threw the blood into a “preternatural commotion” would cause a sound: the murmur (1833: 57). In particular, lesions to valves could cause murmurs. Constriction of the valve could obstruct and disrupt blood flow through the valve, causing a murmur. Alternatively, if valves leaked, the regurgitation of blood through the valve could be turbulent, and also produce a murmur (Hope 1833: 54-56). Hope found he was able to identify different lesions to different valves by attending to when the murmur was heard relative to the main heart sounds, and over what part of the chest the sound was loudest (Hope 1833: 340-345). “The accession of auscultation to the other means of diagnosis has rendered it possible to distinguish valvular disease, both in

⁶⁴ In later research, Hope and Charles Williams together found that the normal heart sounds had a valvular component, as they found that these sounds were modified/obliterated if the valves were prevented from closing (Fleming 1997: 93).
general and very often in particular, with almost complete certainty” (Hope 1833: 336).

This conclusion is in marked contrast with that reached by Laennec. This difference of opinion about the diagnostic power of the stethoscope is interesting because it reveals just how dependent the results of clinico-pathological observations were on the interpretation of clinical experience, and not simply upon un-interpreted, or raw, observation. Jackie Duffin has identified that Laennec, having originally concluded that murmurs were signs of valvular disease, changed his mind and argued that murmurs were not produced by structural lesions to heart valves (Duffin 1998: 192). “These phenomena [murmurs] are the more remarkable, inasmuch as they are the only ones discovered by immediate auscultation, which do not depend on structural lesion of the organs in which they are produced” (Laennec 1829: 566; see Duffin 1998: 192). Laennec found that some patients had murmurs, but did not have valvular disease of the heart, and thereby concluded that valvular disease is not the cause of murmurs. He suggested instead that murmurs could be of nervous origin, and that the neurological state of the patient could affect the muscular contraction of the heart and produce murmurs (Duffin 1998: 192)65. Even though Hope also recognised that patients could have murmurs without having lesions of their hearts, he came to the different conclusion that murmurs were caused by and

65 Historians have criticised Laennec for reaching this conclusion, as today many murmurs are thought to be produced by valvular disease (Duffin 1998: 190-191). Duffin has defended Laennec’s work, arguing that many of the patients he examined may have had tuberculosis, complicated by fever and anaemia, both of which can produce murmurs without lesions of the heart valves being present (Duffin 1998: 193). The expectation that Laennec should have been able to reach the “correct” conclusion that Hope did by correlating murmurs with valvular disease is naïve, and not only because murmurs occur in the absence of valvular disease. As I argue here, the results of correlation also depend on what researchers are trying to correlate, and Hope and Laennec approach this subject quite differently. Discussions of whether Laennec was correct in his conclusion will not help to explain why he came to the conclusion that he did, or why Hope reached the conclusion that he did. To do this it is necessary to discuss how the experiences of these two researchers were interpreted, and how interpretation informed the observations they were making.
could be used to detect valvular lesions. To understand this difference it is important to understand that Hope’s observation of murmurs was coloured by his theoretical knowledge of how murmurs are produced in a different way to Laennec.

Laennec was not at all sure about what produced the sound heard as a murmur. He classified different murmurs according to his impression of how similar they sounded. He related these noises to familiar objects like bellows, wood files, grates, and saws (Duffin 1998: 190). In his attempts at clinico-pathological correlation, Laennec tried to marry each abnormal sound with its own lesion (Duffin 1998: 191, 202; see Laennec 1829: 566-579). He was searching for sounds that were “pathognomonique” for a specific lesion (Duffin 1998: 203). He found that these different sorts of sound cannot be correlated with different sorts of lesion. Hope did not think about murmurs in this way. Hope considered murmurs to be caused by turbulent blood. Consequently, the same lesions could produce different sounds as it developed, as changes to the shape of the lesion would alter the flow of blood past the lesion, altering the sound that was made (Hope 1833: 57, 341-342). Hope was not even trying to correlate specific sounds with specific lesions, as Laennec was. As Hope understood murmurs to be the sound produced by a jet of turbulent blood, he was more interested in correlating the position on the patient’s chest wall where the murmur was loudest, and the line along which the sound propagated, than the particular quality of the noise produced (although he did find this informative as well) (Hope 1833: 328-345). Hope also described a number of observations he had found useful when

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66 So, according to Hope, “it may be stated as a general principle, that the loudness of a murmur is in proportion, not only to the roughness of the obstacle, but also the quantity of fluid transmitted through the valve and put in preternatural motion by the obstacle” (Hope 1833: 57). The sound produced is tied to the shape of the obstruction, and not the type of lesion.
trying to distinguish murmurs that were caused by valvular disease from murmurs that were not (1833: 344-345). He was only able to do this because he believed that what made murmurs the same was that they were sounds produced by turbulent blood (which could have many pathological causes), and not that they were all produced by the same pathology.

Hope argued that Laennec had failed to recognise the diagnostic potential of murmurs “in consequence of [Laennec’s] supposing that similar murmurs were produced by spasmodic contraction of the muscular fibre of the heart” (Hope 1833: 336). Hope has this back to front. Laennec did not think that murmurs could not be used to detect valvular disease because he thought they were caused by muscular spasms. Rather, Laennec thought that murmurs were caused by muscular spasms because he found that they could not be used to detect valvular disease (Duffin 1998: 193). However, Hope is correct to say that he and Laennec did not make the same observations because their physiological understanding of murmurs differed.

Pathological physiology involved speculation and conjecture
Hope did not only use physiology to make observations about heart disease. As mentioned above, he also sought to produce physiological knowledge that could connect valvular heart disease with the symptoms of breathlessness and fluid accumulation “as cause and effect”. To understand how Hope connected valvular disease of the heart with the symptoms of breathlessness and fluid retention described above, it is important to understand how he connected valvular disease to the enlargement of the heart. Hope noticed that chambers behind obstructions

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67 According to Hope “the distinctive characters of valvular murmur are, that is not universal over the heart, but confined in a great measure to the part corresponding with the valve affected; that it persists without intermission for an indefinite length of time, even though the heart be kept in a definite state of calm; and that it is often the filing, rasping, or sawing kind; whereas murmurs from other causes almost have almost always the softness of the bellows kind” (Hope 1833: 345).
to the circulation were often enlarged. Hope described two forms of enlargement—hypertrophy and dilatation (1833: 178-180). Hypertrophy (increase in bulk by thickening of the heart wall) was said to happen just as in any other muscle. Increased activity of the heart muscle would lead to increased blood flow, increased nutrition and growth (1833: 187). “Thus, the arms of the smith, and the legs of the dancer, are unusually robust; while limbs paralysed or not exercised, are pale and emaciated” (1833: 187-188). Corvisart made the same claim, providing evidence for it using the example of the increased musculature of manual workers. “Do we not daily observe the extraordinary enlargement of all the muscles of the body, in porters; of those of the arms, in blacksmiths, bakers, &c?” (Corvisart 1813: 59). Now, Hope suggested that an obstruction to the circulation would result in the heart muscle having to work harder to pump blood past the obstacle, and tend to produce hypertrophy (Hope 1833: 188), and lesions to valves could produce such obstructions. For instance, should a valve become constricted, the chamber behind the valve would need to work harder to pump blood through the narrowed orifice. Alternatively, the regurgitation of blood through an incompetent valve was also an obstruction to the circulation, as the jet of regurgitated blood acts to fill the chamber behind the valve in the circulation (Hope 1833: 198). Such lesions to valves would lead to the hypertrophy of the chamber behind the diseased valve.

Hope further suggested that so long as the pressure inside the ventricle was not too large it could still empty adequately. In such circumstances, only the chamber immediately behind the obstruction would enlarge. This is how Hope explained the finding that very often only the left ventricle was enlarged in patients with disease of the aortic valve (in front of the left ventricle) (1833: 196). However, as disease progressed, this state of affairs did not persist. Should the pressure
inside the ventricle become too great, it would be unable to empty properly, and would dilate (increase in volume by stretching the heart wall). This failure of the ventricle to empty properly constituted an obstruction to the auricular chamber behind the ventricle. “[W]hen the distending pressure of blood preponderates over the power of the ventricle, its contents, from not being duly expelled, constitute an obstacle to the transmission of the auricular blood” (Hope 1833: 196). The same process of thickening and dilatation would then occur in the atrium. Again, Corvisart (1813: 83-84) made a similar suggestion, drawing an analogy between the distension of the chambers of the heart behind an obstruction and the distension of the bladder following the blockage of the urethra, arguing that this distending mechanism could rob a muscular chamber (like the heart or bladder) of its contractile strength:

“The urine, in this latter case, gradually accumulating, distends the musculo-membranous pouch [the bladder], which serves it as a reservoir; the coats of the bladder, thus losing their thickness, acquire an extraordinary volume; they lose, at the same time, with their spring, and almost all of their contractile power” (Corvisart 1813: 83).

Corvisart extended this analogy by arguing that the blood vessels behind the obstructed chamber would eventually distend (if the chamber did not rupture), just as the ureters would distend as the bladder became full (1813: 84). Hope agreed, and further argued that, by this mechanism, the effects of a diseased valve could be passed backwards through the circulation, and produce symptoms. So, the narrowing of the aortic valve could lead to the enlargement of the ventricle immediately behind it, and then to the atrium behind that. The increased pressure in the atria would obstruct the flow of blood in the lungs, which would lead to the “copious secretion of watery mucous” into the lung tissue itself and produce breathlessness (1813: 197-198). “The primary effect of universal
obstruction of the lungs by engorgement is, to produce aedema of their cellular
tissue and dyspnoea” (1833: 205). The increased pressure in the lungs would
then affect the right side of the heart. Hope was especially confident that
congestion in the lungs could affect the right side of the heart because, in his
experiments with donkeys, the bellows that ventilated the donkey was stopped,
and the lungs deflated. This would block the flow of blood from the right side of
the heart, which would distend greatly (Hope 1833: 34). Hope suggested that in
a real disease process where the pressure in the lungs was too high, first the right
ventricle would enlarge, and then the right atrium, until ultimately the great
vessels supplying the right side of the heart would become engorged (1833: 196-
197). This engorgement would track back through the systemic circulation, until
organs like the liver were affected and would swell. The increased pressure in
these blood vessels would cause fluid to be pressed out into the surrounding
tissues and body spaces, producing fluid accumulation or “dropsy” (1833: 198-
199).

This explanation for how lesions of the heart can produce breathlessness and
fluid accumulation has proved very influential. Mackenzie referred to it as the
“back-pressure theory” of heart disease (1913: 5). I want to highlight that this
explanation was produced by speculation and conjecture. Hope and Corvisart
made informed speculations about how lesions of the heart could produce
breathlessness and fluid accumulation. Their speculations were informed by
many clinical and pathological observations, and by analogies drawn from
dancers, blacksmiths, bladders, and observations made at vivisection. Informed

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68 Corvisart, in contrast to Hope, did not explain breathlessness as the result of pulmonary oedema as
Hope did. Corvisart suggested that the enlarged pulmonary vessels and heart could lead to
breathlessness “because of the engorged and compressed lungs can admit but a small quantity of air”
(Corvisart 1812: 121). In either case, these researchers suggested explanations for their clinico-
pathological observations, even if their explanations were different.
as it was, pathological physiology was still the speculative explanation of clinico-pathological correlation.

5 – Pathological physiology as the basis for prognostication and therapy
So, Corvisart and Hope sought to diagnose organic lesions to the heart using ante-mortem symptoms and signs, and to explain the presence of these symptoms and signs physiologically. However, this was still not enough, particularly for Hope. Hope was confident that the stethoscope, properly employed, could provide an effective guide to treatment. Indeed, he criticised Laennec for failing to concentrate on therapeutics:

“The treatment of diseases of the heart offers a spacious field for improvement. Previous to the discovery of auscultation, these maladies could seldom be detected before they were so far advanced as to be incurable; and then was not the time to judge the efficacy of remedies. Laennec, absorbed in his investigation of the diagnosis, paid comparatively little attention to the treatment” (Hope 1833: xvii-xviii).

Hope argued that there was no need for a generalised pessimism about therapeutic knowledge. He thought it unsurprising that many physicians thought diseases of the heart to be incurable, as without the light provided by knowledge of the “nature and causes of disease” it was small wonder that therapies were not used appropriately. “[T]herapeutic weapons are dangerous when wielded in the dark” (Hope 1833: xix). But, as he indicated in the passage above, Hope was optimistic that auscultation could give physicians the ability to detect diseases of the heart in their early stages, before they had produced symptoms, and when they were still potentially treatable.

As discussed above, Hope argued that obstructions to the circulation made the heart work harder, and this in turn caused the heart to enlarge. Crucially, he emphasised that he did not think that patients became symptomatic until after they hearts enlarged (1833: xiv). Hope thought that this insight was of
tremendous therapeutic importance. He argued that auscultation could be used to detect an obstruction in its early stages, *before* it caused enlargement (1833: xix, xvii). The patient could then be managed to try to prevent the enlargement from taking place (1833: 326).

“It is of immense practical importance to keep in view the facts stated, namely, that valvular contraction does not produce formidable symptoms until it has given rise to hypertrophy or dilatation; and that it invariably leads to these affectations unless the circulation is kept tranquil. We thus know that the most efficacious treatment of valvular disease consists in employing such prophylactic measures as are calculated to prevent the supervision of hypertrophy and dilatation, and employing them with the same uncompromising strictness before those affectations have appeared, as if they actually existed” (Hope 1833: 326).

Hope’s advice to physicians was to use auscultation to detect obstructions to the circulation from the murmurs they produced, and to do so *before* they produced enlargement of the heart and symptoms of heart disease. So long as the patient lead a “scrupulously tranquil life…..danger in many instances may be completely averted” (Hope 1833: 367). According to Hope, he had “several times known patients with moderate – even with rather considerable valvular obstruction, attain the age of sixty, seventy, and even eighty, though the symptoms, judging from their account, had commenced early in life” (1833: 367). However, if the patient did not adopt a scrupulously tranquil lifestyle, Hope warned that “there is no organic disease of the heart, except adhesion of the pericardium, which tends more rapidly to its fatal termination” (1833: 367).

I want to highlight that Hope is not only making a therapeutic claim here, he is also making a *prognostic* claim. Hope argued that valvular lesions will rapidly lead to the death of the patient if the patient’s activity was not restricted. These

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69 Hope does comment that rest was the best course in the absence of a way of removing this obstruction, a course of treatment which only became available following the introduction of closed heart surgery in the 1940s.
prognostic and therapeutic recommendations were not the result of empirical correlations made between patients with the lesions and their treated and untreated outcomes. Rather they were the result of speculation and conjecture\textsuperscript{70} based on Hope’s physiological understanding of heart disease.

Hope was very optimistic about the power of auscultation to improve the diagnosis and treatment of patients. However, this optimism was not shared by many other physicians following his return to Britain in 1829 (Bluth 1970). In this period in Britain, the stethoscope was viewed with suspicion, and viewed by many as something of a gadget (Bluth 1970: 202). Even after the publication of his Treatise in 1831, which was very well received, many physicians remained doubtful about the value of the stethoscope during the 1830s (Bluth 1970: 206).

To try to put these doubts to rest, Hope organised a demonstration where he taught inexperienced students how to use the stethoscope, to show how easy it was to develop a basic competency with it (Bluth 1970: 207-208; Nicolson 1993: 145). Hope’s Wife, Anne, in a memoir to her husband that she wrote shortly after his death in 1841, reported that Hope even organised a sort of diagnostic contest between himself and a sceptical physician. The sceptic’s name is not reported, but Hope is said to have won this contest:

“The injudicious conduct of one of the opponents of auscultation served to shew its superior claims to the notice of the profession. Dr. \textemdash\ having observed, with some annoyance, the progress of a theory which he regarded unsound, and being unconvinced by the proofs of the superior facilities it afforded for forming a correct diagnosis, announced that it was high time to put a stop to such proceedings, and that he would come down to St. George’s and drive the auscultators from the field. He said that he would choose half a dozen cases, write the diagnosis and defy all the auscultating gentlemen, with their pipes, to throw more light on the cases than he had already done. Dr. Hope desired nothing more than such a public examination, and he joyfully accepted Dr. \textemdash’s challenge. One case was chosen. Dr. \textemdash said that it was hydro-thorax, but did not write his

\textsuperscript{70} I want to be clear that although speculation and conjecture were used as pejorative terms by many actors in this period (including Corvisart), I do not use them in this way here.
diagnosis, though frequently urged to do so. Dr Hope wrote it down as “Hypertrophy and dilatation of the heart. Hydropericardium. Little, if any, hydro-thorax. Lungs gorged and emphasematous.”

The patient died, and post-mortem examination was to set at rest forever the claims of auscultation.

On arriving at the room appointed for the purpose, Dr. ---------, though not producing a written diagnosis, repeated his opinion that the case was one of hydro-thorax. Dr. Hope read aloud his diagnosis as above specified. The case proved not to be hydrothorax, not one ounce of fluid being found in the cavity of the pleura; and on the other hand, every item of Dr Hope’s diagnosis was verified. This is the case of Bryant, the first mentioned in the Treatise of Disease of the Heart. From this time, Dr. --------- was silent on the subject of auscultation, and the half dozen cases were not again heard of” (Hope A. 1842: 75-76).

The form of this contest should be noted. It was agreed by both parties that a fair test of the diagnostic utility of the stethoscope was to examine a very sick patient who was near to death, make a diagnosis of the organic lesion present, and validate the diagnosis against the results of a post mortem examination. Hope’s ability to use the stethoscope to do this was seen as an empirical demonstration of the diagnostic power of the instrument. By publically going through this performance of examining a patient, making an ante-mortem diagnosis, and checking this diagnosis against the results of a post-mortem examination, Hope expected that the fact of the diagnostic utility of auscultation would speak for itself.

Anne Hope described his efforts to popularise the stethoscope like this:

“He never spoke nor argued in favour of auscultation, but allowed facts to speak for themselves. He was always to be seen, stethoscope and journal in hand, at the bedside of every chest case; he took the most minute notes of all, wrote the diagnosis in as great detail as possible, and, before proceeding to a post mortem examination, publicly placed his book on the table in order that it might be read by all; his diagnosis was invariably correct” (Hope 1842: 74, see Bluth 1970).

Ultimately, Hope’s point of view prevailed. “Thus, by 1831, the transfer of stethoscopy from Paris to Edinburgh had been successfully accomplished” (Nicolson 1993: 148). As Bluth (1970) claims, the introduction of the stethoscope
to the rest of British medical society may have been more controversial (see Nicolson 1993: 149). Even so, by 1842 Anne Hope was claiming of the stethoscope that “its accuracy silenced every objection, and all intelligent and candid became convinced of the utility of the stethoscope” (Hope 1842: 74). In a letter to his friend Dr Alexander Hannay (who Hope credits with having first introduced him to auscultation), Hope reflects on the rise of auscultation in Britain:

“Little less than twenty years have elapsed since we studied auscultation together as house physicians to the Edinburgh Infirmary. At that time, there were few auscultators and many opponents in the land. We have lived to see these circumstances reversed; and to you, whose zeal and talents have contributed so powerfully to the change, it must be gratifying to behold this once suspected department of medical science recognised as one of the greatest discoveries, cultivated with avidity by all classes of our profession, and – what is still better – extensively alleviating the suffering of our fellow creatures” (Hope 1842, letter to Alexander Hannay).

6 - The reception of Hope’s views in the nineteenth century
Even though the practice of auscultation appears to have been accepted quite quickly in Britain, Hope’s explanation for how lesions of the heart could cause dropsy (fluid accumulation in the patient’s body) appears to have been a little more controversial. In 1867, Dr. W.H. Walshe (1812-1892)71 claimed that the mechanism of cardiac dropsy was a “vexed question” (1862: 239). Explicitly disagreeing with Hope, Walshe maintained that hypertrophy of the left ventricle need not lead to general dropsy, and commented that Hope “gives no demonstration of the fact, and the motives of his belief are, as far as he shows, totally speculative” (Walshe 1862: 239). He also reported that in his experience it was possible for a patient to have a range of organic lesions without ever developing generalized fluid accumulation. “Mitral regurgitation or obstruction, or aortic regurgitation or obstruction, may severally exist, and for a lengthened

71 According to Peter Fleming (1997: 106), Walshe was the author of the most popular textbook on heart disease in the middle of the nineteenth century – *Diseases of the heart and great vessels*. 

period, without systemic dropsy supervening” (Walshe 1862: 239). These observations led Walshe to suspect that the development of fluid accumulation was not simply due to lesions of the heart, and there must be some other extracardiac factor involved as well:

“These propositions are, I believe, incontrovertible; they are the mere general expression of facts which are perpetually occurring. I cannot, then, see how the conclusion is to be avoided, that something beyond and in addition to any one, or any group, of the cardiac conditions referred to, is required in order, as a matter of necessity, to entail the occurrence of dropsy” (Walshe 1862: 240).

Although he did not want to deny the value of auscultation in detecting potentially dangerous lesions of the heart, Walshe also warned doctors against assuming that the presence of a loud murmur meant that the patient was inevitably going to become ill. “No greater error can be committed than that of supposing the danger of valvular disease, as a class, may be estimated by the amount of murmur they habitually entail” (Walshe 1862: 305).

That Walshe needed to offer this warning indicates that by the middle of the nineteenth century the view that the detection of murmurs at auscultation was being used by doctors as evidence that the patient had a poor prognosis, even if this view was not universally accepted. By the end of the nineteenth century Walshe appears to be on the losing side of this debate about the role of the heart in producing dropsy, and about the prognostic significance of heart murmurs. By

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72 Walshe added that “the existence of some active cause beyond, and independent of, the heart, is further shown by the facts: that there is no direct relationship between the amount of heart disease and of dropsy; that dropsy comes on suddenly sometimes from extraneous causes, the state of the heart remaining, as far as ascertainable, in precisely its previous condition; and that dropsy diminishes and increases, comes and goes, either spontaneously or through the influence of treatment, while organic changes in the heart remain permanent and unmodified” (Walshe 1862: 240).

73 As Fleming has identified, Austin Flint (a famous American physician with a particular interest in the heart) also argued (in 1862) that the presence of a heart murmur did not necessarily warrant a poor prognosis. According to Flint, “truly fortunate were they who kept aloof from the stethoscope of the auscultator” (see Fleming 1997: 95).
the end of the nineteenth century Sir William Broadbent (1835-1907), one of Victorian Britain’s leading physicians (Fye 1990: 62), dismissed Walshe’s concerns, saying that a few exceptions did not invalidate a general rule that lesions of the heart were dangerous and could on their own produce dropsy:

“While admitting the truth of all Walshe’s propositions, it should be added that they relate of exceptions, and the great fact of the association of heart disease and dropsy remains. Were a few exceptions, however unaccountable, to be allowed to invalidate a general law or deductions from general experience in medicine, few of the clinical or therapeutical conclusions in which we are accustomed to place confidence would stand” (Broadbent 1897: 53).

Broadbent also commented that “dilatation and hypertrophy of the left or right ventricle or of both are a necessary consequence of valvular disease of any severity, if the patient lives” (Broadbent 1897: 46). This highlights the close association which was made between valvular disease and the enlargement of the heart in the late nineteenth century, and the perceived danger of valvular disease, which is in close agreement with Hope’s views. Broadbent was also keen to emphasise the importance of restriction exercise in cases of valvular disease:

“For instance, a boy who is allowed to go about immediately after he has contracted a valvular lesion of some severity, and is suffering, say, from aortic incompetence, will be extremely short of breath, and incapable of walking any distance, will have attacks of severe pain in the praecordium, and perhaps fainting fits, one of which may prove fatal; whereas the same patient, if he is kept at rest till the compensatory changes have had time to develop, will be able to take moderate exercise comfortably and go about his work free from pain and respiratory distress, though he may be incapable of any prolonged or violent exertion” (Broadbent 1897: 47).

It should be noted that Broadbent did not think it necessary to restrict the exercise of a patient with valvular disease to the same extent as Hope had advised. After a period of rest, allowing the heart muscle to “compensate” for the valvular lesion
by thickening and becoming stronger (Broadbent 1897: 46-47), the patient would be able to take moderate exercise. Indeed, Broadbent thought it unfortunate that so many patients with valvular disease were routinely prevented from taking any exercise at all:

“Nothing can be worse than to debar all patients who are found to have valvular disease from games and vigorous exercise, and to forbid them to go upstairs or to walk uphill, and on no cases do I look back with greater satisfaction than those, and they have not been few, in which I have liberated boys and girls from such orders” (Broadbent 1897: 90-91).

Nevertheless, that Broadbent viewed this permission to take moderate exercise as a progressive move indicates that the severe restriction of exercise in cases of valvular disease was commonplace. It should also be emphasised that although Broadbent did allow patients to take moderate exercise, strenuous exercise was still considered very dangerous:

“In the class of cases under consideration, supposing a sufficient time to have elapsed after the acute attack in which the valvular affection was established for the necessary compensatory changes to take place, a girl may be allowed to take long walks, to play lawn tennis, to ride, cycle, swim, and dance, and a boy to play cricket and raquets, to hunt, row, box and fence, provided that these exercises are not attended with undue breathlessness and distress, and that they are entered upon gradually and practised with moderation and discretion. On the other hand, football, paper-chases, long house-runs, training for races of any kind, are scarcely permissible” (Broadbent 1897: 90-91).

There is one additional and very important piece of terminology I should introduce, and that is the term “heart failure”. This term was not used by every doctor interested in heart disease (Broadbent (1897), for instance, never uses it in his book). Nevertheless, the term was in use, and referred to patients in which this state of “compensation” for a lesion had broken down, and the patient had become symptomatic. The symptoms said to be produced by heart failure were
those of breathlessness and fluid accumulation, as described by Corvisart (1812) and Hope (1833) in the early nineteenth century:

“Of all classes of chronic diseases there is none which calls in a more urgent manner for the skilful aid of the medical practitioner than heart disease in its advanced stages, when compensation has broken down and the patient has begun to suffer from heart failure, indicated by such symptoms as dropsy, dyspnoea, dyspepsia, insomnia, and cardialga, to name only the more common troubles. Few men in general practice can be without one or more such patients requiring pretty constant attention, who are grateful for any suggestions which may alleviate the burden of their many distresses” (Saundby 1906: 985).

Conclusion
Contrary to the commonly accepted narrative about the development of western medicine in the eighteenth and nineteenth centuries, practitioners of hospital medicine (like Corvisart and Hope) were very interested in physiological explanation of disease, and with prognostication and therapy. Even though this may seem to dissolve the boundaries between bedside, hospital and laboratory medicine, I argue that it does not. It remains the case that the post-mortem and clinico-pathological correlation were utterly central to the way these doctors explained disease, made prognoses, and treated patients. These doctors sought to “animate by thought” the corpses they dissected, so that they could “trace as cause and effect” (Hope 1833) the connection between lesions to organs and clinical observations that they had observed. Corvisart (1812) called this method of producing physiological knowledge “pathological physiology”, and he regarded it essential to medical research.

With respect to the association made between heart disease and breathlessness and fluid accumulation, Corvisart suggested an explanation which Hope then developed. This was that lesions to valves obstruct the circulation, which leads to the sequential enlargement and obstruction of chambers behind the lesions,
causing fluid to be pressed out into the lungs (causing breathlessness), and fluid to be pressed out into the systemic (bodily) circulation (causing systemic fluid accumulation or dropsy). This physiology prompted Hope to suggest the prognostic and therapeutic claim that patients with murmurs and valvular lesions would develop breathlessness and fluid accumulation if their exercise was not restricted.

This physiological explanation was not produced as the simple result of a clinico-pathological correlation. How could it be? Such a correlation was the thing to be explained, and not the explanation. Nor was this explanation produced in a laboratory through experimentation on animal subjects. It was produced in hospitals, by doctors, who suggested explanations for the clinico-pathological correlations that they observed. These explanations were informed by these clinico-pathological correlations, by analogies with other medical and everyday experiences, and in Hope’s case by vivisection. Nevertheless, this physiological knowledge was produced by speculation and conjecture from a clinical and pathological base, as was knowledge of prognosis and therapy. Hope argued that physiological knowledge was essential to therapeutic investigation, as “therapeutic weapons were dangerous when wielded in the dark” (Hope 1833). Physiological knowledge was even necessary to make clinico-pathological correlations, like the correlation Hope made between valvular lesions and heart murmurs, and which Laennec failed to make. The research of these practitioners of hospital medicine was inseparable from physiology, and does not reduce to an exercise in the natural history of disease.

By the end of the nineteenth century many of Hope’s views had prevailed. The stethoscope was widely accepted as a powerful diagnostic tool. Valvular lesions of the heart, which could be detected by finding heart murmurs at auscultation,
were thought to almost invariably cause breathlessness, fluid accumulation and death if the patient’s activity was not restricted. Hope’s physiological explanation for how this happened was also widely accepted. Mackenzie cast doubt on these views.
Chapter 5 – James Mackenzie and heart failure

Introduction
I have argued that the transition from Hope’s understanding of heart disease to Mackenzie’s marks an important transformation in how heart disease was understood. I am not alone in holding this view. The standard historical narrative of this period in the development of knowledge of heart disease is that it marks the transition from an “old cardiology” to a “new cardiology” (Lawrence 1985; Howell 1985; Fleming 1997: 143-166; Macnaughton 2002; Bound Alberti 2010: 82-89). This terminology is used because the historical actors themselves also identified this shift, and described it in these terms (Lawrence 1985: 1-2).

The reasons for the enthusiastic acceptance of this new approach to heart disease by many doctors has been explored. During the First World War, due to the different ways by which the health of a soldier’s heart was assessed, the application of the new cardiology facilitated a reduction in the number of days cardiac cases spent in hospital, increased the number of men who could return to the front, and reduced the number of soldiers eligible for a war pension (Howell 1985). This made the new cardiology economically and politically valuable to the British state. The rise of experimental physiology in Britain at the end of the nineteenth century has been linked to a raised awareness and acceptance of these new techniques and knowledge amongst clinicians (Lawrence 1985). The tracings produced by the machines of the new cardiology were understood by many to be truly scientific and objective, and thus trusted to provide objective knowledge of disease (Osborne 1993: 529; Wilson 1926: 205-207). Even if the reasons for accepting the new cardiology and Mackenzie’s way of understanding heart disease have been explored, the reasons that this knowledge took the form that it did have not.
Much as the discussion of the characteristics of “hospital medicine” was valuable for understanding the research of Corvisart and Hope at the beginning of the nineteenth century, a discussion of the shift from old to new cardiology will be helpful for understanding the shift from Hope to Mackenzie. And yet, this standard narrative will require some augmentation and development, just as the received view of hospital medicine did.

Christopher Lawrence (1985) summarises this transition as being one that shifted the doctor’s focus from the structure of the heart to its function. Instead of focusing on the pathological anatomy, as nineteenth century doctors had done, the new cardiologists are said to have focused on the physiology of heart disease. Instead of thinking of the heart as a dead and static organ, the new cardiologists thought of the living and moving heart (Lawrence 1985: 1-2, 12; see also Howell 1985: 38; Fleming 1997: 143-166; Macnaughton 2002: 552; Bound Alberti 2010: 82-89).

The discussion of Corvisart’s and Hope’s work in the last chapter makes this account of the shift from the old cardiology to the new problematic. Although early nineteenth century researchers examined the dead heart, they did so to learn about the living heart by doing pathological physiology. Lawrence (1985) provides a more sophisticated treatment of the difference between old and new cardiology than many of the scholars that draw on his work. Lawrence argues that this contrast was not simply between a static heart and a moving one, but rather between a moving mental image of the heart muscle (which is still anatomical), and a tracing of this movement on paper (which is truly physiological) (Lawrence

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74 Chris Lawrence’s (1985) is the locus classicus for studies of Mackenzie and the new cardiology. It forms the foundation of Fleming’s (1997), Macnaughton’s (2002) and Bound Alberti’s (2010) account of the subject, as it does mine. Lawrence argues that the rise of the new cardiology took place alongside the institutionalization of cardiology as a discipline (1985: 28-30).
According to Lawrence (1985: 13), the experimental physiology that developed in laboratories in Britain in the late eighteenth century provided a new way of conceptualizing the heart, which fundamentally reshaped thinking about heart disease in this period. These physiologists used instruments that made graphical recordings of the physiological movements of organs, including the heart, using instruments like the kymograph, the sphygmograph, and the electrocardiograph (Lawrence 1985: 2, 10, 13-14). Practitioners of the new cardiology took these physiological laboratory tools and ideas, and applied them in clinical practice (Lawrence 1985: 14-15). Mackenzie’s development and use of the clinical polygraph was seen as ground-breaking work by the new cardiologists, who said that “Sir James Mackenzie was our prophet” (Lawrence 1985: 18; Fleming 1997: 143). Lawrence argues that Mackenzie’s way of understanding heart disease was shaped first and foremost by his extensive reading of experimental physiology, even though Mackenzie worked in general practice and sought to address clinical problems. “Although Mackenzie’s goal was clinical, his thinking was physiological” (Lawrence 1985: 14; echoed by Macnaughton 2002: 551).

75 New cardiologists included physicians like John Parkinson, John Hay, George Sutherland, William Richie, Robert Marshall, Joseph Emmanuel, and (initially at least) Clifford Allbutt, physiologists like Thomas Lewis, anatomists like Arthur Keith, and pharmacologists like Arthur Cushny (Lawrence 1985: 7, 16-19). Many new cardiologists go on to take up professorships, receive knighthoods for their services to medicine, fellowships of the Royal College of Physicians or the Royal Society (Fleming 1997: 157).

76 Both Lawrence and Fleming are quoting Robert Marshall’s (1964) account of the early days of the new cardiology. This is not the only instance of the use of messianic language to describe Mackenzie’s effect on early twentieth century medicine. Sir Clifford Thomas Allbutt (1836-1925) (inventor of the clinical thermometer, Regius Professor of Physic at the University of Cambridge, and revered physician (Porter 1997: 346; 2006c: 352)) said in a review of Mackenzie’s first book (The Study of The Pulse 1902) that “today, from the Galilee of Burnley, comes a new teacher [Mackenzie] to prove to us that our content was shallow enough; and in an important work he has lifted the whole subject into a fresh light” (see Macnaughton 2002: 552, note 15). Mackenzie himself continued the biblical theme. In a letter to Alex Mair (Mackenzie’s second biographer, see Mair 1973), Mackenzie’s nephew the Reverend David Kier reports that Mackenzie told Kier’s father, who was also a clergyman that “You know, I have a gospel too, that means a lot for humanity, and I’ve got to propagate that gospel” (See letter to Mair from Kier, dated 1960, in Royal College of General Practitioners’ Mackenzie archive, Box 17, file B Mack F1-17).
Although the view that Mackenzie’s thinking was shaped first and foremost by tools and ideas imported from experimental physiology is valuable, it is incomplete. In section 1, I discuss the influence of physiological research on Mackenzie’s work. Scholars have argued that Mackenzie was influenced particularly by the work on the cellular physiology of the heart by W.H. Gaskell (a Cambridge physiologist). Whilst this is true, I will argue that cellular physiology on its own cannot explain many aspects of Mackenzie’s understanding of heart disease (section 1.1). In order to understand Mackenzie’s explanation of how heart failure caused breathlessness and pain, it is necessary to discuss his use of commonplace understanding of the physiology of exercise (section 1.2) and his neurological research (section 1.3). Mackenzie’s neurological research was particularly important to his understanding of heart disease, and it has hitherto been entirely neglected by other scholars. Even so, Mackenzie’s aggressive rejection of traditional methods of diagnosing heart disease, his understanding of health and disease, and the functioning and malfunctioning of organs, cannot be understood by appealing to physiological insights alone. The influence of Mackenzie’s job in general practice on these matters has not been discussed by other scholars.

In section 2, I discuss how Mackenzie’s work in general practice was focused on *prognostication* - the provision of information about patients’ future health (section 2.1). His desire to provide his patients with accurate prognoses led him to develop a distinctive research methodology that integrated laboratory and bedside perspectives (section 2.2). Mackenzie sought to uncover the physiological mechanism by which symptoms were produced, so that different sorts of symptom could be distinguished. Patients with the same sort of symptom could
then be followed *forwards in time* to find out whether this symptom carried a poor prognosis.

In section 3, I discuss how Mackenzie’s focus on prognosis shaped his understanding of heart disease. This focus on prognosis led him to argue that patients with valvular disease and murmurs were not necessarily diseased (section 3.1), that the back pressure theory was incorrect, that hospitals were the wrong place to do clinical research (section 3.2), that the medical profession had erroneously become obsessed with murmurs (section 3.3), and that the practice of auscultation had done more harm than good (section 3.4)\(^77\).

Mackenzie’s views about how to diagnose heart failure by attending to symptoms like breathlessness and pain, instead of heart disease by detecting organic lesions, became very influential. Rather than understanding this shift as one from the study of structure to the study of function, or from the dead heart to the living heart, I argue (drawing on Jewson’s (1976) framework discussed in the last chapter) that it can be characterized as a shift in “medical cosmology”. The hospital medicine of the old cardiology shifted to a distinctive integration of laboratory and bedside medicine that constituted Mackenzie’s new cardiology.

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\(^{77}\) Many of the features of Mackenzie’s work have been individually discussed. Even though scholars often discuss many of these features in a single piece of work, they do not show how these features fit together as an integrated whole. These features include Mackenzie’s fascination with explaining mechanisms of disease (Macnaughton 2002: 555; Lawrence 1985: 14-15), his desire to prognosticate (Fleming 1997: 144; Osborne 1993: 535, Lawrence 1985: 14), his desire to follow patients over long periods of time (Macnaughton 2002: 554; Lawrence 1985: 14), his advocacy of general practice as the correct site of clinical research (Lawrence 1985: 14; Macnaughton 2002: 549), his opposition to research done in hospitals (Lawrence 1985: 16), his use of laboratory tools in research (Lawrence 1985: 14-15), and his mistrust of certain ways of using laboratory tools (Lawrence 1985: 16). Mackenzie’s early research into referred pain, and the role of this research in shaping his understanding of heart disease, has been entirely ignored.
The influence of physiology on Mackenzie’s work

1.1 – The clinical application of cellular physiology

As identified by Lawrence and others, Mackenzie was keenly interested in the cutting-edge physiology of the heart in his day, and particularly in the work of Cambridge based physiologist W.H. Gaskell (1847-1914) (Lawrence 1985; Mackenzie 1913: 27-42). Gaskell had gone to study experimental physiology with Carl Ludwig in Germany, and there he had learned to make graphical recordings of the contraction of hearts excised from experimental subjects (Silverman 2002).

Gaskell took tracings of the movements of the atria and the ventricles simultaneously by directly attaching his apparatus to the hearts of experimental subjects, and described the sequence of events that occurred during a contraction of the heart. He saw that the contraction started in the right atrium near the entrance of the vena cava, and spread over both atria, before passing from the atria to the ventricles and then spreading over the ventricles in an orderly way. Using these tracings, Gaskell described five different functions of the heart muscle: 1) rhythmicity, 2) excitability, 3) contractility, 4) conductivity, and 5) tonicity. Mackenzie described these functions as follows: “the muscular fibres of the heart possess the power of rhythmically creating a stimulus, of being able to receive a stimulus, of responding to the stimulus by contracting, of conveying the stimulus from muscle fibre to muscle fibre, and of maintaining a certain ill-defined condition called “tone”” (Mackenzie 1905a: 519)\(^78\). Gaskell was an advocate of the “myogenic doctrine”, which held that the energy and stimulus for the heart’s contraction arose in the heart muscle itself, and was only modified by nervous

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\(^78\) Most of these functions corresponded to a measurement that could be made on a polygraph tracing (Mackenzie 1905a). Using peripheral pulses, the polygraph records the activity of the atria at the same time as the activity of the ventricles. This produces two waves on the tracing, which might be called “a” and “v”. Rhythmicity would be assessed by the frequency of a, conductivity by the size of the gap between a and v, excitability by whether a could cause v, and contractility by the size of v. Tonicity could not be assessed by graphical methods, making it “somewhat vague” as far as Mackenzie was concerned.
input (Mackenzie 1905a: 579; 1913: 27). He argued that following every contraction, the ability of the heart muscle to be excited, to contract was entirely spent: contraction was an “all or nothing” event (Mackenzie 1913: 30). Following contraction, the muscle fibres were said to be “refractory” – they did not respond to further stimulus, and lacked the strength to contract. Unlike a machine, heart cells could become exhausted. Crucially, this meant that they needed time to recover.

Mackenzie, inspired by these experiments, took tracings of the jugular pulse (providing information about the movement of the atria) and the radial pulse (providing information about the movements of the ventricles) of his patients using his polygraph. As identified by Lawrence (1985: 15), Mackenzie sought to do at the bedside what Gaskell had done in the laboratory. Mackenzie sought to apply this physiological knowledge in clinical practice. “Following up the idea of exhaustion or over-excitability of individual functions, I have sought to connect many of the symptoms of heart failure with these functions” (Mackenzie 1913: 31).

Mackenzie argued that the “function of contractility is the most essential and the most notable function of the heart muscle” (1905c: 703). The force of contraction was measured by looking at the size of the wave made by the pulse on the tracing paper. The greater the force produced by the muscle as it contracted, the greater its contractility was said to be. Mackenzie noticed that if the heart rate was slowed using the drug digitalis (which had for more than a century been used to treat fluid accumulation) the function of contractility (as well as other functions, like conductivity) were increased (Mackenzie 1905b; 1905c). If the heart rate slowed, the heart beat harder. Mackenzie reasoned that this was because a slower heart rate gave the heart muscle more time to recover its strength. He further reasoned
that if the heart did not get enough time to recover, the muscle would become exhausted, and fail to pump enough blood around the circulation (1905c: 703; 1913: 14-15). So Mackenzie connected exhaustion at the cellular level with the exhaustion of the whole patient (1913: 12-15). Consequently, he recommended rest as the cornerstone of therapy for heart disease.

Mackenzie’s use of cellular physiology can be used to explain why he developed a forward failure hypothesis for heart failure, and why he treated patients with rest. However, it does not explain his aggressive rejection of the back-pressure theory and the traditional methods of diagnosing heart disease that emphasised the detection of murmurs and valvular lesions. As I have shown, Hope also had a notion that it was a failure in strength of the heart muscle that was responsible for producing the symptoms of heart disease, and not simply presence or absence of valvular lesions. Both researchers appeal to exhaustion of the muscle to explain the symptoms, and both also prescribe rest as the cornerstone of treatment. Why did Mackenzie not believe, as Hope did, that valvular lesions would inevitably lead to the exhaustion of the heart? Mackenzie’s appeal to cellular physiology does not explain this difference with Hope.

Indeed, Mackenzie reported that he was repeatedly told by his colleagues that his views were “common knowledge” (1913: 18). Early twentieth century doctors commenting on Mackenzie’s work did argue that it need not be viewed as particularly revolutionary. One London doctor, Dr Poynton, argued that he thought Mackenzie had “underrated the knowledge that we in London had of the

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79 For Mackenzie the microcosm of cellular physiology was mirrored in the macrocosm of the patient’s sensations and behaviour. “The daily routine of a man accustomed to bodily labour shows a period of rest and a period of work, the one balancing the other, the period of exhaustion of the reserve force requiring a sufficient period of repose for its restoration…..While it is essential for the efficiency of the individual contraction of the heart that a period of sufficient rest should follow each contraction, it is also essential for the efficiency of the reserve force that a period of rest should follow its exercise. If these rest periods are insufficient, then, inevitably, exhaustion sets in” (Mackenzie 1913: 12-15).
myocardium, but the man with a blazing torch naturally looks in the direction of its light, and of his fine work we have no doubt” (1920: 883). In response to lectures Mackenzie gave on his views in 1911 (Mackenzie 1911a; 1911b), another doctor, Dr Samways, argued that the traditional back-pressure view and Mackenzie’s forward failure theory were just two different ways of saying the same thing. Samways did this using an analogy with a horse pulling a cart up a hill:

“It is as though the profession had argued that the reason the horse could not get the load up the hill was because the load was too much for it, whereas Dr. Mackenzie would have us recognize the real reason was that the horse was not equal to the load. With him the fault of the horse, with us, it has been the load” (Samways 1911: 965).

From the viewpoint of cellular physiology, Samways’ criticism carries weight. If all Mackenzie was trying to do was re-cast heart disease in terms of failure of the heart muscle (the horse), instead of obstructions to the circulation (the cart), then it is not clear why Mackenzie saw his work as revolutionary. To understand why Mackenzie did think his work was revolutionary it is necessary to attend to more than cellular physiology.

Furthermore, Mackenzie did not recommend that heart disease should be diagnosed by measuring contractility using the polygraph. Rather, he argued the diagnosis should be made using symptoms like breathlessness and pain. Cellular physiology alone does not explain why he associated these symptoms with heart disease. In order to understand why he did this, it is helpful to turn to Mackenzie’s use of a commonplace understanding of the physiology of exercise.

1.2 – Breathlessness and the physiology of exercise
Mackenzie found that people, whether well or diseased, complained of similar sensations following extreme exertion. People experienced a sensation of
“breathlessness”, or “air hunger” (1913: 51). Some also experienced a sensation of “tightness and oppression of the chest”, and in some cases, even pain in the chest “which in a few became so severe as to present the symptoms that are recognised as angina pectoris” (1919: 157). But breathlessness on its own is not a manifestation of disease. As Mackenzie described, it was a perfectly normal effect of bodily exertion. Mackenzie developed his own terminology to describe the heart’s function during rest and during exertion. When a person was at rest, Mackenzie reasoned the heart only needed to work as hard as was necessary to supply their resting body with blood. Mackenzie referred to this baseline degree of effort as the “rest force” of the heart: “the minimal force consistent with life” (1911b: 859). When a person exercised, however, the heart would have to work much harder, and could increase its workload to match the demands of the body. “But this response has its limits”, there came a point where the heart could work no harder and it ran out of spare capacity (Mackenzie 1902: 4). Mackenzie called this spare capacity on which a heart could draw the “work force” or the “reserve force” of the heart (1911b: 859; 1913: 11). He argued that a person’s capacity for exercise depended upon their degree of reserve force, and as they approached their physical limits breathlessness would result.

Mackenzie argued that this was all that was happening in cases of heart failure. Patients were coming close to using up the spare capacity of their hearts, and became breathless as a result. The only difference was that the disease of the heart meant that the reserve force of the heart was diminished, and this point came more quickly. “In people with damaged hearts, when the heart failure had set in, the symptoms were the same but produced by slighter effort” (1919: 157; see also 1916: 41).
It should be noted that Mackenzie did not think that there was a standardized level of physical exertion that could be used to detect heart failure. Mackenzie viewed the degree of physical exertion at which a person became breathless to be particular to that individual. Referring to a person’s capacity to respond to effort as their “cardiac field of response”, Mackenzie commented that:

“There is no standard by which it may be measured outside each individual’s own experience. A trained athlete may increase his field of response far beyond a man that takes but little exercise, yet the latter may be as healthy as the former. Each individual unconsciously acquires a fairly definite knowledge of what he can do. In early life the field is a constantly expanding one. In early middle age it begins to contract. But so accustomed does the individual become to its gradual variation, that he readily notes the sudden decrease of the field" (Mackenzie 1902: 4).

It was not the absolute level of physical activity that was of interest to Mackenzie, but rather the degree to which this capacity was restricted from the level the individual patient expected. “What I wish to emphasise here, then, is that it is the abnormal facility with which the signs of exhaustion are produced, and not the signs themselves, which is the earliest indication of heart failure" (Mackenzie 1916: 44).

Mackenzie linked a commonplace understanding of the physiology of exercise and physical fitness to the assessment of the functional efficiency of the heart. This way of understanding heart failure seemed so clearly correct to Mackenzie that he was worried about being accused of stating the obvious: “The view that heart failure first sets in by exhaustion of the reserve force of the heart muscle is so reasonable that it needs but to be stated to be accepted" (Mackenzie 1913: 14). Following his consideration of the physiology of exercise, Mackenzie offered a physiological definition of heart failure that focused on the inability of the heart to pump a sufficient quantity of blood forwards in the circulation: “Heart failure may be defined as the condition in which the heart is unable to maintain an
efficient circulation when called upon to meet the efforts necessary to the daily
life of the individual” (Mackenzie 1913: 5).

Mackenzie’s interest in the physiology of exercise contributes to an explanation
of his preference for a forward failure theory, and to an explanation for his
association of breathlessness with heart disease. Again, however, it is not
possible to explain Mackenzie’s rejection of traditional diagnostic practices by
using his interest in the physiology of exercise. Hope had already made the
association between heart disease and breathlessness, as had many other
researchers in the early nineteenth century. Nor is it possible to explain the
association Mackenzie made between heart disease and pain in this way. To
explain this association, it is important to focus on Mackenzie’s early research
into the physiology of pain, which has thus far been ignored by other scholars.

1.3 – Nervous anatomy, embryology and referred pain
The focus of Mackenzie’s earliest research was not on heart disease. Rather, it
was on neurological disorders. He did not publish his first work on arrhythmias
until 1891, whereas he had been publishing on neurological disorders throughout
the 1880s\(^80\). Mackenzie continued to publish work on pain, reflexes, and herpes
zoster (an infectious disease affecting nerves and the area of skin supplied by
those nerves) throughout his career, and his interest in neurology shaped many
aspects of his work. He was particularly interested in how diseases of internal

\(^80\) Mackenzie’s first paper, published in the *Lancet*, was a case report of a patient that was paralysed
from the waist down, and how this patient’s muscular sense was affected (Mackenzie 1883a). His
second paper, also published in the *Lancet*, was also concerned with a case with spinal injury, and how
this injury affected muscular contraction (Mackenzie 1883b). His next two scientific publications
(Mackenzie 1889 and 1890) were also concerned with neurology, specifically with presenting cases of
herpes zoster (patients afflicted with a virus which affects nervous tissue). Another paper on the
“Associated pain of visceral disease” (Mackenzie 1891a) appeared in the *Caledonian Medical Journal*
even before his first paper on the significance of venous pulsation (Mackenzie 1891b) was published in
the same journal (see Mair 1973: 361-368 for bibliography of Mackenzie’s work).
organs could affect the surface of patients' skin through their nervous interconnections.

Early in his career, Mackenzie had got into the habit of recording the areas of the body over which patients felt pain. He noticed that pain was often felt by patients over distinct areas of skin, and this suggested to him that the region of skin affected bore some relation to the diseased organ. At first he could not think what this connection might be, but the research of a Dr James Ross (a Manchester physician), which itself built on research done by Gaskell, showed Mackenzie how this connection could be made.

In addition to his work on the physiology of the contraction of the heart, Gaskell also had done research into the nervous supply of internal organs. Specifically, Gaskell had mapped the distribution of the sympathetic nerves (those arising from the thoracic spine in the upper back and the lumbar spine of the lower back) to the internal organs. Ross wanted to extend this work, and map the distribution of nerves arising from the spinal cord to areas of skin. Ross sought to “discover the area of skin connected with each posterior nerve root” (Ross 1888: 336).

Sometimes this was easy to investigate anatomically. As nerves emerge from the spinal cord they divide to supply different parts of the body. Some of these divisions run directly to the areas of skin they supply without merging with other divisions from other nerves. For instance, most of the nerves that supply the skin over the back behave like this. In these cases, it is reasonably straightforward to determine which nerve roots supply which areas of skin, by following the nerve along its course by dissection (Ross 1888: 336). However, in many cases the divisions of a nerve will merge with other divisions to form a complex, spaghetti junction like tangle of nerves called ‘plexuses’. Most of the nerves that supply the
limbs and the front of the body (and, indeed, the internal organs), behave like this. In these cases, it is far harder to work out which nerve root supplies which area of skin, as the course of each individual nerve cell cannot be easily determined by dissection.

Given that it was difficult to determine by dissection which nerve root supplied which area of skin, Ross (1888) used a different approach. Instead of appealing to anatomical research to answer this question of nervous physiology, he appealed to embryological research.

Ross hoped to shed some light on the question of which nerve root supplied which area of skin by considering the anatomy of higher vertebrates (such as man) as a modification of the anatomy of lower vertebrates. He took the small aquatic creature amphioxus as his model lower vertebrate. In amphioxus, the distribution of sensory nerves follows a very simple, and segmental, pattern. Very roughly, nerves that emerge from the part of the spine nearer the head of an animal supply the skin which was nearer to the head of the animal. Nerves which emerge a little closer to the tail of the animal supply the skin a little closer to its tail, and so on. This leads to a segmental pattern of nerve supply to the skin, which resembles the stripes of a zebra. Each stripe is supplied by a nerve originating from the spinal cord at the same distance from the head as the stripe. Cutting the nerve as it emerged from the spine of amphioxus caused a loss of sensation to the corresponding segment of skin (Ross 1888: 339) (see figure 5).

As discussed above, although this simple segmental pattern of nerve supply described the anatomy of lower vertebrates quite well, this did not appear to be the case for higher vertebrates, such as man. Ross argued that the difference between higher and lower vertebrates was that, over the course of evolutionary
time, the shape of some animals had changed considerably as compared with their distant ancestors. As lineages of animals developed shoulders, limbs, necks and heads, the skin which covered the areas that developed into these structures were pulled around, and the neat segmental pattern was disrupted. As the skin was pulled around, the nerves supplying the areas of skin were pulled around with them. As these nerves were pulled around over evolutionary time, their courses became complex, and they merged with each other in complex ways to form the plexuses. Nevertheless, the nerve roots maintained their connection to the same embryological areas of skin, even if these areas of skin no longer formed a neat segmental pattern. Consequently, if it could be determined which areas of skin in higher animals were embryologically equivalent to the simple segments of lower animals, then the areas of skin supplied by each nerve root could also be determined. Ross tried to map the areas of skin supplied by different nerve roots by using the results of embryological research:

“[T]he segmental distribution is obscured because the nerves have been dragged out of their course by the displacement of old parts which occur in the development of new organs, and more especially the development of limbs. Our task now consists in giving a rough sketch of the developmental displacements which have been the main cause of diverting the nerves from their primitive segmental course” (Ross 1888: 339).

Combining these anatomical and embryological approaches, Ross (1888) produced a map of the areas of skin that were supplied by the various nerve roots. And yet, Ross was not simply trying to produce a map of the connection of different nerve roots and the skin. Rather, he wanted to use this map to explain observations like those Mackenzie had made, regarding the pain felt over particular areas of skin by patients with disease of certain internal organs. Ross
Figure 5: Diagrams of amphioxus in Ross' and Mackenzie's work. The upper (dark) diagram is from Ross (1888: 338). The lower (light) diagram is from Mackenzie (1913: 64). Mackenzie drew on Ross's understanding of the segmental distribution of nerves in his own work on heart failure.
had to link the map he had just made with the map Gaskell had made of the nervous supply to internal organs. Ross linked these two maps together using a theory of referred pain. Nervous signals from a diseased internal organ are carried via nerves that supply them (the “splanchnic” nerves) into the spinal cord. There the particular nerve cells which are carrying this signal come into close proximity with other nerve cells carrying signals from other nerves that entered the spinal cord at that level. These other nerves include those which supply the skin (which are among the somatic nerves, the nerves of the body). Ross suggested that electrical signals from the splanchnic nerves could “diffuse” to other cells close by in the spinal cord. Consequently, signals from the internal organs could be transferred to nerve cells that normally carried signals from the skin, and interpreted as somatic pain:

“The explanation of these associated pains in dyspepsia is to be found in the fact, that the stomach has been developed as an upper thoracic organ, and that in its downward displacement has carried its nerves with it. The splanchnic nerves of the stomach are derived from the fourth and fifth, and probably the sixth dorsal nerves, and when the splanchnic peripheral terminations of these nerves are irritated the irritation is conducted to the posterior roots of the nerves, and on reaching the grey matter of the posterior horns [inside the spinal cord itself] it diffuses to the roots of the corresponding somatic nerves and thus causes an associated pain in the territory of the distribution of these nerves, which may appropriately be named the somatic pain” (Ross 1888: 351).

So in the particular case of dyspepsia, signals from the stomach, carried in splanchnic nerves to the spinal cord, might diffuse to nerve cells carrying signals from a certain region of skin (which in the case of the stomach Ross reports is in-between the shoulder blades). Consequently these signals from the stomach were interpreted as pain over this region of skin.

Ross also made it clear in this 1888 paper that he believed that referred pain was not simply an interesting physiological curiosity. He argued that the observation
of referred pain was valuable *diagnostically* (Ross 1888: 350). Ross argued that knowledge of referred pain could be used in the same way as other aspects of a neurological examination could be used to locate particular lesions of the spinal cord (Ross 1888: 350). He described the characteristic areas of referred pain\(^8\) for pathology of the stomach, lungs, pleura, brain, the liver, bowels, kidneys, gonads, bladder, rectum, and uterus (1888: 350-361), and (crucially from Mackenzie’s point of view) the heart (1888: 355).

Mackenzie embraced the view that referred pain over the surface of the skin could be used to locate diseased organs with enthusiasm. Mackenzie made sense of a number of other observations to do with changes to the skin and muscles of the body wall using the idea of a reflex arc inspired by Ross’s account of referred pain. He argued that the occurrence of the pilomotor reflex (or “goose-skin reflex”) associated with disease of internal organs, and in the same areas of skin as those affected by pain and herpetic blistering, could be explained in the same way as referred pain. He also argued that pain and increased sensitivity of skin over the diseased organ, and contraction the muscular body wall in the same area, could be explained by a similar, protective\(^8\), reflex mechanism (Mackenzie 1893a: 325; 1906a: 1449). He named these reflexes “viscero-sensory” and “viscero-motor” reflexes (1906a: 1451; 1909: 39).

Mackenzie also connected the idea of referred pain with further observations about the distribution of areas of increased sensitivity of the skin (hyperaesthesia)

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\(^8\) Ross does not limit himself to referred pain. He also described how other bodily functions are affected by a similar mechanism. For instance, he reported that lung disease often leads to flatulence, caused by irritation of the pneumogastric nerve. He calls this an ‘associated disorder’ of pneumonia (1888: 352).

\(^8\) Mackenzie was keen to emphasize that he did not think these reflexes were accidents of physiology, but rather useful, protective responses to some noxious stimulus (1911b: 859). “It is necessary to recognise the purpose of these reflexes, as their intelligent appreciation leads to the recognition of their cause, whereas to look upon them merely as purposeless evils, and as indication for something to relieve the suffering and discomfort, tends to hamper an opportunity of diagnosis and rational treatment” (Mackenzie 1909: 20).
and the eruption of blisters in cases of herpes zoster, as the areas affected by herpes zoster were the same as areas affected by certain organic diseases (Mackenzie 1895: 17). The fact that the areas of blistering were visible (and thus did not rely on the patient’s report of where the skin was painful or hypersensitive) allowed Mackenzie to delineate the areas supplied by different nerve roots with greater accuracy and confidence (Mackenzie 1893b: 516). As a plumber might pour water into the end of one pipe in a house to see where the pipe emerges from the house and empties into a drain, Mackenzie used the infection of nerve roots by a herpes virus to trace the course of that nerve cell to the area of skin where the virus emerged and caused blistering. “Recognising the fact that herpes zoster is usually due to an affection of the root of the nerve (probably the ganglion on the posterior root), the areas in which the eruption &c. occur become intelligible and instructive” (Mackenzie 1895: 17).

Of particular importance to Mackenzie was the observation that the area of skin over which pain was felt in cases of angina pectoris (crushing chest pain felt by some patients with obvious heart disease) corresponded “with wonderful accuracy” to the distribution of the eruption of blistering in cases of herpes zoster (Mackenzie 1895: 17). This area of skin was that of the left chest, extending under the left armpit, and down the underside of the left arm, to the little finger on that hand. Mackenzie argued that referred pain could be thought of as a protective reflex, which could be used generally to distinguish disease of different organs, and particularly to identify patients with heart disease (1895: 21-22; 1906b) (see figure 6).

Late in his life, Mackenzie reflected on how he came to see certain symptoms as indicative of a failing heart, and the role that reflex symptoms played in this process (1919: 155-156). He described the case of a patient he had managed in
Figure 6: Herpes zoster and referred pain. Diagrams from Mackenzie’s book “Angina Pectoris” (1923: 31), comparing the areas of skin affected in cases of pain and hyperalgesia with angina pectoris and blistering with herpes zoster.
1891, of a 58 year-old man suffering with angina pectoris. The man suffered from severe pain across his chest and down his left arm on moderate exertion. Over time the pain would manifest following less and less exertion, until the man could only walk short distances before it set in. These were the only symptoms, repeated examinations failed to detect any physical sign of disease, and at rest the man felt quite well. “One day he fell down from his seat and died at once” (Mackenzie 1919: 156). The post mortem revealed coronary atherosclerosis, and this had prevented the blood getting to the muscle of the heart, which had atrophied. This atrophy had proceeded to such an extent that the heart wall had ruptured, causing the patient to die suddenly.

Mackenzie recalled that this case helped him to see the symptoms of heart disease in a new light. This case helped Mackenzie to see breathlessness and pain as an expression of exhaustion of the heart muscle. The pain was a reflex symptom, acting to protect the patient from working harder than his heart could bear:

“The damaged heart could maintain an efficient circulation when the body was at rest, but that on effort, when it had to act with more energy, it became speedily exhausted. The great pain was an expression of this exhaustion, and an imperative call for the cessation of effort. From this experience it appeared that while the body was at rest a damaged heart may maintain an efficient circulation, but when the heart was called upon to do more work, the attempt to do so speedily exhausted the heart muscle” (Mackenzie 1919: 155-156).

Mackenzie did use cutting edge physiological knowledge to develop new ways of understanding the symptoms associated with heart disease. He argued that breathlessness and pain should be seen as manifestations of exhaustion of the heart muscle. However, one might again argue that this connection between the pain of angina pectoris and heart disease was not especially revolutionary, as
these had been associated since the time of John Hunter (1728-1793) (Fleming 1997: 56). Why did Mackenzie think that understanding pain as a reflex symptom constituted such a profound shift in the diagnosis of heart disease? Attending to physiological considerations alone will not explain this.

Mackenzie developed new explanations for the breathlessness and pain seen in heart disease, and for the physiology of heart failure itself. However, these changes were not simply part of a conceptual revolution – a change to how the same patients were thought about. They were also part of a practical revolution – a change to which patients were diagnosed with heart disease. This practical revolution cannot be explained by appealing to physiology alone. Just as appealing to cellular physiology alone did not explain why breathlessness and pain were seen as signs of heart disease, explaining how breathlessness and pain are produced by heart disease need not affect which patients are diagnosed with heart disease. This change to who was diagnosed as diseased was not simply the result of a conceptual shift that prioritized physiology over pathology, as Lawrence and others describe. Rather, it was an important cause of this conceptual shift. Furthermore, the importance of Mackenzie’s occupational role as a general practitioner played in this shift has been underemphasised. To understand this, it is important to discuss the clinical problems Mackenzie faced in day to day practice, and how these affected his understanding of disease.

2 – The importance of prognostication
2.1 – The job of the general practitioner and the patient’s viewpoint
In general practice, the patients Mackenzie saw presented early in the course of their illness, if they were ill at all. Many of these patients would go on to live long and productive lives, whereas some would not. The key point here is that the prognosis (the doctor’s prediction of the future health of the patient) of the patients
Mackenzie saw *was in doubt*. Furthermore, one of the main questions that these patients asked their doctor was what some symptom or sign meant for their future health. Mackenzie described some of the problems that he regularly encountered in general practice:

“Suppose the patient is a youth; he wants to know if he is fit to play a strenuous game. Suppose he is a man; is he fit to pursue a laborious occupation? Suppose the patient is a woman; should she be allowed to become pregnant? Suppose the individual is a life insurance case; how would you estimate the probabilities of his life? When such a concrete case as this is presented to you, you will be at a loss. You can give your diagnosis with precision, but when the essential knowledge from the patient's point of view, is required, you will be conscious of failure” (Mackenzie 1916: 22).

According to Mackenzie, his training had been focused on the identification of organic lesions. He found that in general practice this was not what his patients wanted to know. In general practice, patients wanted the doctor to tell them about their future health, and how they should modify their lifestyles to keep themselves healthy. Patients expected their doctors to be able to give them accurate prognoses, and Mackenzie accepted this role as a central part of his job as a general practitioner. In advice aimed at medical students he went so far as to write that “prognosis is the coping-stone which completes the edifice of your education. All your teaching and study should be but a preliminary training to enable you to understand this problem – the understanding of which, after all, is the most essential part of your education” (Mackenzie 1916: 21).

Mackenzie and his colleagues often told a story of “certain tragic happenings early on in his practice” that focused his attention on heart disease (Hay 1930: 1034; Wilson 1926: 55-58). This related to the death of a pregnant woman and her unborn child; a death that Mackenzie felt it was his responsibility as a doctor to be able to prevent. Mackenzie recalled that “[s]hortly after entering general practice I had the misfortune to attend a pregnant woman who died undelivered on account of heart failure. After this melancholy experience I felt that this death might not have occurred if I had a better knowledge of the condition’ (Mackenzie 1919: 82). Mackenzie described the problem he took for himself as a result of this event like this: “[p]regnancy, in some, induced heart failure, so I wanted to know, did any of these signs indicate heart failure, or did they foreshadow its occurrence?” (Mackenzie 1919: 83). This case is said to have focused Mackenzie’s mind on heart diseases, and it also illustrates why it was that he took prognostication to be such an important task for the general practitioner.
Recall from the last chapter that Jewson (1976) described “bedside medicine” as being a medical cosmology that is focused on prognostication. Jewson argued that because practitioners were employed directly by their patients, the patient’s viewpoint and interests dominated the clinical encounter. Prognostication was consequently a priority for the doctor. In general practice, Mackenzie also worked at the patient’s bedside and was paid directly by the patient. In keeping with Jewson’s analysis, Mackenzie argued that the general practitioner’s role was to focus on questions that were important from the patient’s point of view:

“A patient consults a doctor because he is conscious that there is something wrong with him. The doctor, in his examination, may detect some sign or sensation which he recognises as a departure from normal. He may recognise the mechanism by which the symptoms are produced and be interested in it from a physiological or a pathological point of view; but these are not the points that are essential to the proper performance of his duties, although they may be contributory to that performance. It is from the patient’s standpoint he must view the matter, and apart from the question of immediate relief, the patient’s standpoint is summed up by the question, “what bearing has the cause of this symptom upon my future?” (Mackenzie 1916: 37).

For Mackenzie it was this question that gave “to clinical medicine an aim peculiar to itself and distinct from scientific investigation in any other field”, and which “should be the governing motive of all clinical investigations” (1916: 37). The general practitioner’s first duty was not to pathology or physiology, but to their patients’ problems as their patients saw them84, and particularly to accurate prognostication.

84 I do not mean to suggest here that Mackenzie was particularly concerned with the patient’s own account of their experience of illness. As Osborne (1993: 537-538) correctly points out, Mackenzie was not interested in using the patient’s language. He translated the patient’s reports into clinical language, and argued that “the tendency to prolixity which some patients show clearly must be repressed” (Mackenzie 1909: 102). “Mackenzie’s system was, in short, an attempt to found a ‘patient’-centred medicine, but not a ‘person’-centred one” (Osborne 1993: 538).
Mackenzie argued that the medical profession had hitherto not paid sufficient attention to prognostication. “As a matter of fact, the advances that have been made in medicine have been so much concerned with other aspects of disease, that the study of prognosis has been almost entirely neglected” (Mackenzie 1919: 147). Mackenzie also voiced his concerns about doctors offering a prognosis to a patient, and offering advice on matters like life insurance premiums, without being able to “give rational grounds” for this advice (1916: 139-140). He complained that in the absence of reliable knowledge of patients’ prognosis, doctors would equivocate about what a symptom or sign meant for a patient’s future, and offer what was called a “guarded prognosis” (Mackenzie 1916: 218). This was to advise a patient with some symptom or sign that “probably it meant nothing, but that he had better be careful” (Mackenzie 1919: 174). He argued that offering this sort of vague advice was actually harmful for the patient. Mackenzie was concerned that patients, acting in accordance with the doctor’s advice, might unnecessarily change their lifestyle or career to protect themselves from imagined dangers, and live in a constant state of worry about their health. “We must realise that a guarded prognosis is really a confession of ignorance, and that the real state of affairs is, that there is present a sign of which the physician is ignorant. As he will not admit his ignorance his poor patient is made to suffer” (1916: 218). Instead of offering vague advice, Mackenzie thought doctors should confess their ignorance, and seek to investigate the prognostic significance of their patient’s symptoms and signs properly (1916: 218).

Mackenzie’s comment about vagueness is important. According to him, other doctors not only failed to appreciate and record the prognostic significance of a symptom or sign, but they also did not make clear what a particular symptom or
sign was. For instance, should a patient die with an irregular heart rhythm, Mackenzie complained that often all that would be recorded in case notes was that “the patient's heart became irregular and she sank and died” (1919: 84). Mackenzie argued that this information was far too vague to be applied in clinical practice. How did the patient’s heart become irregular? Were there different forms of irregularity, and if so what made them distinct from each other? How could a doctor at the bedside recognise this symptom or sign in a patient? Mackenzie did not feel that these questions had been addressed with any degree of precision by the medical profession in his time. “The writings of the greatest masters slurred over the subject” (1916: 138). In the face of these vague pronouncements, Mackenzie recalled that he “resolved to try to improve my knowledge by more careful observation” (1919: 62).

2.2 – Mackenzie’s research methodology: the investigation of mechanism and the method of “wait and see”
Late in his life, Mackenzie recalled that initially it was not clear to him how doctors should go about investigating the significance of symptoms and signs to their patient’s health. He reported that he “went blundering on” without any clear scheme in mind for some time, as “until one’s eyes are opened so that medical questions are looked at from a new aspect, it is impossible to know how to set about investigating them” (Mackenzie 1919: 62). Eventually, he asked himself the question which he later took to be the key question that framed all his research:

“After trying one way after another I put to myself the question, “Do I understand the meaning of one single sign or symptom in all its aspects, for example the mechanism of its production, or the bearing of its cause on the patient's future health?” In this way I was brought to a realization of the fact that I did not understand the full meaning and significance of a single symptom” (Mackenzie 1919: 62; see also Mackenzie 1919: 129; and Hay 1930: 1033).
This question has two aspects, corresponding to what Mackenzie claimed were the two basic stages of his research methodology. The first stage was to investigate the mechanism of production of the symptom or sign, so that it could be differentiated from other, apparently similar symptoms and signs. “You will realise that before you can make any advance in prognosis you must clearly define the condition with which you are dealing” (Mackenzie 1916: 25). The second stage was to then go on to investigate the prognostic significance of this symptom or sign.

According to Peter Fleming “[t]he essence of Mackenzie’s research was the study of function, as distinct from structure, of the human heart in health and disease. This he carried out by the traditional methods of detailed analysis of the patient’s symptoms and a physical examination. The latter he supplemented a polygraph of his own invention” (Fleming 1997: 144; see also Bound Alberti 2010: 82 who echoes this view). This characterization of Mackenzie’s research is incorrect. As I have shown, Mackenzie’s methods were not at all traditional. Lawrence (1985: 14), Osborne (1993: 535) and Macnaughton (2002: 551) are more successful in capturing the basic form of Mackenzie’s research as I have described it. All of these scholars refer to Mackenzie’s work differentiating arrhythmias, which I agree is a very good exemplar of Mackenzie’s method. And yet, these scholars do not discuss how the application of this method in general practice influenced Mackenzie's understanding of the diagnosis of heart disease. Lawrence seems to describe this clinical methodology so that he can assert that Mackenzie’s physiological thinking was more important for shaping his understanding of heart disease than were his clinical goals and methods (Lawrence 1985: 14). I disagree with this, and argue that Mackenzie clinical goal, methods, and occupational role
in general practice had a profound influence on his understanding of heart disease that Lawrence (and others) do not discuss.

A particularly clear example of this research methodology is provided by Mackenzie’s investigation of arrhythmias. Mackenzie designed his polygraph to make tracings of a patient’s pulse two sites at the same time. He could thus take tracings of the radial pulse (providing information of the movements of the left ventricle) and the jugular pulse (providing information about the movements of the right atrium) simultaneously (1916: 141). Like this Mackenzie was able to distinguish different types of arrhythmia, such as the “youthful type of irregularity” (known today as sinus arrhythmia), the “adult type of irregularity” (also known as the ventricular extra-systole), and “irregular heart action with paralysis of the auricle” (later known as “auricular fibrillation”) (Mackenzie 1902: 84, 260, 285; 1913: 211).

Mackenzie’s desire to use ideas and techniques borrowed from the laboratory to uncover the mechanism by which symptoms are produced again chimes with Jewson’s (1976) framework of medical cosmologies. In Jewson’s laboratory medicine, the focus of research is on analysis and explanation. Mackenzie sought to provide the correct analysis of monolithic and vague catch-all categories like “arrhythmia”, so that what he saw as essentially different sorts of symptom could be identified. He did this by explaining how the different symptoms were produced. In-keeping with Jewson’s account, Mackenzie tried to develop explanations for symptoms at the cellular level. Although Mackenzie was not a scientist that worked in a laboratory, he did see himself as a scientist, and he did see medical research as a science that should seek to produce exact and objective knowledge (1909: 1-2; 1916: 1). Things that go together in laboratory
medicine, like laboratory tools, a quest for scientific objectivity, cellular physiology, and the analysis and explanation of clinical phenomena, also go together in Mackenzie's research.

Having distinguished symptoms and signs by their physiological mechanism of production, Mackenzie then sought to determine their prognostic significance. Measurements made using instruments (like the stethoscope, sphygmograph, electrocardiograph, and blood-pressure measurements) were often assumed to provide more objective and reliable information about the patient's health status than did the unaided senses. However, Mackenzie warned that this was not the case (1916: 30; 1919: 190). The fact that a patient had an abnormality detected by a tool developed in a laboratory did not mean that they were diseased. He complained that all too often doctors were dazzled by the penetrating insight that the tools of experimental physiology, borrowed from the laboratory, supposedly provided:

“We are accustomed to regard with amusement the fear and respect with which the backwards races treat their idols, yet we are not free of the same tendency ourselves, although the tendency may masquerade under the guise of science. I frequently see eager young men return from a Continental tour, carrying with them some wonderful contrivance – it may be an ingenious stethoscope, or a curious blood pressure instrument. They carry the instrument home with the firm belief that they possess a small idol which will reveal the mysteries of nature. Some of you may view with awe and reverence the small temple in the cellar where you see Dr. Parkinson, as High Priest, performing rites with that mysterious instrument, the electrocardiograph. You see the victim, sitting on a chair, mute and impassive, like a statue of Buddha, and you may fancy the innermost secrets of his heart are being wrested from him. Surrounding all these instrumental methods of investigation there is an element of mystery and a trustful expectation that the fountains of knowledge are about to be unsealed” (1916: 46).

Far from furnishing a clinician with superior abilities, Mackenzie argued that a laboratory training “unfits a man for his work as a physician”, as such a training leads the doctor to place too much trust in laboratory methods, and fail to attend
to observations that were really important (1919: 185-186). Physiological investigation was important, but it was only the first step on the road to really relevant medical knowledge:

“Many have found the irregular heart a fascinating and instructive study. But, notwithstanding this fascination, and notwithstanding the profound knowledge of the heart’s action that has been acquired, the part of the work which has revealed the mechanism of irregular heart is but preliminary. The real work only begins after the physiology has been worked out, and the guide to further research is found in the question: What happens to the people who show these irregularities?

The answering of this question necessitated a long and weary pursuit, a pursuit brightened, nevertheless, from time to time by the discovery of some new fact. Great numbers of patients had to be followed during many years, and close observations regarding the manner in which they bore the stress and strain of life, in acute illnesses and in laborious occupations, had to be made” (Mackenzie 1916: 142).

Mackenzie emphasized that physiological investigation revealed absolutely nothing about a symptom’s prognostic significance. For Mackenzie, there was only one way to determine the prognostic significance of some symptom or sign, such as an irregularity. The doctor must identify a great number of patients who develop the symptom or sign. This must be done at the point when the symptom or sign first presented, before the patients became gravely ill. These patients could then be followed forwards in time, to see if they became ill (1916: 23; 1919: 32-33, 195). Patients may need to be followed for very long periods of time, decades perhaps, before the prognostic significance of a symptom of sign could be uncovered. “Writers who have only been able to recognise [a] condition within the last few years cannot have had time to acquire more than an elementary and imperfect knowledge of the subject” (Mackenzie 1916: 158). This clinical method, dubbed “the method of wait and see” by Mackenzie’s colleague,

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85 According to Mackenzie, “[i]t is manifest, for instance, that if we are to understand the significance of any symptom, particularly its bearing on the patient’s future, we must watch the individual showing it for long periods – it may be for years – to find out what will happen if the condition is left untreated, and further, to find out whether or not treatment may modify its course” (1919: 32-33).
friend and first biographer MacNair Wilson (1926: 50), was central to both Mackenzie's research and to shaping his thinking about heart failure.

By applying this methodology, Mackenzie was able to distinguish the harmful forms of arrhythmia from benign ones. Auricular fibrillation was identified as a dangerous type of irregularity, as many patients became ill with breathlessness and fluid accumulation either shortly after the onset of the arrhythmia, or after a longer period of time (Mackenzie 1913: 229-230). The youthful type and adult type of irregularities were deemed benign, because Mackenzie saw so many people who presented with them who came to no harm (1913: 186; 200). Mackenzie provided numerous examples of patients with arrhythmias, including young children at play, adult men and women who spent their whole lives in physically strenuous jobs, and women who became pregnant and gave birth several times, who never suffered ill effects from their arrhythmias (1902: 87, 106-116, 149). Of the youthful type of irregularity he said:

“You will see from this that I look upon the youthful type of irregularity as a physiological sign and not as evidence of impairment of the heart. Far less do I look upon it as evidence of disease of the heart. The observations on which my view is based extended over a great number of cases. Having detected the irregularity in young men I have submitted them to no treatment. Yet they indulged in vigorous games and afterwards lead laborious lives or engaged in occupations needing the exercise of great muscular strength. I have seen girls, in whom I detected the irregularity, The importance of the method of wait and see for prognostication does not appear to have occurred to Mackenzie at the beginning of his career. “It was not until I had been engaged in research work for many years that the importance of this kind of knowledge dawned on me, and then it was too late for me to complete records with sufficient fullness. This will be understood when you reflect that to complete such a record may demand the watching of individuals for twenty or thirty years; and as I had only realised this after I had passed middle life, the reason of the shortcoming of my observations will be clear to you” (Mackenzie 1916: 19). In a letter to his brother William, dated December 8th 1892 (just a year after he started using the polygraph) he complained that he was “more than ever impressed with the impossibility of doing the work I have set my hand to with any effect here [in Burnley], surrounded as I am with continuous interruptions and my time taken up by petty affairs, and the lack of opportunity of easy and ready references” (Royal College of General Practitioners Mackenzie archive, box 19, file B Mack F2-4; see also Mair 1973: 119-120). In the same letter he plots an escape from general practice into consultancy work in Manchester. At the beginning of his career, Mackenzie does not seem to have believed that general practice was the best place to do research. After a long period of time working in general practice, he changed his mind.
grow up, marry, and have children. In not one of these cases have I ever seen the slightest sign of heart-failure” (1916: 147).

This passage is revealing of Mackenzie’s understanding of disease, and of the influence of the method of wait and see on his understanding of heart disease. Mackenzie did not see the youthful type of irregularity as a disease that did not carry a poor prognosis. Rather, the youthful type of irregularity was not a disease. Furthermore, the reason it was not a disease was because it did not carry a poor prognosis. The method of wait and see did not simply permit dangerous and benign diseases to be distinguished. It provided the grounds for Mackenzie to distinguish disease from non-disease. The application of this method to the study of valvular disease and heart murmurs shaped Mackenzie’s understanding of heart failure.

3 – Changes to how heart disease was understood
3.1 – Valvular lesions and heart murmurs are not diseases
In the particular case of patients with murmurs, Mackenzie was confident that lesions at different orifices could indeed be identified by the murmurs they produced (1916: 22; 1913: 329; 1919: 149). In this respect, he did not disagree with Hope’s conclusions about the mechanism by which these sounds were produced. Nevertheless, with respect to this crucial knowledge about the prognostic significance of murmurs, Mackenzie found the received view wildly misleading.

He reported that he regularly saw patients with heart murmurs, who could carry out very strenuous activities, who were neither sick at the time nor destined to

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87 It should be noted that Mackenzie did not think that there was no further work to do to understand the physiological significance of murmurs. He thought some lesions to valves did indeed cause patients to become very sick, but he could not tell in advance which ones. He advocated further research to try to differentiate those lesions which carried a poor prognosis from those lesions which did not (1919: 149-150).
become sick with heart failure in the future. In his practice he encountered “a
great number of children, who are supposed to have weak or impaired hearts and
are able to run about, are often needlessly restricted, because of some physical
sign – murmur or irregularity” (1916: 95-96). Amongst the adult population he
found that “[i]f a large number of the healthy young of both sexes, during the
second and third decade of life, be examined, a large percentage will be found to
possess murmurs” (1916: 101). In 1916 Mackenzie also described a case he had
seen twenty five years before, in which he examined a colleague for an
application for life insurance (1916: 117). He discovered that this colleague had
a murmur, originating at the aortic orifice, and he found this colleague unfit for
insurance. In 1916, Mackenzie met up with his colleague again. His colleague
had spent the intervening period living and active life and playing strenuous
sports, and had just recently enlisted in the Army. “Though over fifty years of age,
he has never suffered from the slightest symptoms of heart failure” (Mackenzie
1916: 117). Like this Mackenzie claimed to have collected a large number of
cases where patients, in childhood, adulthood and in more advanced years, had
had heart murmurs and lead strenuous lives without becoming ill. “The
assumption, then, that the sounds of a healthy heart must be clearly struck and
free from murmurs is not based on accurate observation” (1916: 101; 1911a:
795).

Mackenzie felt that the view that a patient with a murmur but without other
symptoms had heart disease which had been “compensated” for by changes to
the heart, and was thus in danger of becoming ill if worked too hard, was
erroneous. “Let it be always remembered that frequently sound and healthy
hearts may show a murmur, and that it is necessary, therefore, to seek for other
evidences on which to base a prognosis” (Mackenzie 1913: 341). Rather than
relying on seeming objective signs of heart disease to make a prognosis, Mackenzie argued that if a patient did not have symptoms of heart disease, then their heart was not diseased. “The subjective symptoms of heart failure, on the other hand, are never absent when the heart’s efficiency is in any way affected” (Mackenzie 1916: 45).

This argument became all the more important following the outbreak of the First World War, when many recruits were examined by doctors to determine whether or not they were fit for service. The classification system used my military doctors to describe diseases of the heart had two main categories: “disordered action of the heart” (DAH), which covered arrhythmias, and “valvular disease of the heart” (VDH), which covered murmur producing valvular diseases (Lawrence 1985; Howell: 1985). If a recruit fell into either of these two categories, he was deemed unfit for service. Mackenzie thought these diagnostic practices to be in error, and published an article with case studies to make his point:

“In August, 1914, a lad of 19 was sent to me by his doctor with the statement that he had been rejected for the army because of valvular disease of the heart. I examined him, found his response to effort good, the heart normal in size, and a well-marked systolic murmur at the-apex. I gave him a certificate saying that the heart was perfectly healthy, and that the murmur was physiological, indicative neither of disease nor impairment. I told him to take this certificate to some other recruiting centre than that at which he had been rejected. This he did, was accepted, joined the army, went through his training, carrying his pack and enjoying his route marches. In course of time he was in Gallipoli in the trenches, leading a strenuous life and feeling fit. One day in May, a shell exploded near him and he was blown over and became unconscious. He was picked up, and taken into hospital. After he recovered consciousness, he felt quite fit, but, unfortunately, a medical officer auscultated his heart, detected a murmur, and at once diagnosed valvular disease of the heart. He was kept on his back for a considerable time, shifted from hospital to hospital, repeatedly examined by medical men, and given treatment, till finally he was examined by a Board in August 1915, and invalided out of the service because of valvular disease of the heart. He called to see me a few weeks later; the heart was exactly in the same condition as it was a year before, normal in size, with a physiological murmur, as sound a heart as there is in the British army” (Mackenzie 1915: 808).
It is around the outbreak of the First World War that the first doctors who limited
their private consulting practices to patients with heart diseases appear in
London. Cardiographic departments were set up in the National Heart Hospital
and University College Hospital. Several London Hospital purchased electro-
cardiograph machines, and Mackenzie took up a lectureship in cardiac research
at the London Hospital (Lawrence 1985: 29). The journal *Heart* was set up,
mirroring the specialist neurological journal *Brain* (Lawrence 1985: 29-30;
Fleming 1997: 157). In 1922, fifteen of the new cardiologists who had been
involved in the wartime effort to treat soldiers returning from the front with heart
conditions formed “the cardiac club”, which eventually developed into the British
Cardiac Society (Lawrence 1985: 32; Fleming 1997: 157). By the early 1920s
“[t]here was a journal, a club, a recognized leadership, a technology, and, most
important of all, a coherent intellectual discipline” forming the seed of a
developing specialism in cardiology (Lawrence 1985: 32). The rise of the new
cardiology is tied to the first world war.

Mackenzie thought that the young man above had been unjustly treated by the
medical profession. This patient was prevented from serving in the military
because of the misguided idea that a heart with a murmur was an unhealthy
heart, when in fact it was perfectly healthy. Mackenzie thought that his views were
radically different to the traditionally accepted understanding of heart disease
because they made a difference *in practice*. Even though doctors (like Poynton
and Samways above) argued that Mackenzie’s views were already well known
and widely accepted, Mackenzie argued that this was not the case, because the
traditional ways of diagnosing heart disease and his selected substantially different groups of patients as diseased:

“...I have come across several cases where lives have been insured for large sums and death has ensued within a few months of the medical examination within a few months of the medical examination for life insurance. The lives have been passed as good, because they revealed no objective symptoms, whereas an inquiry into the functional efficiency of the organ would have indicated a grave state of exhaustion of the heart. On the other hand, I am continually seeing people who are rejected for life insurance, or for entrance into the services, who exhibit some simple sign, indicative neither of heart disease nor heart failure, and which the medical examiner has totally misinterpreted and for which he rejected the candidate” (Mackenzie 1913: 18-19; see also 1911b: 860).

Traditional methods diagnosed disease in patients who would never become diseased, and failed to diagnose disease in patients who were soon to die. Notice that, for Mackenzie, a healthy heart was one belonging to a patient who had a good prognosis; and an unhealthy heart was one belonging to a patient with a bad prognosis. Mackenzie’s understanding of health and disease, function and malfunction, was tied to the ability to prognosticate. He assigned such great importance to prognostication because he worked in general practice. Mackenzie’s thinking about heart disease was fundamentally shaped by his work in general practice.

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88 In other words, the patients with organic lesions of the heart valves were not the same patients as those who had symptoms judged to be caused by heart failure, as indicated by symptoms like breathlessness and pain. Howell (1985: 37), Fleming (1997: 95-96) and Macnaughton (2002: 552) all draw attention to the mismatch between valvular lesions, murmurs, and a poor prognosis, but do not argue that this mismatch shaped Mackenzie’s understanding of heart disease. Mackenzie also found that many patients with angina pectoris did not have coronary artery disease, and that many patients with coronary artery disease did not have angina pectoris (Mackenzie 1923: 80-81). Lawrence (1992: 61) draws attention to the mismatch between the pain of angina pectoris and the pathology of coronary artery disease, and uses this mismatch to explain why early twentieth century physicians like Mackenzie did not classify patients according to whether or not they had coronary artery pathology, instead preferring to classify them according to whether or not they had angina pectoris. This was because angina pectoris was taken to be evidence of exhaustion of the heart muscle (Mackenzie 1923: 81). The discussion of Mackenzie’s neurological research in section 1.3 above explains why angina pectoris was taken to be a protective reflex indicating exhaustion of the heart muscle, and thus contributes to the discussion about why patients were classified according to whether or not they had angina pectoris in this period.
3.2 – Hospitals as the wrong place to do medical research

Mackenzie wondered about what had led the medical profession to hold these most mistaken views about murmurs, and offered answers to this question in his published work (1911a: 794; 1913: 6-7; 1916: 30; 1919: 181-182). Mackenzie’s answers to this question have many aspects, but a prominent theme is that he believed medical research was conducted in the wrong place. Mackenzie was concerned that much medical research was being done in hospitals and in laboratories, rather than in general practice (1919: 154).

I should note that Mackenzie did not entirely reject the use of laboratory tools and techniques. Indeed, he employed them himself in his research. Mackenzie did not want to reject the laboratory, he only wished “to guard against their injudicious use” (1919: 193). Laboratory tools were very useful for uncovering the mechanism by which a symptom or sign was produced, but could not be used to determine the significance for the patient’s future health (1919: 198). Any attempt to prognosticate from laboratory research would only be theoretical speculation. “This evidence [of prognosis and treatment] must not rely of theoretical considerations, but must be based on a report of a sufficient number of individual cases, which have been followed long enough to verify claims” (Mackenzie 1919: 195). Mackenzie also argued that laboratory practice was potentially detrimental to good research because the clinical problems encountered in practice could not be reproduced in animals in the laboratory, and laboratory work drew attention away from the patient and their symptoms (1919: 190-191). As the focus of this chapter is the contrast between Mackenzie and Hope, I will focus on Mackenzie’s comments on knowledge produced in hospitals, rather than exploring more fully his views of knowledge produced in laboratories.
Mackenzie was concerned that in hospitals doctors only came into contact with patients in the advanced stages of disease, and rarely saw patients in the early stages of disease (1916: 31). As discussed in the last chapter, practitioners of hospital medicine (like Hope) only saw patients who were already very sick, and often close to death. As this approach to medical research relied on the correlation of ante-mortem symptoms and signs with the lesions detected post-mortem, the main focus of research was on patients who presented to doctors shortly before their deaths. Further recall that the great majority of Hope’s case studies did not survive more than five months from presentation. The prognosis of these patients was not in doubt, as they were soon to die and undergo a post-mortem examination.

The problem with this method of research, as far as Mackenzie was concerned, was that the only way to determine the prognostic significance of some sign or symptom was to follow patients who display the sign or symptom in question forwards in time from when it first develops, and see whether or not they became ill (1916: 32-33). As patients did not go to hospital until they became very ill, and did not go to laboratories at all, “[i]t is manifest that this cannot be done in laboratories or in hospital wards” (1919: 32-33). Mackenzie’s clinical experience had shown him that it was “only a very small proportion of the sick who visit a hospital” (1916: 32). Hence, hospital physicians would never get to see the majority of patients who displayed certain symptoms and signs. Consequently, Mackenzie thought the opportunity for medical research in hospitals was too restricted (1916: 31).

Mackenzie argued that the best place to do medical research was general practice (1916: 32). The family doctor had the unique opportunity to recognise signs and symptoms of their patients from very early on in the course of the
disease, and thus could follow the development of the patient forwards in time to see if they became ill. If the patient became ill, the family doctor could observe how the symptoms and signs developed, whether any new symptoms and signs were displayed, and how these observations were related to each other (1916: 18-20). Mackenzie argued that it was only in this way that secure knowledge of the prognostic significance of a symptom or sign could be uncovered:

“The only way in which a truly scientific progress can ever be obtained is by watching the progress of the disease from its inception till its termination …..The work can only be done by the individual who has the opportunity, and that is the general practitioner” (Mackenzie 1919: 151).

3.3 – The medical profession’s “obsession” with murmurs in historical perspective
Mackenzie applied these insights in his attempts to explain how the medical profession had been misled over the prognostic significance of murmurs. He argued that in his day, a heart murmur was seen as a sure sign of disease, and one which carried a poor prognosis unless the patient’s activity was significantly restricted. This was “an article of belief so deeply and firmly embedded as to be beyond the province of dispute” (Mackenzie 1911a: 794). He argued that in his day “the whole profession [was] obsessed” with murmurs, and yet doctors could not “teach the reasons for their beliefs” (Mackenzie 1916: 30). Mackenzie argued that in order to break this obsession, doctors should turn to medical history to search for the origin of these “cherished beliefs”:

“In order that the profession may appreciate the position held in regard to heart failure and the significance of symptoms as murmurs or irregularities, it will be profitable to enquire into the origin of some of our most cherished beliefs. If we would really seek to find the true reason for many accepted beliefs, we must first consider those who propounded them” (Mackenzie 1911a: 794).

In several places Mackenzie offered a short, historical explanation for how murmurs had come to be seen as a sure sign of heart disease (1911a: 794; 1913: 240)
In this historical explanation, the discovery of auscultation played a central role:

“About a hundred years ago auscultation began to be systematically employed in the examination of the heart. With that injudicious enthusiasm, which at all times has heralded a new method of observation, fabulous qualities were at first attributed to the stethoscope. People were found to have murmurs before their death; hence at a time before the cause of murmurs were known, and long before any single observer had watched individual cases long enough to understand the significance of the murmurs, the statement went forth with all the weight of the highest authorities that these signs betokened grievous heart trouble. To-day, notwithstanding the enormous amount of attention which has been given to the subject, the whole profession suffers from this untrustworthy observation” (Mackenzie 1916: 99).

Mackenzie argued that the view that murmurs indicated disease of the heart, and carried a poor prognosis if left unattended, was the product of a fallacious piece of reasoning: Patients who were very sick were observed to have murmurs, so patients with murmurs must also be very sick (1916: 99; 1919: 193). Mackenzie objected to this argument, saying that “surely it is not logical to reason that because some valve murmurs indicate defects all valve murmurs are therefore signs of defects” (Mackenzie 1911a: 794). In hospitals, doctors were only exposed to those patients whose valvular lesions and murmurs had turned out to be harmful. Hospital doctors were never exposed to the majority of patients with valvular lesions and murmurs, who never went to hospital. Consequently, hospital doctors were forced to speculate about the future health of these unseen patients, and tended to assume that they were like the patients that were seen in hospitals. Mackenzie argued that hospital medicine invited “loose” and “speculative” conclusions to be drawn about the prognostic significance of sign and symptoms, because it was impossible to follow patients forwards in time from when the sign or symptom first presented (1916: 36).
Indeed, the whole project of trying to correlate ante-mortem symptoms and signs with the results of post-mortem examinations, whilst so central to “hospital medicine”, was suspect for Mackenzie. This project placed far too much emphasis on the detection of organic lesions, and not enough on assessing the functional status of an organ, which was what the patient really needed to know (1916: 37-38):

“Hitherto the presence of some organic lesion, as a damaged valve or sclerosed coronary artery, has been looked upon as the cause of heart failure, and the physician has been content with the finding of such gross changes. But if we would understand the action of any organ, we must not look merely to its post-mortem appearances, but seek for the modified functions that occur during life” (Mackenzie 1913: 8).

Mackenzie drew the general conclusion that using physical signs (like murmurs) to detect organic lesions (like valvular lesions) did not provide much information about the patient’s prognosis, and thus did not provide much information about the functional status of the patient’s organs. Instead he argued that symptoms (like breathlessness) and reflex phenomena (like pain) were much better guides to prognostication, and thus to the functional status of organs (1909: 15-21; 1919: 137).

Having said this, Mackenzie knew that many investigators in the past had not simply been “content with the finding of such gross changes”. He was fully aware that these investigators in the past had attempted to explain the associations they had made between ante-mortem symptoms and signs and organic lesions found post-mortem. As discussed in the last chapter, this was certainly the case. Hope wanted to “trace the association of the two as cause and effect”, and Corvisart had made it clear that he thought it essential to try to “animate by thought all the wheels and springs of this astonishing machine”. Hope had even turned to vivisection in an effort to study the functioning of the living body. However,
Mackenzie was still critical of this organic pathology centred approach as a method of producing knowledge of how the body functioned. This was because this approach invited invalid conclusions about the significance of symptoms and signs, as described above. The finding that a symptom or sign was significant required an explanation. As the attribution of significance was inappropriate, the explanation developed to account for this significance would also be off the mark.

Mackenzie was very critical of the explanations of the symptoms of heart disease offered by his contemporaries:

“These views are the outcome of the discovery of auscultation. Shortly after its discovery, before the meaning of the heart sounds, normal and abnormal, had been understood, bruits were found to be associated with extreme heart failure. This association was at once assumed to be of vital significance, and in later years when a better knowledge of these murmurs permitted the recognition of their causation as being due to defects of certain orifices, their importance as the origin of heart failure was supposed to be established, and gradually the back-pressure theory was evolved to explain heart failure” (Mackenzie 1913: 6).

The “back-pressure” view is what Mackenzie called the theory that an obstruction to the circulation will produce a sequential enlargement and build-up of pressure in the chambers of the heart, proceeding backwards through the circulation (Mackenzie 1913: 5-6; 1911a). It is the theory put forward by Hope to explain the symptoms he associated with valvular disease and enlargement of the heart (1833: 196-199)\(^89\). Mackenzie saw the back pressure theory as supporting the view that murmurs were a sign of disease, because it linked obstructions to the circulation (which produced murmurs) with the symptoms of breathlessness and

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\(^{89}\) Mackenzie never discusses Hope’s research in his work. Mackenzie commented that he had managed to trace the back pressure theory to the 1850s (1911a: 796), but appears unaware of Hope’s earlier presentation of it. Several doctors in the generation following Mackenzie would argue in favour of the back-pressure theory of heart failure (and thus against Mackenzie) (Harrison 1935; Bedford 1939), and would look to Hope as the originator of this view.
fluid accumulation associated with heart failure. It provided a rational for viewing patients with murmurs as diseased, and for restricting their activity.

In addition to his concerns about the misappropriation of significance, and the effect this had on the investigation of function, Mackenzie was also concerned that focusing on very sick patients also hindered medical research. If patients were not followed forwards in time from when they first showed the symptom, there was no way of knowing whether disease actually unfolded in the way the proposed theory suggested it should:

“It may be remarked that this back-pressure view did not arise from careful observation of individual cases passing through different stages, but from attempts to describe the progress of the failure by reasoning backwards from the evidence present in advanced conditions. That is to say, cases with histories of damaged valves have been found with all the evidences of heart failure, and with the changes in the heart and lungs. The physician has then filled in the gaps of the history by assuming that the events happened as required by the theory” (Mackenzie 1913: 6-7).

As far as Mackenzie was concerned, the “back pressure” theory had not been “derived from patient observation in individual cases, but has been evolved as the result of speculation as to the cause of some imperfectly understood symptoms” (1916: 159). Mackenzie thought that attempts to uncover the causes of disease whilst limiting observations to the late stages of disease as “generally little better than guess work” (1919: 5). He believed that his profession’s “obsession” (1916: 30) with murmurs, the significance attributed to this sign, and the back-pressure theory which this obsession had spawned, were all badly mistaken. He argued that the attempt to carry out medical research in venues like hospitals, where patients were only seen in the late stages of disease, severely restricts the opportunities for research and leads to these erroneous conclusions.
In addition to simply being wrong, Mackenzie argued that these “cherished beliefs” (1911a: 794; 1916: 34) of the medical profession about the significance of murmurs and the value of the back-pressure theory had “blinded the profession to the real facts” (1916: 158) and were adversely affecting the care of patients:

“Perfectly healthy men are being rejected for the Army, or invalided out of it, because a murmur has been detected in their hearts. Others who present themselves for life insurance, are rejected or made to pay a higher premium for the same reason, while innumerable individuals are subjected to prolonged treatment and great restrictions in their mode of life because these early superficial observations have misled the profession” (Mackenzie 1916: 99).

This harm he perceived being done to patients drove Mackenzie to argue that the back-pressure theory should be rejected. He made this argument forcefully. In several places Mackenzie even argued that the practice of auscultation had harmed medical research and was harming patients, as it was not the powerful diagnostic tool many doctors believed it to be (1913: 6-7, 322; 1916: 30, 99; 1919: 181-182). For instance, in a section of his textbook *Diseases of the Heart* (1913) entitled the “evil done by the back-pressure theory”, Mackenzie expressed his concerns about the diagnostic value of the stethoscope:

“The combating of this theory might be considered unnecessary and superfluous if it were not for the fact of the grievous mischief it has done to individual patients, and the hampering affect it has had upon the advance of medical science. It is questionable whether the discovery of auscultation so far has done more harm than good” (1913: 7).

In order to prevent this “grievous mischief”, Mackenzie thought doctors needed to become more critical of the knowledge and practices with which they had been indoctrinated. The trouble was that “the bonds of tradition are hard to shake off” (1916: 34). By discussing the historical origins of these erroneous beliefs, Mackenzie hoped to free his profession from its fetters, and help them to
overcome what he saw as “superstition” and an undue “reverence for authority” (1911a: 794; 1916: 34).

4 – The influence of Mackenzie’s work
As discussed in chapter 2, Mackenzie rejected the practice of diagnosing heart disease based on the presence of murmurs and valvular lesions, and he rejected the back-pressure explanation for the symptoms of heart disease. Instead, he argued that heart disease should be diagnosed by attending to the syndrome of heart failure (comprised of symptoms and signs, like breathlessness, fluid accumulation and pain). Indeed, he argued that heart failure could be diagnosed in patients with this syndrome, even if there was no obvious pathology of the heart (1913: 708). Mackenzie therefore found that patients could have occult heart disease.

As Lawrence (1985: 21) points out, the new cardiology was not universally accepted by the British medical establishment. In particular, Clifford Allbutt (who had initially endorsed Mackenzie’s work on arrhythmias), was quite sceptical of Mackenzie’s views about the limited importance of valvular lesions and murmurs (Lawrence 1985: 21). Allbutt was concerned that Mackenzie and his followers had been too quick to condemn the traditional views on how heart failure came about:

“Here however I would venture to the disrespect with which certain cardiac murmurs have been treated by Sir James Mackenzie – to whose researches we are so deeply indebted – and by his disciples. This is an extreme reaction against the obsequious regard for these signals which not unnaturally prevailed in the generations immediately after Laennec” (Allbutt 1917: 139).

Allbutt said that he could recall vividly a large number of cases of heart failure, who in their past had had some incident, such as an attack of rheumatic fever, which had damaged their valves and had subsequently developed heart failure.
Allbutt thought that this clinical phenomenon supported the traditional backpressure views, should certainly not be dismissed so casually by Mackenzie and his followers:

“In looking back at my many years of practice I recall very vividly, in respect of the present subject, the subsequent lives in many a patient in whom a mitral regurgitant murmur was for years the precursor of subsequent cardiac incapacity and ultimate failure” (Allbutt 1917: 140).

Many of Mackenzie’s observations about the production of medical knowledge, and his historical expositions of the development of this knowledge in the case of heart failure, seem designed to counter this sort of objection. Allbutt claimed to be able to recall a large number of patients who had developed heart failure, and who had had murmurs years before. Recall that Mackenzie argued that this did not mean that a large proportion of patients with heart murmurs would go on to develop heart failure (Mackenzie 1911a). Also recall that Mackenzie did not believe that damage to valves was irrelevant to the aetiology of heart failure. He believed that some murmurs certainly were responsible for the development of this condition. The trouble was that as yet doctors could not tell in advance which patients with murmurs were going to develop heart failure and which ones were not (1919: 150). What Mackenzie objected to was the practice of using the murmur as sure sign of impending trouble, and restricting the life of the patient as a consequence. He felt the traditional back-pressure understanding of heart failure encouraged doctors to adopt these practices, and this was why he objected to this theory as strongly as he could.

In light of these comments by Allbutt, I should highlight that I am not arguing that the shift from old to new cardiology was universally accepted. At first, it was only enthusiastically pursued by a few physicians (Lawrence 1985: 21). Many physicians during the First World War continued to consider heart disease from
the point of view of anatomical pathology, and to classify patients into those with valvular and non-valvular heart disease (Lawrence 1985: 21-22; Howell 1985: 38). Even Allbutt curbed his early enthusiasm for the new cardiology, and argued that the importance of valvular lesions and murmurs should not be dismissed (Lawrence 1985: 21-22). Fleming points out that, even in the 1920s, many physicians and researchers continued to think of heart failure in terms of the back pressure theory (1997: 147-148). The new cardiology did not obliterate the old, but rather came to be more prominent in the early twentieth century. Different ways of understanding heart failure ran in parallel and interacted with each other in this period. Nevertheless, Mackenzie’s work did have a significant impact in re-shaping how heart disease was understood in Britain in the early twentieth century.

This influence is exemplified by changes made to William Osler’s textbook (The Principles and Practice of Medicine – which Lawrence (1985: 5) describes as “possibly the most widely used medical textbook of the early twentieth century”). The section on diseases of the circulatory system on heart disease in the first (1892) edition of this textbook describe disease almost entirely in terms of the anatomical location of the lesion (Osler 1892: 581-659). Diseases of the pericardium, endocardium, valves and myocardium are discussed, following the anatomical scheme seen in Hope and Corvisart’s texts. Disease of the myocardium are not discussed in terms of its function, but rather in terms of aneurism, rupture, growths, parasites, wounds and foreign bodies, which are all lesions visible at post mortem. Only ten pages at the end of the section are devoted to problems that are not visible lesions, like arrhythmias, palpitation, and angina pectoris (1892: 649-659). Even so, Osler still discusses angina pectoris in anatomical terms, saying that it is “not an independent affection, but a symptom
associated with a number of morbid conditions of the heart and vessels” (1892: 655). In contrast to this, the 1921 edition of this textbook (written after Mackenzie’s rise to prominence), includes a section of “cardiac insufficiency” amongst affectations of the myocardium (Osler and McCrae 1921: 781). In this section Osler advances the views of the new cardiology. “With lessening of the muscular power of the heart, the rapidity with which the blood circulates is diminished, and the tissues fail to receive their proper supply of oxygen and food, and to be adequately relieved of their waste products – this is cardiac failure” (Osler and McCrae 1921: 781). This book also refers to the reserve power of the heart, and how this power “disappears in heart failure” (1921: 781). Mackenzie’s ideas were being taken up into mainstream medical thinking in the early twentieth century90.

Conclusion
A commonplace view about Mackenzie is that his research was shaped first and foremost by his reading of experimental physiology, and particularly by W.H. Gaskell’s work on the cellular physiology of the heart. There is much truth in this view, and it is bolstered by some of the material I have presented in this chapter. Mackenzie’s understanding of the physiology of exercise complemented his reading of Gaskell’s work, and helped him to see breathlessness as a sign of heart disease. Mackenzie’s neurological research on the physiology of pain, Osler died in 1919, and the 1921 edition of his textbook is credited to the late Sir William Osler and Thomas McCrae. The 1921 text displays a curious combination of forward failure theory and back-pressure theory: “Weakness of the left ventricle fails to give proper filling of the arterial system and general anaemia of the tissues results. Failure of the left auricle means stasis in the lung vessels with deficient aeration of the blood, and a tendency of oedema of the lung or to effusions into the pleural cavity. Failure of the right auricle and ventricle gives cyanosis of the organs, dyspnoea at rest on slight exertion, with stasis in the abdominal organs and oedema” (1921: 781). This text shows that forward failure (diminished supply of organs in front of affected chambers) and back-pressure (stasis and oedema behind a lesion) physiologies could be mixed together in complex ways in this period. For a discussion of the transatlantic exchange of medical knowledge personnel in the early twentieth century see Woolley (2002), especially pages 231-260.
which has hitherto been neglected by other scholars and which also drew on Gaskell’s work, helped him see angina pectoris as a reflex symptom indicating heart disease.

And yet, these new physiological explanations for breathlessness and pain do not in and of themselves explain Mackenzie’s aggressive rejection of traditional ways of understanding and diagnosing heart disease. Angina pectoris, breathlessness and exhaustion of the myocardium had all been associated with heart disease in the eighteenth and nineteenth centuries, and were fully compatible with the traditional view that organic lesions of the heart caused these symptoms. Some commentators argued that Mackenzie’s views were not revolutionary as they were already well known, and even claimed that Mackenzie’s forwards failure theory and the traditional back-pressure theory were just different ways of saying the same thing. Mackenzie rejected these arguments, and argued that his understanding of heart disease was dramatically different to the way heart disease had traditionally been understood. To see why Mackenzie rejected traditional views about heart disease it is necessary to appeal to more than his reading of physiology.

Mackenzie’s research methodology and his understanding of disease were shaped by his job as a general practitioner. This job focused his attention on prognostication, as this was a central concern for the patients he encountered. He used laboratory tools and ideas to analyse and explain how symptoms and signs were produced, so that he could follow patients with the same sort of symptom forwards in time, and determine whether or not these symptoms or signs were prognostically significant. His desire to accurately prognosticate fundamentally shaped his understanding of health and disease, and of function and malfunction; so much so that if the presence of a symptom or sign did not
carry poor prognosis with it, then for Mackenzie it was not a symptom or sign of disease. This understanding of disease led Mackenzie to reject many of the central tenants of the old cardiology. He argued that valvular lesions and murmurs were not necessarily signs of disease, that hospitals were the wrong place to do medical research, and that the practice of auscultation may have done more harm than good. This understanding of disease and function did not derive from his reading of physiology, but rather from his occupational role in general practice. His arguments that heart failure should be the focus of a physician’s diagnostic attention was not simply a result of his reading of physiology, but also the result of his clinical experience that detecting organic lesions did not allow him to prognosticate accurately in general practice. Mackenzie’s thinking was both physiological and clinical.

The difference between the old cardiology and the new was not that the old cardiology studied structural abnormalities whilst the new cardiology studied functional abnormalities. Early nineteenth century researchers were just as interested in physiological explanations of how symptoms were produced as were their early twentieth century counterparts. Old and new cardiologists were also interested in producing knowledge of prognosis and treatment. The difference lies not in what these doctors were interested in, but rather in how they pursued these interests. The old cardiology preferred clinico-pathological correlation and pathological physiology, whereas Mackenzie (a key representative of the new cardiology) preferred his distinctive combination of the investigation of a symptom’s mechanism of production and the method of wait and see.

Mackenzie’s methodology has elements in common with both Jewson’s (1976) laboratory and bedside medicine, but it does not seem to fit neatly into either medical cosmology. This finding might be taken to show that Jewson’s analysis
is flawed. However, it can also be interpreted as showing the powerful insight provided by Jewson’s analysis. Mackenzie’s desire to analyse and explain his patients’ symptoms did drive him towards tools and ideas developed in laboratories, and his occupational role at the bedside focused his attention on prognostication. He *fused* these two approaches into a distinctive and productive medical cosmology of his own. The shift from the old cardiology to the new can be thought of as a shift from hospital medicine to a fusion of laboratory and bedside medicine.
Chapter 6 – Conventionalism and medicine: how early twentieth century philosophy of science suggests how medical facts can be invented and discovered

Introduction
In the first chapter of this thesis I argued that doctors in the present day are struggling to justify their opinions about how best to diagnose heart failure. For instance, the main argument used to justify the European Society of Cardiology’s position on how to diagnose heart failure accurately is circular. I linked these problems with the ancient philosophical problem of Meno’s paradox. I further argued that this problem manifested because these doctors are searching for timeless truth about how to accurately diagnose disease. I argued that this search for timeless truth presented an apparently insurmountable challenge to doctors. I then presented a longue durée history that reconstructed the development of our knowledge of heart failure from the early 1800s to the present day. This was done in the hope that looking at historical literature would reveal how our knowledge had been justified.

This effort was only partly successful. The longue durée history did provide an explanation of why it is certain opinions about how to diagnose heart failure accurately are held by doctors in the present day. However, it is far from clear that our present-day knowledge of heart failure was justified, in the sense that the evidence and arguments used to support these present day positions force us to accept these positions as “The Truth”, or close to “The Truth”. The longue durée history identified seven significant changes to how heart failure/heart disease was understood from the early 1800s to the present day. The majority\(^91\) of these

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\(^91\) The relevant historical actors argued anomalous observations meant that they had to revise their views on heart failure. Only the transition between stage four and stage five seems to have taken place without a conflict with experience to resolve.
changes were precipitated by unexpected and anomalous observations that were interpreted by historical actors as evidence that their views on heart failure had to be changed. Even though these actors believed that the available evidence forced them to modify their understanding of heart failure in the way that they did, I have argued that this is not the case. In each case these conflicts with experience could have been resolved perfectly well by alternative means. The positions these actors adopted in the face of these conflicts with experience were not inevitable given the evidence. Rather, they were historically contingent.

Even if these different ways of understanding heart failure are historically contingent, this does not mean that they were just made up. Even though the actors involved did not have to adopt the ways of understanding heart failure that they did, this does not mean that they were free to believe anything they wanted. The knowledge of heart failure that they accepted was not simply stipulated by them. Rather, in each case they were constrained by the way the natural world is.

Following on from my longue durée history, I have reviewed the contemporary literature form the history and philosophy of medicine, searching for a philosophical approach that could capture both of these aspects of the production of medical knowledge. I was unable to find any contemporary historians or philosophers of medicine who could provide a detailed account of how biological knowledge of disease could at once be historically contingent and dependent on the way that the natural world is. On the one hand, some historians and philosophers of medicine hold that the biological aspects of disease are fully determined by the way the natural world is. Accepting this, we have no choice in the matter if we wish to avoid self-deception. On this view, the biology of a disease is an entirely discovered thing and not at all historically contingent. On the other
hand, other historians and philosophers of medicine argue that the disease with which a patient is suffering is fully determined by what people say and do. As such, it is only the choices made by people that constrain disease identity. On this view, knowledge of disease is an entirely invented thing and not constrained by the way the natural world is.

Advocates of each side in this discussion have expressed their concern that acceptance of the other will adversely affect the care of patients. On the one hand, philosophers and historians who argue that our knowledge of disease is invented are concerned that failure to reflect on the historically and socially contingent character of medical knowledge will lead to medical knowledge claims being accepted uncritically. “What is presented as fact, particularly as a scientific fact, often has a cachet similar to deliverances of divine revelation to a community of believers. It may be considered a value free, timeless reality. This engenders difficulties” (Engelhardt 1996: 196). On the other hand, philosophers who argue that our knowledge of disease is entirely discovered find the view that “patients died of what their doctors said they died of” (Cunningham 2002) equally unacceptable, because it entails that no doctor can ever be mistaken. Doctors have a responsibility to try to care as well as they can for their patients. If any opinion about how to care for a patient is as valid as any other opinion, the whole project of trying to offer high quality care is undercut. As Boorse puts it, “if we abstract away from all questions of truth and falsity, then cows jump over the moon” (Boorse 1997: 77).

In my view, the concerns of both sides in this discussion are valid. What should be sought is an intermediate position that can show how knowledge of disease the contingent result of social and historical processes, without collapsing into an extreme form of relativism. In an effort to reveal how it is possible to produce
knowledge of disease that is historically contingent and that tracks the way to
world is, I have carried out a more detailed study of the transition from the first to
the second stage of my longue durée history. This has involved looking most
closely at the work of the English physician James Hope (1801-1841), and the
Scottish physician James Mackenzie (1853-1925). Having done this, the
objective of this chapter is to draw on this material to make such an intermediate
position available.

My starting point is to review and compare two key claims made by Hope and
Mackenzie about the diagnostic value of the stethoscope (Section 1). These two
doctors held opposing points of view about the diagnostic value of the
stethoscope, but both believed that the truth of their position was an undeniable
fact. However, I will argue that, on closer inspection, neither of these points of
view were the result of brute observation. Rather both of these doctors had strong
intuitions about what heart disease was, and used these intuitions to produce
facts about heart disease.

Following this, I draw on the work of some conventionalist philosophers of science
to argue that conventionalism can provide a good account of how Hope and
Mackenzie produced their facts about heart disease. There are many similarities
differences between the various philosophers who have described themselves,
or who have been described, as conventionalists. For my purposes I take a
philosopher to be a conventionalist if they hold the following three views, which
are commonly attributed to conventionalists (Rescorla 2015; Bland 2011; Ben-
Menahem 2006; Ubel 1998-1999; Giedymin 1982). Firstly, some apparently
simple empirical claims are, despite appearances, not actually simple empirical
claims at all. Rather, such claims are accepted by stipulation, and function as
“disguised definitions” (Poincaré 2001a: 45). These claims are the conventions
that give conventionalism its name. Secondly, these conventions are used to *produce* further claims, variously referred to as “facts” (Fleck 1979: 40), or “laws” (Poincaré 2001b: 332). These further claims are not simply stipulated, and their acceptance depends directly on the way the world is. Thirdly, different conventions can legitimately be accepted by different groups of historical actors. Conventions can be changed. With this view of conventionalism in mind, I describe the work of three conventionalist philosophers, and use their insights to begin to develop a philosophical position that is at once fully historically contingent and constrained by the way the natural world is.

To begin, I review a suggestion made by Tristram Engelhardt, which I read as a conventionalist position (section 2). Engelhardt (1996) advocates a Kant-inspired epistemology that has what he calls a “Hegelian accent”. Engelhardt agrees with Kant in that the objects that appear to us conform to our intuitions, but also want to emphasise that these intuitions can change over time (a view Engelhardt attributes to Hegel). I think this view is nicely captured by Kant’s metaphor of a judge and a witness (2007: 16), which captures nicely how our intuitions (represented by the line of questioning taken by the judge) and the natural world (represented by the witness) interact to produce knowledge. This is particularly the case if we allow the judge to change the line of questioning over time, thus making the production of knowledge historically contingent. I make use of this metaphor to throw light on how Hope and Mackenzie are producing knowledge of heart disease.

Even if Engelhardt’s views are valuable, they are underdeveloped (Lennox 1997). In particular, how the notion that our intuitions can change over time might be squared with a Kantian viewpoint has not been fleshed out. Rather than trying to speculate about how Engelhardt imagines Kant’s work can be combined with
Hegel’s in this context, it is useful to draw on the work of Henri Poincaré (section 3). Poincaré argued, as Kant had done, that scientific facts and laws could only be produced after certain intuitions (which he called “principles” or “conventions”) had been adopted (Poincaré 2001b: 331; 2001a: 45). But Poincaré denied that a certain set of intuitions were forced upon the human mind, as he argued Kant had believed. Instead, Poincaré argued that it was possible to adopt different conventions, and thus produce different sets of scientific facts. As I will show, this view can be used to understand how Hope and Mackenzie produced different facts to one another.

Again, even though Poincaré’s work is valuable, it is still problematic. A particular problem for Poincaré is that he saw conventions as things that were inserted into what he called “primitive” facts (which he described as relations between sense data) to produce principles and scientific facts. Despite this, Poincaré could not provide an account of how these so called primitive facts were produced. The difficulty this presents can be resolved by appealing to the conventionalism of Ludwik Fleck (section 4). Rather than speaking of “principles” and “laws”, Fleck discussed his conventionalism in terms of “active” and “passive” associations (Fleck 1979: 10). Like Poincaré, Fleck argued that facts could be split into active (conventional) and passive (law-like) elements (section 4.2). But, when Fleck spoke of splitting a fact, he was not referring to a primitive fact, but rather to the scientific facts themselves. Indeed, Fleck did not believe that there were any truly primitive facts. This move not only relieves Fleck of the burden of having to give an account of primitive laws before he can give an account of active associations, but also allows him to incorporate things like training, skills and practices into his understanding of an active association (section 4.3). This understanding of an active association fits much more closely with the sort of intuitions and
judgements that Hope and Mackenzie were making as they produced facts, than does Poincaré’s account of primitive laws and sense data.

I am searching for an epistemology that can explain how facts can be at once invented and discovered. By the end of this chapter I will have provided good reasons to hope that Fleck’s conventionalism is such an epistemology. The focus of this chapter is on how the facts produced by Hope and Mackenzie can be understood as invented. How they are at the same time discovered is the focus of the next chapter. Even so, Fleck’s epistemology is in tension with the way I have discussed “discovery” thus far in this thesis. In chapter 3, I identified discovery with a limitation of the freedom to choose how things are. This is because discovery, as commonly understood, is to find something out about the way the natural ‘world is in itself’, which is not up to the discoverer. I have referred to the natural world many times in this thesis, in the hope of finding some way the way the natural world is constrains the production of knowledge. I had hoped to develop an account of discovery that can show how it is possible for our knowledge to capture some aspect of the way the natural world is, to grab hold of the Kantian noumenon and wrestle a chunk of it free. If Fleck’s epistemology is accepted, however, this hope must die. Fleck denied the possibility of knowing the way the natural world is. He even denied that claims about the way the natural world is are intelligible. He explained that whenever he referred to “objective reality”, he was only using a convenient figure of speech, about something that cannot be understood. Any references I make to the natural world henceforth should be understood in this way, and I make no attempt to develop an account of the natural world in this chapter. By the end of this chapter, I will have no more use for the notion of the natural world. How a sense of discovery can still be
produced without an account of the ‘world in itself’ will be discussed in the next chapter.

By drawing out Fleck’s conventionalism (section 4), through discussing Poincaré’s conventionalism (section 3) and Engelhardt’s appeal to Kant (section 2), I develop an epistemological account of how Hope and Mackenzie produced such different facts about the diagnostic value of the stethoscope (section 1).

1 – Producing facts about the diagnostic value of the stethoscope

In the last sections, the transition from James Hope’s way of understanding heart disease to James Mackenzie’s way was described in some detail. Here I want to draw attention to one important area of disagreement between these two doctors: their views on the diagnostic value of auscultation.

As described in the previous sections, Hope was very much of the opinion that auscultation was a powerful diagnostic tool, which allowed doctors to identify and distinguish many different sorts of valvular disease. Recall that according to Hope “[t]he accession of auscultation to the other means of diagnosis has rendered it possible to distinguish valvular disease, both in general and very often in particular, with almost complete certainty” (Hope 1833: 336).

Furthermore, Hope believed that the diagnostic power of auscultation was a brute fact, one which any reasonable person would have to accept if they only attended carefully enough to the way that the world is. As Hope’s wife put it, he “never spoke or argued in favour of auscultation, but allowed the facts to speak for themselves” (Mrs Hope 1842: 74).

In contrast to this, Mackenzie believed that auscultation was not a powerful diagnostic tool. Indeed, recall that, as far as Mackenzie was concerned, the results of auscultation had misled the doctors, and he thought that it was
“questionable whether the discovery of auscultation has so far done more harm than good” (Mackenzie 1913: 7).

This is a striking claim. The stethoscope is seen today as emblematic of medical practice in general, and cardiology in particular. According to Hope (1839, letter to Alexander Hannay), by the late 1830s the stethoscope had come to be regarded as “one of the greatest discoveries” of medical practice, and the art of auscultation was “cultivated with avidity by all classes of [the medical] profession”. Mackenzie recalled that his training had instilled in him similar beliefs about the diagnostic value of auscultation (Mackenzie 1915: 807). And yet, Mackenzie, a man who was seen by the subsequent generation of British physicians as the father of modern cardiology (Lawrence 1985), argued forcefully and repeatedly that the introduction of the stethoscope had misled the medical profession, harmed patients, and hindered progress in medicine (Mackenzie 1911a: 794; 1913: 6-7; 1916: 30; 1919: 181-182). What is more, Mackenzie also believed that the truth of his view about auscultation was also a brute fact, and anyone that thought otherwise simply was not attending carefully enough to the way the world is. According to Mackenzie, the view that “the sounds of a healthy heart must be clearly struck and free from murmurs is not based on accurate observation” (1916: 101).

So we have two doctors, each with considerable experience of clinical practice, who disagree completely about the diagnostic value of auscultation. Furthermore, they both believe that the truth of their view can be established by brute observation. For Hope the facts “speak for themselves”, and yet for Mackenzie Hope’s conclusion “is not based on accurate observation”. Each of these doctors believed that anyone who disagreed with them just had not looked carefully enough at the way the world is.
But is this the case? Do the facts “speak for themselves”? A closer look at how these two doctors reached their conclusions about the diagnostic power of auscultation reveals that their views (despite what they thought) are not brute facts. Rather, both doctors had strong *intuitions* about what heart disease was, and used these intuitions to produce facts about heart disease.

Consider Hope’s case. Hope found that he could predict which patients had heart disease by using the stethoscope to listen for murmurs. Hope checked that the patients did indeed have heart disease at post-mortem, by opening up the hearts of these patients and looking to see if valvular lesions were present. Accepting that the presence of murmurs and valvular lesions were simple and uncontroversial observations that all researchers would agree on\(^{92}\), we might well grant with Hope that the association between valvular lesions and heart murmurs “spoke for itself”. Disagreement about whether or not murmurs and valvular lesions were associated in the wayHope said they were could only be resolved by attending closely to the results of physical and post-mortem examinations, just as Hope invited people to do. But this is not the association that Hope claimed was a brute fact. Hope did not claim that the association between heart *lesions* and heart murmurs was a brute fact, he claimed that the association between heart *disease* and heart murmurs was a brute fact. Even if we accept that the former claim is true, Hope would only be entitled to make this latter claim if he could establish the association between heart disease and valvular lesions of the heart. But Hope does not discuss why he thinks heart disease is associated with valvular lesions of the heart. As far as Hope was concerned, to have a valvular lesion *simply was* to have a heart disease. In other words, Hope had a strong

\(^{92}\) I do not accept this, but grant it for the purposes of this discussion.
intuition that heart disease and valvular lesions of the heart were connected, which was so strong that he did not question it or require empirical evidence for it.

A similar situation becomes apparent when we look closely at Mackenzie’s work. Mackenzie found that patients with heart murmurs frequently did not have heart disease. Mackenzie found that this was the case because many patients with heart murmurs never became ill, despite living long and physically strenuous lives. In other words, he found that people with heart murmurs often did not have a poor prognosis. Accepting that people who had a poor prognosis can be simply and uncontroversially identified\(^{93}\), we might well grant with Mackenzie that the association of heart murmurs and a poor prognosis “was not based on accurate observation”. Disagreement about whether or not most with heart murmurs also had a poor prognosis could only be resolved by watching patients with murmurs for long periods of time, just as Mackenzie invited people to do. But the answer to this question is not what Mackenzie thought was a brute fact. Mackenzie did not claim that the lack of association between a poor prognosis and heart murmurs was a brute fact. Rather, he claimed that the lack of association between heart disease and heart murmurs was a brute fact. Even if we accept that the former claim is true, Mackenzie would only be entitled to make the latter claim if he could establish the association between heart disease and a poor prognosis. But this association was not a question of evidence for Mackenzie, he just asserted that a person who did not have a poor prognosis simply cannot have had heart disease. In other words, Mackenzie had a strong intuition that heart disease and poor prognoses were connected, which was so strong that he

\(^{93}\) Again, I am not claiming that this is the case, but need to grant that it is for the purposes of discussion.
thought knowledge of prognosis was “the coping stone on the whole system of medicine” and that “all other branches are contributory to it” (Mackenzie 1919: 148).

So both of these doctors had very strong, and yet very different, intuitions about what heart disease was. Furthermore, they produced different facts about heart disease because they had different intuitions about what heart disease was. Hope observed that heart murmurs were associated with valvular lesions found post mortem. Because he had a strong intuition that patients with valvular lesions had heart disease, he found that patients with murmurs had heart disease. Although Mackenzie disagreed with Hope in that he did not think that heart murmurs were closely associated with heart disease, he nevertheless agreed with Hope in many important respects. As discussed above, Mackenzie was confident that narrowed and incompetent valves could be identified by the murmurs they produced, just as Hope was (1916: 22; 1913: 329; 1919: 149). The differences between the views of these doctors did not arise because of differences of opinion about the association of murmurs and valvular lesions. Rather, they came to these quite different conclusions because they had different intuitions about what heart disease was. Hope connected heart disease with valvular lesions, whereas Mackenzie did not, so Hope thought murmurs were associated with heart disease, whereas Mackenzie did not. These doctors had different intuitions about what heart disease was, and therefore produced different facts about heart disease.

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94 The use of the term “lesion” to describe changes to a valve that would produce a murmur is not appropriate when applied to Mackenzie’s thinking, as the term lesion implies pathology, and pathology implies the presence of disease, and Mackenzie did not think of incompetent and narrowed valves as necessarily diseased. Nevertheless, he would have agreed with Hope that murmurs could be used to detect narrowed and incompetent valves.
The role that intuition plays here in the production of facts about disease presents intriguing possibilities for explaining how knowledge of disease can be historically contingent. If knowledge of disease depends upon intuitions, and intuitions can change over time, then our knowledge of disease can change over time. The recognition of this role for intuition should please those philosophers and historians who argue that our knowledge is the contingent result of social and historical processes (Engelhardt 1996; Kingma 2013; Cunningham 2002; Wilson 2000). And yet when looking for a philosophical position that incorporates this role for intuition the role that the natural world plays in constraining our knowledge must not be forgotten.

2 – Engelhardt’s appeal to Kant
In the introduction I claimed that I have been unable to find any contemporary philosophers of medicine who have described a way in which the invented and the discovered aspects of medical knowledge might be integrated. While this is true, an important suggestion as to how such a position might be developed has been made by Tristram Engelhardt (1996: 226). Engelhardt has suggested that a philosophical position inspired by Immanuel Kant’s views may be applicable to medicine, and I argue that this suggestion is well worth developing.

Engelhardt is often presented as an extremely strong sort of normativist, one that “fails to account for the common view that there is more to the term ‘disease’ than just a statement of our values” (Ereshefsky 2009: 224; see also van der Steen and Thung 1988: 94; Lennox 1997: 76-77; Boorse 1997; Kingma 2013: 42). This reading of Engelhardt is well supported, not least by Engelhardt’s own arguments that conditions like masturbation, drapetomania and dysesthesia aethiopis were “authentic” diseases (Engelhardt 1974). Indeed, Engelhardt has endorsed the view that “medicine is ideology” (1976: 256, 262). But, in a less radical mode,
Engelhardt can also be found to endorse positions that do see a role for the natural world in the production of medical knowledge. For instance, Engelhardt has argued that disease classifications are “as much invented as discovered” (1985a: 67). He has also emphasised that disease classifications “are not arbitrary within any societal and cultural context” (1984: 33). Engelhardt recognises that although “[p]ortrayals of reality are cultural products”, there are still “constraints placed upon us by the given character of objects” (1996: 226). These statements imply that disease classifications are not entirely invented and artificial, and that there is some role for the natural world in their production. Furthermore, sometimes Engelhardt’s views on the production of medical knowledge have a distinctly Kantian flavour: “objects appear to us through our concepts and in terms of the conditions of our experience” (Engelhardt 1996: 226).

Engelhardt appeals to Kant’s work directly, and suggests that Kantian insights can used to understand how medical knowledge is produced. What Engelhardt thinks Kant got correct was that “we do not know things as they are in themselves, as they are totally apart from our categories, but only as they are given to us through our categories of understanding” (Engelhardt 1996: 228, footnote 1). What Engelhardt is referring to here is Kant’s “Copernican revolution in thought” (Ledermann 1970: 44)95. Rather than trying to produce intuitions that go beyond direct experience by observing the objects that appear to us, Kant argued that we should realise that the objects that appear to us are partly the result of our intuitions (Kant 2007: 18). Engelhardt applies this insight to medical epistemology, suggesting that our knowledge of disease categories is not

95 I do not agree with Ledermann’s account of Kant’s Copernican revolution in thought, but he does identify it and he tries to apply it to medicine.
produced by observing the characteristics of diseased patients as they appear to us, but rather the characteristics of diseased patients appear to us because of our intuitions about disease categories.

However, Engelhardt disagrees with Kant in that he does not hold that our intuitions are timeless and unchanging, and this is why he thinks that Kant’s views require a Hegelian accent (Engelhardt 1996: 228, footnote 1). “Hegel recognized, as Kant did not, that categories of knowledge are historical” (Engelhardt 1996: 228, footnote 1). Here Engelhardt emphasises that the intuitions used to produce medical knowledge can change over time, and thus how the natural world appears to us can also change over time. Engelhardt consequently argues that our knowledge of medicine is historically contingent, and conditioned by social and cultural values.

I think that this Kant-inspired approach to the production of medical knowledge could be very helpful, as it chimes with what Hope and Mackenzie do as they produce medical knowledge. Neither doctor presents evidence to justify their most fundamental intuitions about disease. Hope does not try to justify his intuition that to have a valvular lesion was to have a heart disease by collecting cases with valvular lesions and looking to see if they had heart diseases. Mackenzie does not try to justify his intuition that a person with a heart disease must have a poor prognosis by collecting together patients with heart diseases and looking to see if they had a poor prognosis. If either doctor tried to do these things, they would encounter the same problem as doctors today do when trying to measure diagnostic accuracy, as without their intuitions about what heart
disease is they would be unable to look and see what its characteristics were. The presence of these strong intuitions means that this problem does not arise\(^\text{96}\).

Engelhardt’s Kant-inspired suggestions are intriguing, but underdeveloped. In particular, it is not clear from Engelhardt’s work how such a Kant-inspired epistemology can constrain our knowledge and show how we are not free to believe anything we want. Consider the example of heart disease, which Engelhardt uses to make his point about the Kantian nature of how disease is perceived:

“Consider the transformation of experienced reality accomplished by the diagnosis of heart disease. A slight shortness of breath or the swelling of the ankles after a long day of work becomes a sign of disease. Sleeping on two pillows is no longer an innocent occurrence, but a possible stigma of a deadly disease. The individual’s view of life is changed by a set of expectations regarding the dangerousness of heart disease and the possibility of an early death” (Engelhardt 1996: 189-190).

Engelhardt argues that our intuitions about heart disease make us see shortness of breath, ankle swelling and the inability to sleep when lying flat on our backs as signs of disease, as legitimate things to worry about, and a good reason to fear an early death. Even if this is the case, it is not clear from this example how our knowledge of heart disease is constrained by the way the world is. Wouldn’t Engelhardt’s (1996: 190) claim that we “see the world through social, scientific, and value expectations” be just as true if these expectations were incorrect? Consider Engelhardt’s (1974) example of masturbation, as discussed in a previous chapter. The intuitions of nineteenth century doctors about the disease masturbation made them see the bent legs of a rachitic patient, the seizures of an epileptic patient and the behaviour of an insane patient as signs of excessive

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\(^{96}\text{Even so, this approach to medical epistemology also throws up difficulties of its own, like why should we trust these intuitions?}\)
masturbation. It led them to believe that genital mutilation could relieve the suffering of these patients. As I argued in a previous chapter, I consider these beliefs about masturbation to be false, and the methods of managing patients associated with it to be abusive. Engelhardt’s work does not clarify the difference between intuitions that allow the world to be experienced in certain ways (as might be the case for the heart disease example above) and beliefs that are simply mistaken (as I argue is the case for the masturbation example). Even if it is possible to escape the problems associated with the search for universal truth by appealing to intuitions, how is what we accept as true constrained by the way the natural world is on this view?

I agree with Lennox (1997), who argues that Engelhardt’s appeal to Kant does not show clearly how our knowledge can be socially and culturally conditioned without collapsing into an extreme form of relativism. I also agree with van der Steen and Thung, who argue that “Engelhardt pays so much attention to the themes of values and context in his discussion of clinical medicine, that the role of science becomes obscure” (van der Steen and Thung 1988: 94). Nevertheless, I think that by developing Engelhardt’s Kant-inspired suggestion it will be possible to produce medical knowledge that is at once invented and discovered. In particular, I would like to appeal to a metaphor used by Kant to help clarify what it is that I find helpful about Kant’s insights in this context. In the preface to the second edition of his critique of pure reason, Kant used a metaphor of a judge and a witness to clarify his position:

“Reason, holding in one hand its principles, according to which alone concordant appearances can be admitted to count as laws of nature, and in the other hand experiments which it has devised according to these principles, must approach nature in order to be taught by it. It must not, however, do so in the manner of a pupil who agrees with everything the teacher says, but of an appointed judge, who compels the witnesses to answer the questions which he himself has phrased” (Kant 2007: 16).
The natural world, here represented by the witness, does not simply volunteer testimony to the court. Unless asked a question, the witness will remain silent. The judge, who represents us as knowers, can compel the witness to answer questions, but these questions must first be composed. Decisions about how to compose the questions are made by the judge, by us as knowers, and therefore are invented. How these questions are composed certainly does affect the answers given to the court. Furthermore, this affect is not limited to the topic of the question. How the question is phrased has an effect in framing the answer to the question, affecting the answer in a very profound way.

Notice, however, that having full control over the questions that are asked does not give the judge full control over the answers given by the witness. The witness, representing the natural world, does play a very important role in the production of testimony. Analogously, nature has a very important role to play in the production of knowledge. But notice that the witness does not have full control over the answers given, as these are also influenced by the questions asked. Analogously, the way the natural world is does not fully determine the knowledge we gain from the world. Indeed, knowledge of how the world is in itself is not available to us as knowers, in the same way as knowledge of the events that the witness is describing is unavailable to the court. “What things may be in themselves we do not know, nor need we care to know, because, after all, a thing can never come before me otherwise than as an appearance” (Kant 2007: 274; see also Engelhardt 1996: 228, footnote 1).

So, testimony of the witness is a product of the interaction between the questioning of the judge (the artificial and invented), and the answers of the witness (the natural and discovered). The way the questions are phrased has a
profound effect on the answers given, but does not fully determine the answers
given. Without both the judge and the witness, there would be no testimony. Even
though both have a necessary role to play in the production of testimony, neither
fully determines what the testimony will be. Engelhardt’s suggestion of a Kant-
inspired account of how medical knowledge is produced I think points the way to
showing how discovery and invention are integrated.

However, Engelhardt’s Kant-inspired epistemology is underdeveloped. The thing
that is attractive about a Kant-inspired epistemology is that it allows our intuitions
(the judge) and the natural world (the witness) to interact. It was precisely this
interaction that allows for our knowledge to be historically contingent and
constrained by the way the natural world is. To feel the constraint of the natural
world, I need to be able to identify the voice of the witness, and I cannot even tell
what is supposed to be the voice of the witness from what is supposed to be the
voice of the judge in Engelhardt’s examples. Furthermore, if I cannot distinguish
the intuitions of researchers from the constraint of the natural world, then I cannot
see from Engelhardt’s examples how these intuitions change over time. Rather
than trying to speculate about how Engelhardt might achieve these goals by
combining Kant’s work with Hegel’s, it is useful to draw on the work of Henri
Poincaré.

3 – Poincaré’s conventionalism applied to medicine
On Kant’s view, objects appear to us as a result of our intuitions. Poincaré agreed
with this, and argued that certain intuitions needed be accepted before scientific
facts could be produced. Poincaré focused particularly on intuitions about the
axioms of geometry and Newton’s laws of motion, which he argued needed to be
accepted before scientific facts about the movement of objects and forces could
be articulated. So he argued, for instance, that Newton’s first law, contrary to first appearance, was not a fact that could be shown to be true by observation:

“Is, then, the principle of inertia, which is not an a priori truth, an experimental fact? Have there ever been experiments on bodies acted on by no forces? And, if so, how did we know that no forces were acting?” (Poincaré 2001a: 75).

In order to determine whether or not there are forces acting on a body by looking at how that body is moving, how a body moves when under the influence of no force needs to be known. So Poincaré argued, with Kant, that certain intuitions about forces needed to be accepted before the presence of forces could be detected.

Again, the acceptance of an intuition can be understood as a way to escape problems associated with the search for universal and timeless truth about forces. Say we want to measure forces, but cannot because we cannot detect them directly without laws of mechanics. So we must infer the presence of a force by the way that objects move. To do this we must know the relationship between the forces applied to the object and how they move, and we cannot discover this empirically because to do this we would need be able to detect both forces and movement, and forces are the thing we are trying to detect. Poincaré and Kant both argued that the adoption of intuitions about force, like laws of mechanics, was what allowed researchers to observe which forces acted on which bodies.

However, for Kant we had no choice but to accept certain intuitions, as these were supposedly part of the conceptual apparatus that humans used to perceive the world. Kant argued that all humans intuitively accepted Euclidean geometry
and the laws of mechanics Kant described. Poincaré disagreed with this, arguing that these intuitions could not be synthetic *a priori* intuitions because it was possible for people to accept *other* intuitions instead, and use these to perceive the world. As *synthetic a priori* intuitions were supposedly fixed for all human beings, finding that alternative intuitions could be employed meant that these intuitions could not be *synthetic a priori*.

In particular, Poincaré argued that the development of non-Euclidean geometries demonstrated that our intuitions about the axioms of geometry could not be *synthetic a priori* intuitions, as there were alternatives that could be conceived of and used:

“[W]hat is the nature of geometrical axioms? Are they *synthetic a priori* intuitions as Kant affirmed? They would then be imposed upon us with such a force that we could not conceive of the contrary proposition, nor could we build upon it a theoretical edifice. There would be no non-Euclidean geometry” (Poincaré 2001: 44).

Similarly, Poincaré argued that the laws of mechanics could not be *synthetic a priori* intuitions, because it was possible to conceive of other laws of mechanics and use these to make observations of the forces acting on bodies (Poincaré 2001 [1905]: 74-83). Indeed, Poincaré pointed out that past societies had actually adopted other intuitions about mechanics:

“A body under the action of no force can only move uniformly in a straight line. Is this a truth imposed on the mind *a priori*? If this be so, how is it that the Greeks ignored it? How could they have believed that motion ceases with the cause of motion? Or again, that every body, if there is nothing to prevent it, will move in a circle, the noblest of all forms of motion?” (Poincaré 2001a: 74).

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97 These laws of mechanics were not quite the same a Newton’s laws, but closely related to them (Kant 2004: 75-92).
So for Poincaré, some of the claims made by scientists (like the laws of mechanics) were, despite appearances, not themselves empirical claims about the world. Rather, these claims were intuitions that allowed empirical claims about the world to be made. And yet, Poincaré argued that these intuitions were not forced upon the human mind. Different societies at different times in history had adopted different intuitions, and the world has consequently appeared differently to each of these societies. As these claims were neither empirical claims nor synthetic a priori intuitions, Poincaré argued that they were principles or conventions that were accepted in order to produce scientific facts.

Poincaré argued that in order to produce scientific facts or laws it was always necessary to accept certain conventions. To illustrate this he considered the hypothetical example concerned with the detection of the forces which act on the stars (Poincaré 2001b: 331). Consider a group of astronomers who expect the stars to move in the way that they would if they were acted upon by the force of gravity alone. They consequently expected any particular star to move as if it was acted upon by the resultant force produced by the gravitational attraction of all the other stars in the sky; with each of the contributing forces being proportional to the product of the masses of the two stars in question and inversely proportional to the square of the distance between them. If the stars were observed to move in this way then Poincaré said they would be observed to “obey Newton’s law” of universal gravitation (Poincaré 2001b: 331). It should be noted that the observation that “the stars obey Newton’s law”, if understood as a simple collection of observations about the position of stars in the sky, need not be connected with the observation of forces that act on the stars. In Poincaré’s terminology, this set of observations amounted to a “primitive law” which described a relationship between these very simple observations or “facts in the
rough” (Poincaré 2001b: 331-332). Poincaré suggested that the proposition “the stars obey Newton’s law” could be split into two other more theoretically complex propositions involving gravitation, so that these very simple observations could be brought to bear on the issue of what forces were acting on the stars. One of these propositions would be a convention or principle that defined gravitation, and the other would be a scientific fact or law about the forces that acted on the stars. The convention was a definition and not vulnerable to experience, and the law was an empirical claim that could be tested using the set of simple observations:

“We can break up this proposition: (1) The stars obey Newton’s law, into two others; (2) gravitation obeys Newton’s law; (3) gravitation is the only force acting on the stars. In this case the proposition (2) is no longer anything but a definition and is beyond the test of experiment; but then it will be on proposition (3) that the check can be exercised” (Poincaré 2001b: 331).

Poincaré also gave this insight a general form. He argued that primitive laws could always be split into principles and laws by scientists:

“For the procedure is always the same. The primitive law enunciated a relation between two facts in the rough, \(A\) and \(B\); between these two crude facts is introduced an abstract intermediary \(C\), more or less fictitious (such was in the preceding example the impalpable entity, gravitation). And then we have a relation between \(A\) and \(C\) that we may suppose rigorous and which is the principle; and another between \(C\) and \(B\) which remains a law and subject to revision” (Poincaré 2001b: 332).

So Poincaré argued that for every scientific law (the relation between \(C\) and \(B\)) that scientists produced there was a corresponding convention or principle (the relation between \(A\) and \(C\)) which allowed for more primitive laws to be brought to

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98 Poincaré also used the position of a spot on the scale of a galvanometer (2001b: 325), and the perceived brightness of sunlight, as examples of facts in the rough (2001b: 322). By adopting certain conventions these facts in the rough become scientific facts. So for example the observation of the movement of the spot on the galvanometer will become the observation that a current is passing, and the observation that “it grows dark” becomes the observation of an eclipse.
bear on the law, and to test it. “Every law may be broken up into a principle and a law” (Poincaré 2001b: 332).

This analysis of scientific laws is directly applicable to the case studies of disease provided by Hope and Mackenzie. Hope found that there was a relationship between the presence of valvular lesions and heart murmurs. This relationship itself does not say anything about the presence of disease, and corresponds to a primitive law as described by Poincaré. So, using the general scheme introduced by Poincaré, valvular lesions correspond to $A$ and heart murmurs correspond to $B$. For Hope, this relationship between valvular lesions and heart murmurs reveals something about heart disease because of his intuition that to have a valvular lesion was to have a heart disease. So Hope inserted the abstract intermediary heart disease, corresponding to $C$, in-between valvular lesions ($A$) and heart murmurs ($B$). Hope used valvular lesions to define heart disease, and thus accepted the relationship between valvular lesions ($A$) and heart disease ($C$) as a convention or principle. In doing this, he made it possible to observe that there was an empirical relationship between heart disease ($C$) and heart murmurs ($B$), and thus argue that the stethoscope was a powerful diagnostic tool.

Mackenzie disagreed with this, and claimed that the stethoscope was not a powerful diagnostic tool. Mackenzie observed that the relationship between heart disease ($C$) and heart murmurs ($B$) was not as Hope said it was. But, he agreed that the relationship between valvular lesions ($A$) and heart murmurs ($B$) was as Hope said it was. The root of this disagreement was the differences in the conventions these two doctors used to define heart disease (the relation between $A$ and $C$). Whilst Hope connected valvular lesions ($A$) and heart disease ($C$) by convention, Mackenzie did not. Mackenzie connected a *poor prognosis* (which served as $A$ for Mackenzie) with heart disease ($C$), and thus produced the
empirical fact that heart disease was not associated with heart murmurs. The differences between the positions of these two doctors can be understood as the result of a difference over what was accepted as a convention or principle about heart disease.

Even though Poincaré’s conventionalism is a valuable aid for understanding how Hope and Mackenzie produced facts, some aspects of this epistemology are problematic. In particular, some aspects of Poincaré’s appeal to primitive laws do not chime with what Hope did. For Poincaré, scientific laws are produced by splitting primitive laws into conventions and scientific laws. This means that for Poincaré primitive laws (the relationship between A and B) must be produced before conventions (the relationship between A and C) and scientific laws (the relationship between C and B) are produced. This is problematic because primitive laws are never discussed or used in this way by Hope. Hope does not first discuss the relationship between valvular lesions (A) and murmurs (B), only then to discuss the relationship between heart disease (C) and these things. When Hope discussed valvular lesions he is talking about a heart disease. The connection between heart disease and valvular lesions (between A and C) was made before Hope found an association between valvular lesions and murmurs (A and B). Indeed, this link between lesions discovered post mortem and disease was an integral part of the research program in the Paris hospital system, which was already taking shape before Hope was even born\(^\text{99}\). Contrary to Poincaré’s

\(^{99}\) Even if Hope’s work does not chime with Poincaré’s account of how conventions and laws are produced, Mackenzie’s work may be a closer match. As discussed above, Mackenzie frequently made the claim that many patients with murmurs did not have a poor prognosis, thus making a claim equivalent to Poincaré’s primitive law. In his early career he also clearly associates the term disease with post mortem findings, just as Hope did. It is therefore possible that he first noticed the primitive law (which seems to have been something of a shock for him), and then produced the convention that heart disease should be associated with a poor prognosis to make this observation relevant to the diagnosis of heart disease, which is very much in line with Poincaré’s view.
claim (2001b: 332), primitive laws are not always produced before conventions and scientific laws.

A second problem arises from Poincaré’s claims about primitive laws. As has been discussed, for Poincaré scientific laws were variable: they could change depending on the conventions adopted by researchers. I have used this insight of Poincaré’s to explain the differences of opinion between Hope and Mackenzie about the diagnostic value of the stethoscope. Nevertheless, for reasons I will discuss in the following chapter, it was very important to Poincaré’s philosophy that some laws remain invariant. Poincaré argued that there must be some observed relationships that all observers would always agree upon (2001b: 336). As the possibility of adopting different conventions always made scientific laws variable, Poincaré sought these invariant laws where there were no conventions. As primitive laws were statements of the relations between “crude facts”, produced without appeal to conventions, Poincaré argued that universal agreement about the truth of primitive laws, and that these therefore were invariable:

“What now is the nature of this invariant it is easy to understand. The invariant laws are relations between the crude facts, while the relations between “scientific facts” remain always dependent on certain conventions” (Poincaré 2001b: 336).

This view that primitive laws are universally agreed upon and invariant is problematic, as can be seen by considering the case of the diagnostic value of the stethoscope. In this case I have identified the relationship between valvular lesions (A) and murmurs (B) as a primitive law, and therefore identified the presence of a valvular lesion or a murmur as a crude fact. As crude facts, the presence of valvular lesions and murmurs should be agreed upon by all, and so should the relationship between valvular lesions and murmurs. Now, even though
Hope and Mackenzie may well be in close agreement on these matters (see above), it is far from clear that this agreement would be shared with all observers. Ultimately, murmurs are just sounds heard with the stethoscope, along with the noises that are described as the “normal” sounds made by the heart. Learning to distinguish the different sounds the heart makes is a considerable achievement, and is a skill that takes medical students time and effort to master. As described in earlier chapters, Laennec tried to make a very fine grained correlation between a number of specific sounds heard with the stethoscope with specific changes found at post mortem examination (Duffin 1998: 190-194). Hope (1833), by contrast, thought that the same sort of lesion could produce quite different sounds, and so did not try to attach a specific sound to a specific diagnosis. The apparently simple act of correlation is much more complex that it may at first seem. The same may be said for the differentiation of normal from morbid anatomy. It is fanciful to think that anyone could open up a chest and describe the structures of the heart in the same way as an expert pathologist. Given this, it is problematic to claim that the supposedly primitive relation between valvular lesions and murmurs would be agreed on by all observers. As these observations are also variable, what makes a sound a murmur, and what makes a structure in the heart a valvular lesion, is perhaps also dependent on some conventions adopted by observers.

Poincaré was very aware of this issue. He explained quite clearly that, in his view, there was no strict distinction to be drawn between scientific facts and facts in the rough (2001b: 321-322). He argued that primitive laws were not truly primitive and free from conventions. Rather, they were simply claims produced using fewer conventions. The insertion of a convention into a more primitive law produced a more scientific law:
“There is no precise frontier between the fact in the rough and the scientific fact; it can only be said that the enunciation of a fact is *more crude*, or on the contrary, *more scientific* than such another” (Poincaré 2001b: 329).

So Poincaré described a nested hierarchy of claims, which become less primitive and more scientific as more and more conventions are added. He illustrated this with a toy example inspired by the observation of an eclipse (Poincaré 2001b: 322-323). The process begins with the most primitive observation “it grows dark, says the clown”. By adding conventions, the more scientific claim that “the eclipse happened at nine o’clock, says the astronomer” is produced. By adding still more conventions, the even more scientific claim this “results from the earth’s turning around the sun, says Galileo finally” is produced.

So perhaps the invariant, universally agreed upon statement that Poincaré sought can be found in the very primitive statement “it grows dark, says the clown”. And yet, Poincaré is very clear that even this is not invariant. He admits that the term “dark” may or may not refer to an infinite number of subtly different shades. Thus different observers may be referring to different shades when they use the term “dark” (Poincaré 2001b: 323-324). Poincaré argued that it was important to separate the claim “it grows dark” from the sense impression of darkness to which this claim referred. He also claimed that it was only in this sense impression that the invariant could be found.

“[I]t is necessary to distinguish between the impression of obscurity felt by one witnessing an eclipse, and the affirmation; it grows dark, which this impression extorts from him. In a sense it is the first which is the only true fact in the rough, and the second is already a sort of scientific fact” (Poincaré 2001b: 323).

However, in other places Poincaré expressed a different point of view about the invariance about sense impressions. Later on in the same text (2001b: 345),
Poincaré admitted that different observers may have different sense impressions when looking at the same object. He imagined a scenario where one person has the sensation Poincaré would call “red” when looking at a cherry, and the sensation Poincaré would call “green” when looking at a leaf. He admitted that it is possible that another observer may have such different sense impressions to the first that the sensation “green” is produced in this other observer when looking at a cherry, and the sensation “red” is produced when looking at a leaf. Given this, it seems even Poincaré would admit that trying to locate the invariant in sense impressions is problematic.

In a final effort to locate what is invariant, common to all and objective, Poincaré argued that what is invariant could be located in the relations between sensations (Poincaré 2001b: 345-347). He claimed that even if different observers experienced colours differently, they would always agree about which objects were the same colour. So even if one observer had the sensation Poincaré called “red” when looking at a cherry, whereas another had the sensation Poincaré called “green” when looking at a cherry, they each would agree that they had the same sensation when looking at a poppy. It was in this common experience of the same sensation when looking at a cherry and a poppy that Poincaré sought to locate the invariant.

But even this is problematic. If we grant that the relations between sensations are invariant and common to all, then the difference in sensation experienced by one person when he or she looks first at a cherry and then a leaf should be a common experience for all. The phenomenon of red/green colour blindness provides an instance of this not being the case, as people who see colour in this way do not see the differences that other people do, and see differences in colour that other
people do not. Poincaré’s attempt to locate what is invariant and common to all in the relations between sensations is also problematic.

Given this, where can Poincaré’s invariant facts and laws be located? If these truly primitive laws cannot be located, how can we begin the process of inserting conventions into primitive laws to produce more and more scientific laws?

So Poincaré’s conventionalism comes close to realizing Engelhardt’s suggestion of a Kant-inspired epistemology that is historically informed. Poincaré argued that certain conventions need to be adopted before scientific laws can be produced, just as the judge needs to ask questions before the witness can produce testimony in Kant’s metaphor. Poincaré also recognised, as Kant did not, that the conventions that are adopted can be changed, and that different societies in different points in history have adopted different sets of conventions. However, Poincaré’s conventionalism requires that primitive laws be articulated before conventions can be developed, and he fails to provide an account of these ultimately primitive laws. In other words, he argued that the witness does volunteer some primitive testimony to the court. The judge is supposed to add conventions to this primitive testimony to produce scientific testimony, which is more valuable to the court. But Poincaré could not say what this primitive testimony is, which makes his conventionalism problematic. Even if Poincaré’s conventionalism is problematic, the conventionalism of the Polish philosopher Ludwik Fleck fares much better.

4 - Ludwik Fleck’s Conventionalism
In my view, Fleck developed a very rich and satisfying form of conventionalism, which is capable of showing how medical knowledge can simultaneously be historically contingent and constrained by the way the world is. Given that I argue that Fleck’s conventionalism is superior to Engelhardt’s and Poincaré’s, it may
not be clear why I have discussed Fleck’s work alongside these other forms of conventionalism. The reason I have done this is because I am trying to show how Fleck can and should be read as a conventionalist. This is despite arguments, including those made by Fleck himself, which reject the view that Fleck was a conventionalist.

According to Patrick Heelan, Fleck thought that “scientific facts are recognised by being the passive response of the world (experience as ‘given’) to the active deployment of a historically changing system of perceptual inquiry” (Heelan 1986: 287), which is very much in line with the conventionalist reading of Fleck I am going to give. However, Heelan argues that Fleck was not a conventionalist for much the same reasons that Fleck did (Heelan 1986: 288). These objections will be addressed below along with Fleck’s (section 4.1).

This is not simply a terminological dispute. Fleck’s conventionalism is expressed in terms of active and passive associations. Fleck’s account of active and passive associations has been read in a variety of ways, not all of which are compatible with a conventionalist position.

For instance, Fleck’s passive element of knowledge has been misinterpreted by some to be a constraint on knowledge that functions independently of any social, historical or cultural factors (Toulmin 1986; Löwy 1988; Löwy 2004a). Stephen Toulmin suggested that for Fleck the passive elements of knowledge express “an empirical relationship that demands passive acceptance from all scientists working in that science regardless of their current theoretical views” (Toulmin 1986: 279; see also Löwy 1988 for an endorsement of Toulmin’s views of the passive element of knowledge). Similarly, Ilana Löwy equates the passive element of knowledge with the “hard residue of material reality” (Löwy 2004a: 283).
439), which functions independently of any social, historical and cultural factors to constrain our knowledge (Löwy 2004a: 439, 441). In my view, these ways of understanding the passive element of knowledge are a misreading of Fleck, who did not think that there were any elements of knowledge that could be considered independent of social, historical and cultural influences (Fleck 1979: 10, 50). And yet, this misreading of Fleck is understandable given that he occasionally makes reference to “objective reality” and “the object to be known” (Fleck 1979: 40, 38). Furthermore, as Löwy (2004a) points out, Fleck does argue that a fact cannot be “reconstructed in its objective entirety simply from historical factors, along with those of individual and collective psychology. Something inevitable, steadfast inexplicable by historical development is always left out of such attempts” (Fleck 1979: 79, 10).

Such passages seem to be leading scholars to attribute to Fleck a belief in ahistorical constraints on knowledge that he did not endorse. Fleck’s views on the passive element of knowledge are therefore tricky, and I think would be clarified by learning to read Fleck as a conventionalist. Fleck’s notion of the passive element of knowledge is therefore tricky and would benefit from clarification, which can be achieved by learning to read Fleck as a conventionalist.

Additionally, not all alternative readings of Fleck’s notion of the active and the passive can be interpreted as a straight-forward misreading, as Fleck is sometimes inconsistent in how he uses these terms. Sofia Siwecka (2011: 38-39) says that Fleck’s distinction between the active and the passive has to do with the perceived strength of the association between concepts. Siwecka (2011: 39) says that the particularly strong connections are the passive ones, and have the status of facts, whereas the active connections are weaker, and are merely considered as hypotheses. This is incompatible with a conventionalist reading of
Fleck, but may not simply be a misreading of him. Fleck claims that “[a]s any poet knows, a web of fantasy spun for long enough, always produces inevitable, “spontaneous” substantive and formal [and therefore passive] connections” (Fleck 1979: 101). Fleck therefore claims that a passive link can be produced simply by increasing the number of active associations until their mutual re-enforcement results in some claims that are particularly robust, which is similar to what Siwecka (2011) describes. In a similar vein, Jonathan Harwood claims that Fleck’s active elements of knowledge correspond to “narrowly institutionalised (thus contentious) knowledge claims”, whereas passive elements in knowledge correspond to “[b]roadly institutionalized (thus unexceptionable) knowledge claims” (Harwood 1986: 184). As Fleck uses these different, and possibly inconsistent, notions of the passive element of knowledge, it become particularly important for me to highlight the conventionalist reading of him that I think is useful.

Several scholars have also admitted that they find Fleck’s account of the active and passive elements of knowledge confusing and in need of further clarification\(^{100}\), which can be done by drawing out Fleck’s conventionalism.

Some scholars have already pointed to the conventionalist elements in Fleck’s work that I am going to highlight. Jerzy Giedymin and Thomas Schnelle have identified Fleck’s active and passive associations with conventionalism (Giedymin 1986: 186; Schnelle 1986b: 248-253). Wojciech Sady has identified similarities between Fleck, Kant and Poincaré. According to Sady “Fleck adopted Kant’s thesis on the active role of cognitive a priori: an empty mind would neither

\(^{100}\) Scholars who have stated that Fleck’s account of the active and passive elements of knowledge is in need of further clarification include Thomas Kuhn (1979: xi) and Stephen Toulmin (1986: 277). These difficulties will be addressed in depth in the next chapter, using a conventionalist framework developed in this chapter.
“Yet if syphilis can be defined in various ways, the definition selected still determines some conclusions. In this respect a certain amount of latitude appears to exist. It is only after the choice has been made that the associations produced by it are seen as necessary. As is well known this is a viewpoint held by the conventionalists. For instance it is a matter of free choice to define syphilis or the great pox simply as the carnal scourge. But this would necessarily imply the inclusion of gonorrhea, soft chancre and so on, as well as the abandonment of a therapeutic complex, and possibly of a rational method of treatment altogether” (Fleck 1979: 8).

So Fleck held that changing how syphilis is defined leads to the production of different facts about syphilis, such as about how syphilis responds to therapy. Fleck therefore agreed with what he saw as the conventionalist thesis that it is only possible to produce seemingly objective scientific facts (for instance about how syphilis could be treated) following the adoption of certain conventions (for instance about how syphilis was defined). He also thought that it was possible for
different conventions to be adopted by different societies at different points in history\textsuperscript{101}. And yet Fleck did object to what he saw as another conventionalist thesis, that the decision over which convention to adopt was a matter of \textit{free choice}.

As Jerzy Giedymin has pointed out, Fleck accused conventionalists of arguing that the choice of convention only depends upon the “whims of individual scientists” (Giedymin 1986: 186). The only philosopher that Fleck specifically referred to as a conventionalist was Ernst Mach (Fleck 1979: 8-9). Specifically referring to what he called Mach’s “formal point of view”, Fleck accused conventionalists of disregarding the strong social and cultural forces that prevent researchers adopting different conventions to their peers:

“First, the adherents of all these formal points of view pay far too little attention, if any, to the cultural-historical dependence of such an alleged epistemological choice – the alleged convention.....If these general cultural-historical relations in the history of knowledge are taken into account, conventionalism will be considerably restricted. Free rational choice will be replaced by the special conditions of which we have just spoken” (Fleck 1979: 9-10).

Fleck also pointed out that researchers often argued fiercely over definitions, which he thought provided further evidence that conventionalists had missed something important. Even if the use of different conventions were equivalent in some formal sense (as for instance Poincaré thought different geometrical conventions were), this did not mean that researchers would see them as equivalent in practice. Just because it was possible to use different sets of

\textsuperscript{101} Fleck also emphasised the importance of attending to the historical development of our knowledge to a much greater extent than did Poincaré. For instance, according to Fleck it “is nonsense to think that the history of cognition has as little to do with science as, for example, the history of the telephone with telephone conversations.....I believe that the concept of syphilis is unattainable except through a study of its history” (Fleck 1979: 21). How history functions in this way will be considered in the conclusion, and in chapter 10 in the appendix.
conventions to achieve the same result did not mean that researchers valued different sets of conventions equally:

"History shows that violent arguments can rage over the definition of concepts. This demonstrates quite independently of any utilitarian reasons just how little such conventions, which from the point of view of logic may seem equally possible, are in fact held to be of equal value" (Fleck 1979: 9).

However, Fleck’s criticisms of conventionalism are unfair. As Giedymin pointed out, Mach certainly “did not see the development of the conceptual apparatus of science in this “formal” way, as detached from and independent of the historico-cultural background” (Giedymin 1986: 186). So for instance, while Mach does argue that it would be possible to understand electricity as a potential function (as is the case for heat) rather than as a substance made up of electrons (Mach 1910: 44), this does not mean that this apparent choice is unaffected by historical and social factors. Mach argued that our current understanding of electricity is an accident of history, and the result of the relevant experiment being carried out in a certain historical order. The point of Mach’s argument is to reveal that what seems as a sure empirical fact (that electricity is a substance comprised of electrons) is actually the result of contingent historical and social processes. In this respect, Mach’s argument is very similar to Fleck’s.

If Fleck’s criticisms were directed at Poincaré, they may have been closer to the mark. It is true that Poincaré focused much less attention on describing the historical and social factors that constrained researchers’ choice of conventions than Fleck or Mach did. Nevertheless, it would be incorrect to say that Poincaré thought that a researcher’s choice of convention was completely free. For one thing, Poincaré thought that some conventions were more convenient than others. When faced with a surprising observation, Poincaré did argue that there
was a formal sense in which researchers were free either to abandon or to
preserve a convention. But in reality, Poincaré argued that one of these options
would almost always be far more convenient than the other, and the researchers’
freedom to make this choice would be restricted:

“The choice between the two attitudes is free, and is made from
considerations of convenience, though these considerations are most often
so strong that there remains practically little of this freedom” (Poincaré 2001:
331).

Furthermore, Poincaré thought that researchers were guided to these highly
efficient ways of expressing scientific facts by experimental work102. So, for
Poincaré, the choice of convention was not arbitrary, as it was strongly influenced
by the empirical results of experimental work.

“Are the laws of acceleration and the composition of forces only arbitrary
conventions? Conventions yes; arbitrary, no – they would be so if we lost
sight of the experiments which led the founders of the science to adopt
them, and which, imperfect as they were, were sufficient to justify their
adoption” (Poincaré 2001a: 86).

So Poincaré did not think that the choice of convention was arbitrary, and simply
left to the whim of the scientist. Nevertheless, Poincaré did equate “convenience”
with computational efficiency. Any set of conventions that permitted a more
efficient expression of facts was more convenient (Poincaré 2001a: 45).
Poincaré’s notion of convenience is thus similar to Mach’s notion of “the economy

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102 Poincaré argued that researchers would sometimes elevate a law that they felt was particularly well
confirmed empirically to the status of a principle, which would then be used to produce further laws
(Poincaré 2001b: 331-332). Fleck also described a similar process (the transformation of “passive”
associations into “active” ones) (Fleck 1979: 95, 101, 107), which is another similarity between Poincaré
and Fleck. However, Fleck described this process as fundamentally social, involving the circulation of
knowledge to different populations with different levels of expertise (Fleck 1979: 106-107). Poincaré
thought this process would still continue if researchers recognised these social factors and dismissed
them as unimportant, whereas Fleck made no such normative claim.
of thought”. Given this, it is possible that Fleck would have considered convenience to be a logical and formal property, as opposed to a cultural one.

Nevertheless, Poincaré did recognise cultural influences that constrained the choice of convention. Poincaré argued that once a relationship had been accepted as a convention or a principle, it was “no longer subject to the test of experiment” (2001b: 332). So if the relationship between the force of gravitation and Newton’s law of universal gravitation was accepted as a convention or principle, then observations that the stars do not obey Newton’s law would tend to be interpreted as showing that there were other forces acting in the sky, rather than as showing that the force of gravitation does not vary according to Newton’s law (Poincaré 2001b: 331). Poincaré described this process of accepting a convention as “unconscious nominalism” (2001b: 331): “nominalism” because it is stipulated that the force of gravity varies exactly according to Newton’s law, and “unconscious” because scientists are often not aware that any such stipulation has been made. Poincaré thought of conventions as “definitions in disguise” (2001a: 45). So for Poincaré, conventions were unconscious and unacknowledged agreements made by scientists that some claims were to be considered invulnerable to the test of experiment. It seems quite appropriate to describe such agreements as a culture, and not as a “free rational choice”.

Furthermore, Poincaré was aware that violent arguments had raged over the acceptance of certain conventions, and referred to the historical arguments over whether or not the earth rotated:

“Could Galileo and the Grand Inquisitor, to settle the matter, appeal to the witness of their senses? On the contrary, they were in accord about the appearances, and whatever had been the accumulated experiences, they would have remained in accord with regard to the appearances without ever agreeing on their interpretation. It was just on that account that they were
obliged to have recourse to procedures of discussion so unscientific” (Poincaré 2001b: 327).

So Poincaré was aware that scientists argued over which conventions to adopt, and did so for “unscientific” (and therefore psychological, historical, social and cultural) reasons. Poincaré was not trying to argue either that scientists thought that they were free to adopt different conventions, or that that should actually be free to do so arbitrarily in practice. Fleck’s allegation that conventionalists thought that scientists were free to choose any convention they pleased was unfounded. Indeed, several authors have highlighted the conventionalist elements in Fleck’s own work (Giedymin 1986: 184-186; Schnelle 1986b: 250; Sady 2001, 2012; Löwy 1988). Specifically, Fleck’s discussion of “active” and “passive” associations has been identified as being fundamentally conventionalist in nature (Giedymin 1986: 186; Schnelle 1986b: 250). Even so, discussions of Fleck’s work have not tended to focus on his conventionalism, instead focusing on the more social aspects of his epistemology. It is worth exploring the conventionalist aspects of Fleck’s epistemology in some detail, and comparing it to Poincaré’s work, so that the advantages of Fleck’s conventionalism can be seen. One advantage that Fleck’s work has over Poincaré is the attention Fleck paid to how conventions actually change historically. Another is that Fleck developed his conventionalism with medicine, as opposed to geometry and mechanics, in mind. But a key reason that Fleck’s work is so important is that he developed a species of conventionalism that did not require an account of crude facts or primitive laws.

103 For instance, Ilana Löwy (1988) offers a summary of the aspects of Fleck’s work that may be of interest to humanities researchers, and Fleck’s conventionalism is not discussed.
4.2 – Fleck’s conventionalism: active and passive associations

As mentioned above, an important feature of Fleck’s epistemology is what he called “active” and “passive” associations or linkages (Fleck 1979: 9-10, 40, 49-50, 79, 82-83, 95, 100-101, 141, 178). Fleck argued that all facts could be divided into these active and passive parts (Fleck 1979: 83). The passive part of a fact was what appeared as “real”, “objective” and “true” (Fleck 1979: 10). The active part of a fact were accepted and maintained for social, cultural, and historical reasons (Fleck 1979: 9-10). Fleck maintained that it was impossible to produce a factual claim from passive associations alone, and that it was always necessary to use active associations as well (1979: 49). Fleck summarised the relationship between active and passive elements of knowledge as follows:

“Cognition therefore means, primarily, to ascertain those results which must follow, given certain preconditions. The preconditions correspond to active linkages and constitute that portion of cognition belonging to the collective. The constrained results correspond to passive linkages and constitute that which is experienced as objective reality” (Fleck 1979: 40).

To illustrate what he meant by this, Fleck used the example of the fact that the atomic weight of hydrogen is 1.008 (Fleck 1979: 83, 95, 101). It is only possible to produce the passive (and seemingly “true”, “objective” and “real”) association between hydrogen and the atomic weight 1.008 following the acceptance of 16 as the atomic weight for oxygen:

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104 Fleck also referred to the active and passive “elements of knowledge” when discussing active and passive associations (1979: 79, 83, 95), and he uses these different terminologies interchangeably. An element of knowledge is the association between two things. As I will discuss below (section 4.3), Fleck associates a great variety of things together in his discussion of elements of knowledge. Objects, concepts, and practices are all candidate ‘things’ to be associated together in an element of knowledge. Fleck is also happy to treat elements of knowledge both as statements expressing the association of two things, and as this relationship itself. For example, he says that all laws can be divided into active and passive parts (treating these elements as statements resulting from the analysis of a law) (Fleck 1979: 83); and he also says that “the introduction of alcoholic extract” into the procedure for the Wassermann reaction was an active element of knowledge (treating the linkage between this changed procedure and the concept of the blood test for syphilis itself as the element of knowledge, regardless of whether this is expressed in a statement) (Fleck 1979: 79).
“The origin of the number 16 for the atomic weight of oxygen is almost
consciously conventional and arbitrary. But if 16 is assumed as the atomic
weight for O, oxygen, of necessity the atomic weight of H, hydrogen, will
inevitably be 1.008” (Fleck 1979: 83).

The atomic weight of oxygen is just stipulated, and is consequently exactly 16.
The association between oxygen and the atomic weight 16 is therefore chosen,
accepted and maintained by our society. This is why Fleck referred to
associations like these as “active”, as they are the result of something actors in a
society do. Passive associations, by contrast, were for Fleck the inevitable and
necessary consequence of accepting certain active associations.

Fleck applied this insight about active and passive associations to medicine, and
his main case study was of the disease syphilis. Fleck pointed out that for a long
period in Europe (from the late 1400s to the late 1700s) syphilis was understood
to be “the carnal scourge”, an affliction of the genitals sent by God as a
punishment for lust and unethical sexual intercourse (Fleck 1979: 2-3). As such,
patients with afflicted genitals were grouped together as suffering from the same
disease. Fleck argued that grouping these patients together was an active
association, and therefore the contingent result of social and historical forces.
Fleck also argued that it was impossible to produce passive associations, like
claims about whether or not syphilis/the carnal scourge could be treated with
mercury, without first making the active association of syphilis and the carnal
scourge:

“...In our history of syphilis the combination of all venereal disease under the
generic concept of the carnal scourge was thus an active association of the
phenomena, explained in terms of cultural history. In contrast, a restriction

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105 Fleck does not mean logical inevitability and necessity here, but rather inevitable in the sense that
we find we no longer have a choice in the matter even though alternatives are logical and conceivable
possibilities. This sort of inevitability is possibly well captured by saying that, given certain active
associations, the passive associations are forced upon us by the way the world is, but Fleck did not
approve of this formulation. This will be discussed further in the next chapter.
of the curative effect of mercury in the sentence quoted earlier asserting that “sometimes mercury does not cure the carnal scourge but makes it even worse” represents a passive association with respect to the act of cognition. It is of course evident that this passive association alone could not even be formulated if it were not for the concept of carnal scourge” (emphasis original, Fleck 1979: 10).

Fleck’s discussion of active and passive associations resembles Poincaré’s discussion of conventions and scientific laws. Active associations function in the same way as Poincaré’s conventions or principles do (see Sady 2001; 2012). Active associations, like conventions, need to be adopted before scientific laws can be formulated. Additionally, both Fleck and Poincaré claimed that laws can be split into active associations/conventions on the one hand, and passive associations/scientific laws on the other. However, there is an important difference between Poincaré and Fleck with regard to what exactly is being split. Recall that for Poincaré primitive laws (the relation between A and B) were split by inserting conventions (C) into them, in order to produce principles (the relation between A and C) and scientific laws (the relation between C and B). This left Poincaré with the problem of having to produce an account of what primitive laws are and how they are produced. In contrast to this, Fleck held that “[o]bservation without assumption, which is psychologically non-sense and logically a game, can therefore be dismissed” (1979: 92; also see Fleck 1986b: 59-78). In Poincaré’s terminology, Fleck believed that there were no truly crude or primitive facts, as he thought that all facts could be split into active (and conventional) and passive (law-like) components (Fleck 1979: 95). So when Fleck discussed the splitting of a fact or rule into active and passive components, he was speaking of splitting the scientific fact itself.

Consider the relation between the Wassermann reaction and syphilis, which was Fleck’s central object of study. Fleck regarded this relation as “an undoubted fact”
(1979: 97). The seemingly passive and objective relation between syphilis and the Wassermann reaction could only be produced once certain conventions, or active associations, were adopted. So, in Poincaré’s terminology, this fact was a scientific fact, as it required the acceptance of certain conventions to be produced. Fleck made an analysis of this fact, and identified what he thought were the active associations (or conventions) required for this fact to be produced. These active associations included the association of a procedure that used alcoholic extracts and the Wassermann test (Fleck 1979: 79), and the dissociation of “the agents causing gonorrhoea and soft chancre” and syphilis (Fleck 1979: 17). So Fleck identified the conventions used to produce scientific facts (the relations between A and C) by making analyses of scientific facts (the relations between B and C).

Therefore, when Fleck spoke of splitting a fact into its active and passive components (1979: 83)\textsuperscript{106}, he was not speaking of inserting a convention into a primitive or crude fact as Poincaré had done. Rather, he was referring to the identification of the active components of what seemed like an entirely passive and objective scientific fact. So rather than inserting a convention (C) into a primitive fact (the relation between A and B), Fleck always started his analysis with a scientific fact (the relation between B and C) and showed how it could not be produced without adopting certain conventions (the relation between A and C). On this view, conventions are not things that are inserted into primitive laws. Rather, they are the result of the analysis of scientific laws. Consequently, no account of primitive laws needs to be provided in Fleck’s conventionalism. This gives Fleck’s conventionalism a significant advantage over Poincaré’s.

\textsuperscript{106} Fleck spoke of “dividing” facts: “Every rule and chemical law can be divided into active and passive parts” (Fleck 1979: 83).
Fleck’s conventionalism is also easily applied to the work of Hope and Mackenzie. Recall that for Hope, the claim that heart disease could be accurately detected by auscultation was a fact that “spoke for itself”. A Fleckian analysis of this apparently objective fact would reveal that it could not be formulated without first accepting certain active associations; specifically the association between heart disease and lesions of the heart, and the association of heart murmurs with certain sounds heard at auscultation. According to Mackenzie, the view that “the sounds of a healthy heart must be clearly struck and free from murmurs is not based on accurate observation” (1916: 101). Again, a Fleckian analysis of this apparently objective fact would reveal that it could not even be formulated without first accepting other active associations; specifically that heart disease is associated with a poor prognosis. Fleck’s conventionalism allows these analyses of facts to be made without needing to appeal to crude facts.

4.3 – The active elements of knowledge as practices and skills
I have argued that Fleck’s conventionalism has advantages over Poincaré’s because Fleck does not require an account of crude facts. I should also point out that Fleck’s notion of an active association has further advantages over Poincaré’s notion of a principle. Specifically, Fleck’s notion of an active association also allowed him to associate a much wider variety of things than is possible in the case of Poincaré’s principles, and this gives Fleck the opportunity to incorporate skill, training, experience and tacit knowledge into active associations. Poincaré’s principles are a formal way of relating different sense observations; they are a form of correspondence rule. As such, all one is allowed to associate to make a principle are sense data, or other collections of sense data. In contrast to this, Fleck never described active associations as collections of sense data. This should not be surprising, as (as has been discussed) he
explicitly rejected the possibility of pure observation without assumption (Fleck 1979: 92). Instead he described the active element of knowledge as an *association* between many different sorts of things, but never between sense data. For instance, Fleck considered the association of syphilis (as the carnal scourge) with those patients that had any venereal disease to be an active association (1979: 10). So here we have the association of a disease with a particular clinical presentation designated as active. Similarly, at a different period in history, Fleck thought the *lack of association* between syphilis and the causative organisms of gonorrhoea and soft chancre to be an active association (1979: 17). So here we have the lack of association between a disease and certain microbes designated as active. Fleck also argued that the association between syphilis and recovery following mercury therapy was, at one point, an active association. So the association between a disease and an event or happening could be an active association for Fleck.

Furthermore, Fleck did not restrict himself to associations between diseases and other things or events. *Practices* and *skills* could also be incorporated into active associations. As discussed, Fleck argued that use of alcohol when preparing extracts in the Wassermann reaction was an active element of knowledge (1979: 79). The same could be said for the use of normal, healthy organs to prepare extracts instead of syphilitic ones (which Fleck discusses as well, 1979: 73, 78). So here we have the association of certain practices with the correct performance of an activity (the Wassermann reaction) being considered as active associations. Only by making these active associations could researchers become able to experience the passive association between a positive Wassermann reaction (a

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107 Fleck, with the Vienna circle in mind, also rejected the possibility of protocol sentences (1979: 89).
property of blood) and syphilis. But even this emphasis on the importance of practices is not enough for Fleck, who also highlights the importance of *training* and *skill* to making effective active associations.

“Weissmann’s reports about his reaction contain only the description of the relation between syphilis and a property of blood. But this is not the most important element. What is crucial is the *experience* acquired by him, by his pupils and in turn by theirs, in the practical application and effectiveness in serology. Without this *experience* both the Wassermann reaction and many other serological methods *would not have become reproducible and practical*…..anybody performing the Wassermann reaction on his own must first have acquired comprehensive experience before he can obtain reliable results. Only through this experience will he participate in the thought style, and it is experience alone that enables him to perceive the relation of syphilis and blood as a definite form” (Fleck 1979: 96).

Even if it could be done, for Fleck the description and organisation of raw sense data could never allow effective active associations to be made. To do this requires experience and skill, and these cannot be attained by reading reports. “But it is this individual experience, which can only be acquired personally, that yields the capacity for active and independent cognition” (Fleck 1979: 96). And yet, Fleck also emphasised that having any kind of experience often would not be adequate for the development of skill. Fleck argued that it was very difficult for a researcher to develop certain skills on their own (Fleck 1979: 22, 48, 90). To learn to see and act in certain highly skilful ways requires contact with other people who were already skilful:

“Scientific experience in particular derives from special conditions established by the history of ideas and by society. Traditional patterns of training are involved in this experience, which is, however, not accessible to everyone” (Fleck 1979: 48).

It is difficult to discuss whether Hope and Mackenzie would have considered the active associations they made as tacit and inexpressible, because neither of these researchers recognised that they were making active associations. Recall
that Hope, when claiming that auscultation was a powerful diagnostic tool, thought that he was simply allowing “facts to speak for themselves” (Mrs Hope 1842: 74). Similarly, Mackenzie argued that the claim that the sounds of a healthy heart must be clearly struck and free from murmurs was “not based on accurate observation” (1916: 101). In Fleck’s terms, both of these researchers thought that they were entirely passive in the face of these facts, and that their truth was simply dictated by the way the world just is. They are both unaware of the active associations required to produce these facts. Furthermore, as discussed in a previous chapter, Mackenzie was very critical of many of his colleagues who he said were “content with the finding of such gross changes” as the morbid anatomy uncovered at post mortem examination (Mackenzie 1913: 8). Therefore, Mackenzie was aware of and could criticise the association between disease and morbid anatomy found post mortem. Even if Mackenzie was not capable of detecting his own active associations, he was capable of detecting the active associations made by his colleagues, which were the same active associations made by Hope. Again, Fleck discusses precisely this phenomenon:

“To the unsophisticated research worker limited by his own thought style, any alien thought style appear like a free flight of fancy, because he can only that which is active and almost arbitrary about it. His own thought style, in contrast, appears imperative to him, because although he is conscious of his own passivity, he takes his own activity for granted. It becomes natural and, like breathing, almost unconscious, as a result of education and training as well as through his participation in the communication of thoughts within his collective” (Fleck 1979: 141).

Although Mackenzie did not view searching for associations between the results of the physical examination and those of the post mortem examination as “a free flight of fancy” (see Mackenzie 1919: 2), Fleck’s claim that it is easier for researchers to recognise the active associations made by others than it is for them to recognise their own is accurate in Mackenzie’s case.
Fleck was also correct to emphasise skill as an important aspect of active associations. Both Mackenzie and Hope recognised auscultation as a skill. As discussed in previous chapters, Hope felt the need to move to Paris for a year to become proficient in this skill, and Mackenzie recalled his disappointment on finding out that his hard-one proficiency at auscultation proved to be of little value. As Mackenzie did not associate heart disease with valvular lesions and murmurs, in order to investigate heart disease he needed to associate it with something else, as discussed in previous chapters, he associated heart disease with certain symptoms, such as chest pain, a feeling of oppression in the chest, and a diminished response to effort. Indeed, the ability to recognise these symptoms provides a good example of an embodied skill that is not reducible to an account of sense data. For instance, as discussed in earlier chapters, a diminished response to effort referred to a state where the patient became prematurely breathless on exertion. Mackenzie argued that this state could not be recognised simply by observing the degree of breathlessness of the patient. It also required the integration of knowledge of the degree of exertion the patient had undergone, and of a judgement about whether the state of the patient was appropriate for that particular patient. Thus, the recognition of a diminished response to effort was a complex act of judgement involving input from both the patient and the doctor, which would be difficult to capture in writing.

Mackenzie explicitly reflected on the embodied nature of the clinical skills that allows a physician to obtain reliable and significant information from patients. This was particularly the case when asking patients to describe their symptoms in the early stages of disease, which (as has been discussed in earlier chapters) Mackenzie argued were of paramount importance to diagnostics:
“[I]t must be understood that the signs of disease in the early stages are the most difficult to detect and to understand. They are mostly subjective phenomena, and it requires great experience and skill to obtain from a patient a coherent description of his sensations” (Mackenzie 1919: 26).

Mackenzie was also adamant that it was impossible to learn about disease without coming into direct contact with patients. Learning about disease in an abstract way, in an environment like a laboratory where the scholar could gain no direct experience of patients, would never provide the appropriate experience to acquire the necessary skills to work as a doctor:

“The scholar is invariably sent to work in some place, as a laboratory, where he cannot get a personal contact with disease, whereas, if a proper view was taken of the subject, the first step in his training should be to acquire a knowledge of the disease he intends to investigate – a knowledge that can only be acquired by personal contact with individuals suffering from disease” (Mackenzie 1919: 53).

Additionally, Mackenzie recognised the important role that training played in permitting researchers to produce and recognise facts. “[I]t is absolutely necessary that the investigator have a long and careful training if he is to be capable of eliciting and understanding the facts” (Mackenzie 1916: 10, see also Mackenzie 1919: 26-27 for the same point in the case of training medical educators). Fleck’s views on the importance of skill, experience and training for making active associations have much in common with Mackenzie’s.

So, whereas Engelhardt only managed to gesture towards a Kant-inspired medical epistemology that is historically conditioned, Fleck’s conventionalism provides a well-developed and satisfying articulation of just such an epistemology. Returning to the Kantian metaphor of the judge and the witness, Fleck made an analysis of the passive facts that are produced by medical researchers, which are equivalent to the testimony produced in court. Fleck
concluded that these passive facts cannot be produced unless certain active associations are first adopted, which is equivalent to the judge having to formulate and ask questions before testimony can be produced. In opposition to Kant, however, Fleck argued that different sets of active associations can be adopted to produce different sets of passive facts. This is equivalent to the judge being able to change the form of the questions asked, and this is what gives Fleck’s conventionalism the historicity (or the “Hegelian accent”) that Engelhardt suggests is required. Furthermore, Fleck did not limit his notion of an active association to associations between articulated knowledge claims, but also included practices and skills amongst the active element of knowledge.

Even though Fleck’s views on knowledge production can be seen as related to Kant’s, care needs to be taken when describing Fleck’s position as Kant-inspired. Fleck says very little about Kant, and what he does have to say is highly critical (1979: 28, 47; see also Fleck 1990). As discussed, Fleck rejected Kant’s arguments that there is a fixed set of intuitions to which our knowledge conforms, just as Poincaré had done (Fleck 1979: 47). Fleck also objected to Kant’s appeal to the noumenon. Fleck felt very uncomfortable with the notion of the ‘world in itself’, and criticised Kant for making use of it (Fleck 1979: 28). He suggested that just as an absolute concept of “up” and “down” may have prevented people long ago from understanding how the earth could be round instead of flat, absolute concepts of “existence”, “reality” and “truth” were in his day preventing people from understanding how truth in part depends upon the knower (Fleck 1979: 28). This is perhaps unfair to Kant, who held that we cannot know anything about the noumenon (Engelhardt 1996: 228, footnote 1). Any problems that result from the claims that are made about the noumenon cannot therefore be blamed on Kant.
Nevertheless, Fleck still argued that making any use the concept of the world in itself was foolish, as no sense could be made of such a notion. “[A]ll insistence on the ‘essences and things’, like all search for the ‘thing in itself’, would not be natural science at all” (Fleck 1986: 56). Consequently, Fleck did not want to refer to the world in itself. It is for this reason that Fleck prefers to use a negative formulation when talking about the passive elements of knowledge. Fleck never says that the passive associations arise because of the influence of the natural world. Rather he says they arise from the influence of something that cannot be explained by social, historical, psychological and cultural factors alone. “Nonetheless, there are always other [passive] connections which are not explicable in terms either of psychology (both individual and collective) or of history” (Fleck 1979: 10, 79). Using this negative formulation, Fleck has no need to offer an account of the natural world. Fleck hardly ever discussed anything equivalent to the witness in the Kantian analogy, and he claimed that wherever he did speak of “objective reality” he only did so for grammatical reasons (Fleck 1990). Active associations are used by the judge to ask questions, and testimony is the passive result. Just like it is possible to hear an echo in complete darkness without having to consider how that sound is produced, Fleck noticed that our questions are answered without needing to consider what does the answering. We simply make active associations, and find that our attempts to make other associations are resisted.

Conclusion
Earlier chapters in this thesis have presented the challenge of showing how medical knowledge can at once be historically contingent and constrained by the way the natural world is. This chapter has begun to answer this challenge by showing how a species of conventionalism may be up to the task.
I began by discussing the facts about the value of auscultation in the diagnosis of heart disease that were produced by the doctors James Hope and James Mackenzie. Hope claimed that auscultation was a very powerful diagnostic tool, whilst Mackenzie claimed that it was not. I showed that each of these doctors believed that the facts they produced were the result of brute observation, and that the facts “speak for themselves”. Contrary to this, I argued that each doctor was only able to produce their fact about the diagnostic value of the stethoscope because they had first adopted certain very strong intuitions about heart disease. For Hope, this was the intuition that heart disease was associated with the presence of morbid anatomy found post mortem. For Mackenzie, this was the intuition that heart disease was associated with a poor prognosis. By adopting these intuitions, these doctors avoided the problems associated with the search for universal truth about how to accurately diagnose disease, discussed in the first chapter. Even so, appealing to intuition does not necessarily explain how medical knowledge is constrained by the natural world, and to this end I have looked to conventionalism.

Three conventionalist positions have been presented in turn, each building on the one before, to produce a highly satisfying species of conventionalism. The first was Engelhardt’s Kant-inspired epistemology with a “Hegelian accent” (Engelhardt 1996: 227-228). Although the examples Engelhardt uses to illustrate his position do not show how knowledge is constrained by the natural world, I find his appeal to Kant promising. To explain why, I have made use of Kant’s metaphor of a judge and a witness. In this metaphor, the questioning of the judge (representing the intuition of researchers) solicits a response from the witness (representing the natural world) to produce testimony (representing knowledge). The way the judge phrases questions will alter how the witness responds, but will
not fully determine the way the witness responds. Similarly, the intuitions of Hope and Mackenzie influence the facts that they produce, but do not determine them. This Kantian insight seems quite a promising way of showing how facts can be both historically contingent and constrained by the way the world is.

Nevertheless, Engelhardt does not clearly distinguish between the roles of the judge and the witness in his own examples. Consequently, it is not clear how he distinguishes intuitions that shape how the world is perceived and mistaken beliefs, nor is it clear how these intuitions can change historically. In order to address these issues, I have appealed to Poincaré’s conventionalism. Poincaré describes how certain conventions or “principles” need to be adopted before “laws” and “scientific facts” can be produced. Poincaré thus clearly separates the role played by the intuitions of researchers and the resulting constraint of the natural world. Poincaré also argued that the principles adopted can change, and have changed historically, and that these changes produce different facts about the world. Poincaré’s conventionalism gets close to articulating the sort of conventionalism required in this context.

However, Poincaré described conventions as things that are inserted into primitive laws to produce scientific laws and facts. Consequently, he needed to provide an account of how to produce primitive laws, and this he was unable to do. This problem is resolved by appealing to Fleck’s conventionalism. Despite Fleck’s own objections, Fleck’s epistemology has many conventionalist elements. Specifically, Fleck develops a notion of active and passive associations, which function in the same way as Poincaré’s principles and laws. According to Fleck passive associations cannot be made without first making some active ones, but the passive associations that are made are not simply constrained by the social, historical and cultural forces that constrain the active associations. Furthermore,
Fleck allows skills, experience, training, and practices to be incorporated into his account of active associations, which relates very well to the work of Hope and Mackenzie.

Fleck can therefore be usefully read as a conventionalist. However, because there are passages in Fleck’s work that make alternative readings of him possible, it has been useful to discuss Fleck’s conventionalism alongside other conventionalist positions. This has made it possible to draw out the conventionalist aspects of Fleck’s work, whilst at the same time showcasing the advantages of Fleck’s conventionalism; a task as yet un-tackled even by those scholars who read Fleck as a conventionalist (Giedymin 1986; Schnelle 1986b; Sady 2001, 2012).

Fleck’s conventionalism allows us to understand the differences in the facts produced by Hope and Mackenzie as the result of their adoption of different active associations. For Hope, the association between heart disease and the results of the post mortem examination were an active element of knowledge, as was the association between murmurs and certain results of the skilful use of the stethoscope. Having made these active associations, Hope could experience the passive association between heart murmurs and heart disease, and thus argued for the diagnostic value of auscultation. Mackenzie, on the other hand, made the active association between heart disease and a poor prognosis. Given this, Mackenzie could not find a passive association between heart disease and heart murmurs, and consequently argued against the diagnostic power of auscultation.
Chapter 7 – Ludwik Fleck and facts as invented and discovered

Introduction
In the last chapter I argued that facts produced by Hope and Mackenzie, such as those about the diagnostic value of auscultation, were not the result of brute observation. These facts did not “speak for themselves”. Rather, they were the contingent result of these historical actors adopting certain active associations. As Hope and Mackenzie adopted different associations to one another, they produced different facts about the diagnostic value of auscultation.

And yet, this way of describing how facts are produced may appear problematic. Recall that my objective is to show how facts can be both entirely historically contingent, whilst at the same time being constrained by something other than historical, social and cultural factors. I need to show how medical knowledge can be at the same time invented and discovered. Recall also that active associations are made and sustained in particular historical, social and cultural contexts. If the facts produced by Hope and Mackenzie are the result of adopting certain active associations, then how are these facts constrained by something other than historical, social and cultural factors? What is it that prevents the conventionalist position I have outlined in the last chapter collapsing into an extreme form of relativism? How can these facts be said to be discovered as well as invented?

I begin the chapter by highlighting that many scholars have read Fleck as an extreme kind of relativist, who holds that what counts as objective reality is determined fully by historical, social and cultural factors (section 1). Fleck did not approve of this reading of his work, but philosophers (in Fleck’s time and today) have not found his defence against the charge of extreme relativism convincing. I argue that Fleck’s epistemology can be defended against the charge of extreme
relativism by paying close attention to the relationship between the active and passive elements of knowledge, and in particular to how these interact to constrain researchers. I argue that the constraint provided by the active element of knowledge is entirely self-imposed, and is fully determined by cultural history and collective psychology (section 2). Therefore, active associations alone can provide no defence against extreme relativism. And yet, the constraint provided by some of the things Fleck used as examples of the passive element of knowledge is not self-imposed, and might be used to defend against the charge of extreme relativism (section 3). However, Fleck’s account of the passive element of knowledge is not consistent. Sometimes the constraints he claims result from the passive element of knowledge are self-imposed (section 4). Consequently, it is necessary to make a selective reading of Fleck’s account of the passive element of knowledge in order to locate a suitable defence against the charge of extreme relativism. By reading Fleck as a form of conventionalist, as discussed in the last chapter, it is possible to locate an account of the passive element of knowledge that can be used to defend Fleck against the charge of extreme relativism. Indeed, following Poincaré, I will argue that this conventionalism provides a form of objective knowledge (section 5).

Using the examples that Fleck drew from chemistry and medicine, I will show that even though passive associations cannot be produced without first making active ones, the ability to make passive associations is constrained by something other than the active associations that have already been made. So, even though active associations are necessary for passive associations to be made, they are not sufficient. Therefore the historical, social and cultural factors that constrain the active element of knowledge are insufficient to account for the constraint experienced as the passive element of knowledge. As we constrain ourselves,
we experience an additional loss of freedom. It is this additional loss of freedom which prevents the collapse into extreme forms of relativism, and which supports a conventionalist version of objectivity.

These insights can be applied to the work of Hope and Mackenzie. I will argue that the facts produced by Hope and Mackenzie were indeed the result of their adoption of certain active associations, but these active associations are insufficient to explain the constraint these two doctors experienced as fact. For instance, even though Hope did not have to associate heart disease with valvular lesions, once he had done this he lost the freedom to say that the stethoscope was not a powerful diagnostic tool. Even though Mackenzie did not have to associate heart disease with a poor prognosis, once he had done this he lost the freedom to say that the stethoscope was a powerful diagnostic tool. I will argue that these two doctors experienced an additional constraint to that which was imposed by the active associations they made when producing these facts. As such, the facts produced by these doctors were not fully determined by the historical, social and cultural factors that determined the active associations that they made. Such historical, social and cultural factors are insufficient to explain this additional constraint, which I will argue can be thought of as a form of objectivity. The experience of this additional constraint is what counts as a discovery.

1 – Fleck and the allegation of extreme relativism
As discussed in the last chapter, Fleck produced a species of conventionalism that did not require an account of crude facts. Fleck argued that all facts could be split into active and passive components, and thus that there were no facts that all parties are forced to accept as true as a result of the way the world is. Even though this gives Fleck a considerable advantage over Poincaré, who I argued
needed (and failed to provide) an account of crude facts, Fleck’s rejection of crude facts raises the problem of relativism.

Poincaré relied on crude facts to defend his conventionalism against the charge of relativism. As for Poincaré conventions were inserted into crude facts to produce scientific facts, what the resulting conventions and laws might be were considerably constrained by the crude facts. Just as metalworkers are not free to make anything they please, because of the properties of the metal they have to work with, scientists are not free to make any scientific facts they please, because of the crude facts they have to work with:

“And then, has one the right to say that the scientist creates the scientific fact? First of all, he does not create it from nothing, since he makes it with the fact in the rough. Consequently he does not make it freely and as he chooses. However able the worker may be, his freedom is always limited by the properties of the raw material on which he works” (Poincaré 2001b: 328).

Poincaré could defend his conventionalism against the charge of an extreme form of relativism (in which scientists are free to create any scientific fact they wish, and where the truth is simply what the scientist says it is) by rooting his conventionalism in crude facts. By denying that there are any crude facts, Fleck lost this ability and apparently left himself vulnerable to the charge of extreme relativism. By arguing that Fleck’s conventionalism is a valuable way of understanding the work of Hope and Mackenzie, I have left myself open to the same charge.

Fleck, however, denied that he was a relativist\textsuperscript{108}. He argued that relativism entailed that the same claim could be true for one person and false for another.

\textsuperscript{108} Some scholars have denied that Fleck was a relativist, but have done so by claiming that he thought that there were timeless facts, about which all competent observers are forced to agree because of the way that the world is. For instance Stephen Toulmin has argued that Fleck held that some facts may be
He maintained that when working within a thought style, a claim would either be true or false, not both.

“Truth is not “relative” and certainly not “subjective” in the popular sense of the word. It is always, or almost always, completely determined within a thought style. One can never say that the same thought is true for A and false for B. If A and B belong to the same thought collective, the thought will be either true or false for both. But if they belong to different thought collectives it will just not be the same thought!” (Fleck 1979: 100; see also Fleck 1990: 268).

As has been regularly discussed by Fleck scholars, Fleck argued that all perception is directed by “thought styles”, which are cultivated and sustained by groups of researchers engaged in inquiry (the “thought collective) (Fagan 2009: 273; Mößner 2011: 362). Fleck defined a thought style as “the entirety of intellectual preparedness or readiness for one particular way of seeing and acting and no other” (1979: 64, 99). A thought style is comprised of the group’s shared knowledge, methods and communicative behaviour (Mößner 2011: 365). Fleck argued that many aspects of a thought-style (including active associations, as one aspect of shared knowledge that directs perception) were accepted and maintained as a result of historical and cultural forces acting within the thought collective (Fleck 1979: 99). Therefore, Fleck argued that historical and cultural forces direct perception, a claim that many have taken to entail an extreme form of relativism.

accepted as beyond dispute because they “express an empirical relationship that demands passive acceptance from all scientists working in that science regardless of their current theoretical views” (1986: 279). Ilana Löwy (1988: 135) has also endorsed this view. Löwy has also argued more recently that Fleck held that “the intrinsic properties of the material world” acted independently of any sociological forces to pull scientific opinion towards an ultimate, timeless truth (2004a: 439). However, as discussed in a previous chapter, Fleck explicitly and consistently denied that there are any such timeless facts or ultimate truth. Fleck argued instead that there was “no firm ground of facts” (1979: 92), and that “there is probably no such thing as complete truth or complete error” (1979: 20). Consequently, he held that scientists were never simply “passive” when it came to the production of facts. “Not a single statement can be formulated from passive links alone” (Fleck 1979: 49). Fleck held that there were no facts of any kind that had to be accepted simply because of the way the world is.
For Fleck, a relativist was someone who believed the same statement could be both true and false at the same time (Fleck 1990: 268). As this was not his position, he argued that he was not technically a relativist. When working in two different thought styles, apparently similar claims could be true in one style and false in another, but these claims would not really be the same claim, so there was no need to worry about contradictions. This response by Fleck has led Markus Seidel to argue that Fleck was not a relativist, but rather a relationist (in the mode of Karl Manheim), who rejected the notion of “absolute objectivity” but accepted the notion of “objectivity in a certain thought-style” (Seidel 2011: 226).

However, this defence has not satisfied Fleck’s critics, because it does not explain how knowledge is constrained by factors other than the social, historical and cultural factors that Fleck argued constrained a thought style. One of Fleck’s earliest critics, Tadeusz Bilikiewicz, objected to Fleck’s views, arguing that researchers should at least try “to distinctly separate what proved to be scientifically true from what proved to be false” (Bilikiewicz 1990: 275). Seidel himself has questioned the difference between relationism and relativism (2011: 236). “Is there really any genuine difference between a self-refuting relativism and a supposedly coherent relationism if in relationism inner-style truth is always dependent on and relative to a specific thought style? Isn’t that just a new fresh name for a very old idea?” (Seidel 2011: 236). Similarly, John Wettersten has argued that “[a] central problem which arises for Fleck’s view is how science can be objective”, and that Fleck “could not resolve the problems of relativism which his approach raised” (Wettersten 1991: 478, 493). If truths are only relative to a thought style, and a thought style is constrained only by social, historical and cultural factors, then it is possible that truths are constrained by social, historical and cultural factors alone. By only offering the above defence against the charge
of relativism, Fleck failed to address the concerns of his critics. The charge of extreme relativism is still commonly levelled at Fleck (Fagan 2009: 279; Löwy 1988: 135), and I am still vulnerable to the same criticism.

It should be noted that not all scholars think that Fleck’s alleged relativism is a bad thing. As described in chapter 3, Andrew Cunningham (2002) argues that “you die of what your doctor says you die of. And that’s that”; and he uses Fleck’s work to support this position. Steven Shapin (1986: 327) has referred to the eighteenth century observation *Bathybias haeckleii* as a case study that makes Fleck’s point. When looking at what was referred to as *Bathybias*, some researchers saw a form of a-cellular, primordial life; whilst others saw the result of a precipitation reaction between sea water and the alcohol preservative. Shapin argues that *Bathybias* was in fact both fact and artefact, because this is what different researchers saw, thereby reducing truth to what researchers decide is the case. As discussed in a previous chapter, H. Tristram Engelhardt has argued that the disease of masturbation, as diagnosed in the eighteenth century (when it was thought to cause epilepsy and rickets), was as legitimate a diagnosis as any other; and he again cites Fleck as a philosopher who would support this view (1996: 191-192). These scholars all use Fleck’s work to support extreme relativist positions that hold that facts are whatever people decide that they are, and I will argue here that this reading of Fleck is problematic.

Other scholars praise Fleck’s relativism, but stop short of endorsing such extreme relativist views. For instance Yehuda Elkana has praised Fleck for rejecting the notion of a “the truly absolute reality which is our highest human intellectual aspiration”, and showing how an individual’s perception of reality is shaped by wider social, historical and cultural contexts (Elkana 1986: 310-311). Adrian Wilson (2000: 276) has praised Fleck for rejecting what he calls a “naturalist-
realist” account of diseases, in which our concepts of disease act as a “mirror of natural reality”, and instead adopting an “historicist-conceptualist” account of diseases, in which our concepts of disease are historically contingent and legitimately shaped by social, historical and cultural forces. Even so, neither of these scholars discuss how the pitfall of extreme relativism is avoided by Fleck.

Some scholars have sought to defend Fleck against the charge of extreme relativism (Löwy 2004a, 1988; Toulmin 1986; Fagan 2009). These scholars highlight that Fleck made several references to the role played by “objective reality” or by “the object to be known”, and claimed on several occasions that something other than social, historical and cultural factors constrain medical knowledge (Fleck 1979: 40, 38, 79). Despite this, Fleck’s critics are still not satisfied. They have argued that Fleck contradicted himself by appealing to objective reality. For instance, Jonathan Harwood has argued that Fleck’s attempt to claim both that knowledge is shaped “by constraints imposed by the real world” and that “thought-style “dictates” and “coerces” how and what a scientist sees and thinks” is a contradiction (Harwood 1986: 182). Thomas Schnelle (1986b: 253) makes a similar claim. Henk van den Belt and Bart Gremmen (1990) have also criticised Fleck for advocating a “non-egocentric” tolerance of different thought styles, whilst at the same time criticising the notion of “specificity” adhered to by many of his colleagues. By acting according to his interests as a dissenting bacteriologist and being critical of his colleagues, they argue that Fleck “makes a mockery” of his own epistemological position (van den Belt and Gremmen 1990: 467). “Relativism seems a luxury only to be allowed when no direct interests are at stake” (van den Belt and Gremmen 1990: 478). These scholars do not see how Fleck’s allegedly relativist epistemology allowed for knowledge to be constrained by the natural world. Not only did Fleck fail to
adequately answer the charge of relativism, but his work also appears to confuse many critics who see a conflict between Fleck’s claim that all knowledge is historically contingent and his claim that that knowledge is constrained by something other than social, historical and cultural factors (Fleck 1979: 64).

And yet, this is exactly what Fleck’s epistemology is capable of doing. The claims that facts are not “definite, permanent and independent of any subjective interpretation” (Fleck 1979: xxvii) and that what counts as a fact is constrained by something other than social, historical and cultural factors are fully compatible. To see this it is important to pay close attention to how the active and passive elements of knowledge relate to each other, and to how they constrain researchers. This must be done with care, however, as scholars have commented that the relationship between the active and passive elements of knowledge in Fleck’s epistemology is difficult to grasp. For instance, Stephen Toulmin also argued that Fleck was “echoing” Kant by claiming that facts are what we make of our experience, and that Fleck goes beyond Kant by showing how scientific judgement is shaped by social, historical and cultural forces (Toulmin 1986: 277). Nevertheless, Toulmin still thought that “Fleck’s account of the nature of ‘facts’ remains the most opaque part of his position” (Toulmin 1986: 277). Toulmin singles out Fleck’s discussion of active and passive associations as difficult to understand, particularly given the apparent tension between Fleck’s claims that facts are both made and inevitable. “Obscurities remain, in particular, in his distinction between the ‘active’ and ‘passive’ elements in knowledge, and in his claim that the outcomes of science are inevitable” (Toulmin 1986: 277). I address these difficulties below.

Similarly, Thomas Kuhn admitted, in the preface to the English translation of *Genesis* (1979: xi), that he found Fleck’s discussion of passive and active
elements of knowledge “unenlightening”. In particular, Kuhn saw that “[w]hat the thought collective supplies its members is somehow like the Kantian categories, prerequisite to any thought at all”, and yet Kuhn could not see how Fleck’s use of the distinction between the active and the passive helped to explain how this happens (Fleck 1979: xi).

In my view this is something of a shame, because Fleck’s distinction between the active and passive elements of knowledge could have been useful to Kuhn. Following the reconstruction of Kuhn’s views made by Paul Hoyningen-Huene (1993), it looks to me as if Kuhn and Fleck were driving at a similar epistemic goal – to explain how the subjects of knowledge (researchers) constitute the world that appears to them, without thereby fully determining its character. That Kuhn’s position is that researchers have no direct access to the world in itself has been widely understood, but his claims that his position does not entail “mere relativism” have not (Kuhn 1996: 205-207). Kuhn views have been read by both advocates and detractors as a sort of “extravagant idealism”, in which articles of knowledge are “entirely at the whim of the subject who imagines them” (Hoyningen-Huene 1993: 268, 75; Barnes 1982: 23; Scheffler 1967: 19). Hoyningen-Huene argues that Kuhn attempted to address (and thereby correct) this idealist reading of his work by invoking the notion of “stimuli”, which supposedly emanate from the world in itself, and prevent observers looking at the same scene from seeing just anything (1993: 51):

“The determinate, proprietary features of stimuli are meant to have the function of warding off the complete relativization of the concept of reality to individuals or communities, which threatens when we adopt the critical epistemological standpoint characteristic of Kuhn’s theory. Although we may never determine their features, stimuli do the job of resisting the impending arbitrariness of perception and theory formation and thus prevent the onset of thoroughgoing relativism, in virtue of their own being and their proprietary determinacy” (Hoyningen-Huene 1993: 50).
On this view, stimuli from the world in itself interact with the observer’s perceptual apparatus (e.g. eyes and ears), and the observer’s cultural background, to produce knowledge (Hoyningen-Huene 1993: 49). Knowledge is thus co-produced by the knowing subject (the observer) and the object to be known (the world in itself). Hoyningen-Huene (1993) analyses Kuhn’s efforts to show how subjects of knowledge constitute the world they experience in terms of “object-sided moments” arising from the world in itself, and “subject sided moments” supplied by researchers. “Kuhn makes use of the opposition between (genetically object-sided) “observation and experience” and (genetically subject-sided) “element of arbitrariness” (Hoyningen-Huene 1993: 75). Hoyningen-Huene argues that, For Kuhn, the concept of that which is object sided is the result of a subtraction of all subject sided moments from knowledge (1993: 268; 1989: 394). This subtraction should yield a stable set of experience given to researchers by the way the world in itself is, and which prevent knowledge from being entirely arbitrary. “In addition the results of this process of subtraction are unequivocal in the sense that, regardless of which phenomenal world it starts out with, the same world-in-itself results” (Hoyningen-Huene 1993: 268). The problem with this view is that, seeing as Kuhn argued that observers cannot experience the world in itself, the “proprietary determinacy” of stimuli coming from the world in itself can do no work in his epistemology (Hoyningen-Huene 1993: 63):

“For if this purely object-sided world is to have power for which it was introduced in the first place, it must have determinate and determinable features; nothing indeterminate or indescrivable can serve as an explanans (for something determinate)” (Hoyningen-Huene 1993: 63).

So, Hoyningen-Huene (1993) identifies significant issues with Kuhn’s epistemology, which result from Kuhn’s appeal to subject-sided and object-sided
influences. These issues manifest in Hoyningen-Huene’s discussion of how the world in itself resists the theoretical expectations of researchers, in the production of anomalous observations:

The resistance of the world-in-itself (or stimuli) may, to some extent, penetrate the network of similarity relations, despite the fact that these similarity relations are, in a certain respect, already attuned to such resistance by virtue of their incorporation of genetically object-sided moments. The repeated occurrence of significant anomalies in the history of science can, in fact, prove one motive for assuming the existence of a theory-independent, and by its own determinate, proprietary features resistant, world-in-itself” (Hoyningen-Huene 1993: 227).

As Hoyningen-Huene has it, it is the “proprietary features” of the world-in-itself that penetrate and thus resist the theoretical expectations of researchers. But, to appeal to object-sided proprietary features that show up in observations made by researchers is to appeal to something fixed and given, and thus not historically contingent. Furthermore, how can such features show up like this, if they cannot even be identified? In his epistemology, Kuhn appeals to features of the world in itself that are known to be fixed and given, whilst at the same time denying that such features exist.

Consequently, Kuhn’s account of how the phenomenal world is constituted is not suitable for my purposes. However, Hoyningen-Huene’s (1993) reconstruction (or perhaps reinterpretation) of Kuhn’s work is more helpful. He makes some important comments about how the proprietary resistance of the features of the world-in-itself is felt. When significant anomalies appear:

“the consensus which reigned up to this point is shattered in a manner not explicable by social causes alone. For those forms of resistance which, though experienced in the perception and thought of many individuals, result from purely social circumstances may be a prerequisite of uniformity, but they can’t destroy uniformity. Consequently, the proprietary resistance of the world-in-itself must be a participant in the production of significant anomalies” (Hoyningen-Huene 1993: 270).
Here, Hoyningen-Huene is no longer focused on how the proprietary features of the world in itself might disrupt the consensus of researchers. Instead, he identifies that in an anomaly researchers find that their expectations are resisted in a way that cannot be explained by appealing to social circumstances alone. Similarly, the difficulty scientists have in accommodating this anomaly is not limited to “the difficulty involved in seeking the social acceptance of one’s own opinion” (Hoyningen-Huene 1993: 270). In other words, researchers have found themselves not to be free in ways that cannot be accounted for by the constraint imposed by their social circumstances alone. It is this additional loss of freedom, over and above that which can be explained socially, that is of interest. As identified at the end of the last chapter, it is possible to discuss this loss of freedom, or resistance, without discussing the proprietary features of the world in itself that are doing the resisting. This is just as well, as both Kuhn and Fleck claim it is not possible to meaningfully discuss the features of world in itself. Whilst Kuhn’s use of subject-sided and object-sided proprietary features does not allow him to take account of this loss of freedom, and thereby ward off the charge of extreme relativism, Fleck’s use of active and passive associations does.

2 – The constraint provided by the active element of knowledge is entirely self-imposed. Consider again the examples that Fleck used to illustrate the relationship between the active and the passive elements of knowledge. Recall from the last chapter that Fleck’s discussion of the fact that the atomic weight of hydrogen was 1.008 (Fleck 1979: 83, 95, 101). Fleck argued that it was only possible to produce this passive fact following the active stipulation that the atomic weight of oxygen was exactly 16. Fleck does not discuss how this happens in much detail, and it is
useful to expand a little on how this fact about the atomic weight of hydrogen may be produced.

At the time Fleck was writing, the atomic weight of hydrogen was the weight of an atom of hydrogen relative to the weight of an atom of oxygen, if an oxygen atom is taken as weighing 16 atomic weight units. The measurement of the atomic weight of hydrogen at this level of precision was achieved in the late nineteenth century, and one of the methods used to do this was by comparing the weights of equal volumes of hydrogen and oxygen gas.

Accepting that equal volumes of gas at equal temperatures and pressures will contain an equal number of particles, an equal volume of oxygen gas and of hydrogen gas at room temperature and pressure will contain the same number of molecules. Pure gaseous oxygen has the molecular formula $\text{O}_2$, which means that a molecule of oxygen is made up of two atoms of oxygen attached together to make a single particle. The same is true for pure gaseous hydrogen, which has the molecular formula $\text{H}_2$. Therefore, an equal volume of oxygen gas and hydrogen gas will contain an equal number of atoms. Therefore, the ratio of the weights of the same volume oxygen and hydrogen gas will be equal to the ratio of the weights of oxygen and hydrogen atoms.

In the late nineteenth century it was possible to weigh a volume of gas using very accurate scales. However, this on its own does not measure the weight of an individual atoms in the gas, as the number of atoms in the volume of gas are unknown. This problem can be solved by stipulating what the weight of the atoms in one of the volumes of gas is, and by using this stipulated figure to calculate the weight of the atoms in the other gas relative to it. In Fleck’s time, it was stipulated that the atomic weight of oxygen was exactly 16. Whatever the result obtained by
weighing the volume of oxygen, the researchers would say that the atomic weight of oxygen was 16 atomic weight units. If the volume of oxygen weighed four grams, the researchers would say the mass of an oxygen atom was 16 atomic mass units. If the volume of oxygen weighed 16 grams, the researchers would still say an atom of oxygen weighed 16 atomic mass units.

As the ratio of the weights of the two equal volumes of gasses is the same as the ratio of the atoms of those gasses, measuring the weight of the hydrogen was simultaneously a measure of the weight of the hydrogen atoms. The only caveat was that rather than producing a measure of the weight of a hydrogen atom in grams, this method allowed researchers to measure the weight of a hydrogen atom as a proportion of the weight of an oxygen atom. So if the volume of oxygen weighed 16 grams, the atomic weight of oxygen was stipulated as 16, and the volume of hydrogen also weighed 16 grams, the atomic weight of hydrogen would have been measured as 16 atomic weight units. Alternatively, if the volume of hydrogen weighed only 8 grams, then the atomic weight of hydrogen would be 8 atomic weight units. As it turns out, if the volume of oxygen weighed 16 grams, then the volume of hydrogen would weigh 1.008 grams, and the atomic weight of hydrogen would be measured as 1.008 atomic weight units.

Having explored Fleck’s example in a little more detail, I can use it to illustrate important aspects of the relationship between the active and the passive element of knowledge. As discussed in the last chapter, Fleck described the association between the atomic weight of oxygen and the number 16 as “almost consciously conventional and arbitrary”109 (Fleck 1979: 83). This is why he called it an active association, because it is fully determined by the choices made by researchers.

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109 Fleck’s description of this active association as “arbitrary” is perhaps a little strong. As Poincaré said: “Conventions, yes; arbitrary no” (Poincaré 2001a: 86) – see below.
Researchers chose 16 as the atomic weight for oxygen, and in the procedure for weighing hydrogen described above would maintain this association come what may. This decision imposes a constraint upon the researchers. Because they have stipulated that the atomic weight of oxygen is 16, they lose the freedom to find that the atomic weight is 15, 14, or any other number. The key point is that this loss of freedom depends entirely on the choices made by these researchers. Regardless of the results obtained for the weights of the volume of oxygen and hydrogen gas, the researchers will say the atomic weight of oxygen is 16. This constraint is entirely self-imposed.\(^{110}\)

The claim that the constraint arising from the active element of knowledge is self-imposed should not be taken to imply that the adoption of active associations have nothing to do with the results of empirical observation. Fleck admitted that empirical observation does indeed inform the adoption of active associations. “All these points of view are based upon observation, perhaps even on experiment, and none can simply be declared wrong” (Fleck 1979: 8). The stipulation that the atomic weight of oxygen is 16 was not arbitrary. It resulted from the experimental observation that the weight of a volume of oxygen gas was *approximately* sixteen times that of the weight of the same volume of hydrogen gas. Nevertheless, even if it is informed by empirical observation, the decision to adopt one active association rather than another is still a “choice” (Fleck 1979: 8-10). Poincaré made this point beautifully:

“*Our choice among all possible conventions is guided by experimental facts, but it remains free, and is only limited by the necessity of avoiding every contradiction, and thus it is that postulates may remain rigorously true even*
when the experimental laws that determined their adoption are only approximate" (Poincaré 2001a: 45).

Poincaré argued that experiment could guide researchers in their choice of conventions, “but there its role ends” (Poincaré 2001a: 69). Researchers in Fleck’s time could have decided to stipulate that hydrogen’s atomic weight was exactly 1 (as many researchers had done in the eighteenth century), or that carbon’s atomic weight was exactly 12 (as researchers do today). In order to explain why oxygen’s atomic weight was exactly 16, we have to explain why researchers made this choice. Furthermore, if we are to explain why oxygen’s atomic weight was exactly 16 even when researchers discovered that that a volume of oxygen gas is not exactly sixteen times the weight of a volume of hydrogen gas, we have to explain why researchers chose that it should be. Under these circumstances it is not possible for these researchers to be wrong about the atomic weight of oxygen, as oxygen’s atomic weight is whatever they say it is. In Fleck’s time, the glue that held oxygen and the atomic weight 16 together was these researchers’ collective will.

As these researchers were conscious that the atomic weight of oxygen was stipulated, they explicitly argued about the advantages and disadvantages of adopting the atomic weight of oxygen as the standard to assess other atomic weights (Holden 2004; Hamerla 2003: 367; Richards 1900). In 1899 an international committee of eminent chemists literally voted, by post, on the question of whether oxygen or hydrogen should be used as the standard to measure atomic weights (Richards 1900). Those in favour of oxygen won the vote, and oxygen was accepted as the standard\(^{111}\). To understand why these

\(^{111}\) The history of the decision to adopt oxygen as the standard for the measurement of atomic weights is obviously more complicated than how I have presented it here. For instance, many chemists not on the international committee on atomic weights were unhappy with this result, and the international
researchers imposed this constraint on themselves is to understand the history of this decision. This is why Fleck (1979: 10) says that active associations can be explained in terms of cultural history.

It should be noted that this is also true for active associations that are not explicitly acknowledged as choices. In his history of syphilis, Fleck discussed several alternative definitions for the disease. These included the active association of syphilis with things like the “mythical-ethical concept” of the “carnal scourge”, or an affliction of the genitals that gets better when treated with mercury, or venereal disease that was not gonorrhoea or soft chancre (Fleck 1979: 2-8). Despite these different options being logically available, Fleck denied that the people who made these active associations were aware that other options were available to them. “Sixteenth century physicians were by no means at liberty to replace the mythical-ethical concept of syphilis with one based upon natural science and pathogenesis” (Fleck 1979: 9). These physicians were constrained by their culture into defining syphilis in a particular way. As such, a constraint like this is one which members of a culture impose upon one another. The glue that in the sixteenth century held syphilis and the mythical-ethical concept of the carnal scourge together was still the will of these physicians and other people in this culture, even if they were unaware of this act of volition. This is why Fleck (1979: 10) described this sort of constraint as explicable in terms of cultural history and collective psychology.

In summary, the making of active associations produces a self-imposed constraint or loss of freedom for researchers, which is explicable in terms of committee continued to publish results for atomic weight measurements using both oxygen and hydrogen as a standard until 1906 (Holden 2004). Nevertheless, the point that this decision was a conscious choice is well made.
cultural history and collective psychology. And yet, this is not the only constraint experienced by researchers in the wake of their making active associations. Recall from the previous chapter that Fleck argued that the active element of knowledge was always necessary for the passive element of knowledge (and therefore facts) to be produced (Fleck 1979: 9-10). He described the active elements of knowledge as preconditions for the experience of the passive elements of knowledge (Fleck 1979: 40). Fleck also claimed in several places that, in contrast to the active associations made by the thought collective, the resulting passive associations that can be made are not fully determined by historical, social and cultural factors. Speaking of passive associations, he said “there are always other connections which are also to be found in the content of knowledge that are not explicable in terms either of psychology (both individual and collective) or history” (Fleck 1979: 10).

When discussing the Wassermann reaction (which is the blood test for syphilis that is the focus of his book), Fleck not only argued that the active element of knowledge provided an important constraint during the development of this reaction, but also argued that these historical, cultural and social constraints were not sufficient to account for the Wassermann reaction’s development:

“These last statements must not, however, be taken to mean that the Wassermann reaction can be reconstructed in his objective entirety simply from historical factors along with those of individual and collective psychology. Something inevitable, steadfast, and inexplicable by historical development is left out of such attempts” (Fleck 1979: 79).

Fleck held that the collective and individual choices made by researchers that make up the active element of knowledge do not by themselves determine the passive element of knowledge. Although active associations are necessary for
the production of facts, they are not sufficient for the production of facts. An additional constraint to that provided by the active associations alone is experienced, and this constraint is not self-imposed. To see this it is useful to return to Fleck’s chemical example.

3 – The constraint experienced as the passive element of knowledge is not self-imposed.
To see how the passive element of knowledge is not a self-imposed constraint, it is useful to explore the consequences of making the atomic weight of oxygen exactly 16. As discussed in the previous chapter, unless some stipulation about the atomic weight of oxygen (or at least some other substance that could act as an alternative standard) is made, it would not be possible to measure the atomic weight of hydrogen in this way. Furthermore, what the atomic weight of hydrogen is measured to be depends upon what the stipulated value for the atomic weight of oxygen. If the active association concerning the atomic weight for oxygen changed, the measured atomic weight for hydrogen would also change. If the stipulated atomic weight of oxygen was doubled (to 32), then the measured atomic weight of hydrogen would also double (to 2.016). If the stipulated atomic weight of oxygen was halved (to 8), then the measured atomic weight of hydrogen would also be halved (to 0.504). If the stipulated atomic weight of oxygen is kept the same at 16, then the measured atomic weight of hydrogen would be 1.008.

The key point is that the self-imposed loss of freedom with respect the atomic weight of oxygen produces a further loss of freedom with respect to the atomic weight of hydrogen. Once the researchers decide that the atomic weight for oxygen is 16, they lose the freedom to find that the atomic weight of hydrogen is exactly 1, or 2, or 1.5, or any number apart from 1.008.
But what is the source of this further loss of freedom? Like the loss of freedom to find that the atomic weight of oxygen is anything other than 16, the loss of freedom to find that the atomic weight of hydrogen is something other than 1.008 does depend on the choices made by the researchers. And yet, unlike the loss of freedom to find that the atomic weight of oxygen is anything other than 16, it does not depend on these choices alone. As discussed above, the researchers would say that the atomic weight of oxygen is 16 regardless of the results obtained from the weighing of the volumes of oxygen and hydrogen gas. The constraint for this active association was entirely self-imposed. In contrast to this, the measured result for the atomic weight of hydrogen does depend upon the results of these observations. Although the researchers cannot make observations to measure the atomic weight of hydrogen without making choices about what the atomic weight of oxygen is, the result of this measurement does not depend on their choices alone.

Again, Poincaré expressed this beautifully. Poincaré viewed the choice of convention to be closely analogous to the choice of a language used to describe result. In my view this analogy is highly problematic, and will be discussed further below. Nevertheless, using this analogy Poincaré argued that the decision of which conventions to use did not determine the facts that were subsequently produced:

“If you put the question to me: Is such a fact true? I shall begin by asking you, if there is occasion to state precisely the conventions, in other words, what language you have spoken; then once settled on this point, I shall interrogate my senses and shall answer yes or no. But it will be my senses that have made the answer, it will not be you when you say to me in English or in French” (Poincaré 2001b: 325).

Choosing conventions (and analogously the active element of knowledge) allows us to make scientific observations, but is not sufficient to determine what we
observe (as the passive element of knowledge). Therefore, the additional constraint we experience is not the result of the active associations alone.

Poincaré also argued that we can tell that scientific facts are not simply invented because we can use them to make predictions, and use these predictions to do useful things. “Science foresees, and it is because it foresees, that it can be useful and serve as a rule of action” (Poincaré 2001b: 320). Some predictions succeed, whereas others fail. “The alchemists had recipes for making gold, they loved them and had faith in them, and yet our recipes are the good ones, although our faith be less lively, because they succeed” (Poincaré 2001b: 321). It does not matter how much we wish them to succeed, these recipes for making gold from base metals fail. Even if we wanted them to fail, our recipes (for instance) for making water from hydrogen and oxygen gas succeed. “In sum, facts are facts, and if it happens that they satisfy a prediction, this is not an effect of our free activity” (Poincaré 2001b: 329). In my view Poincaré is correct – that our recipes can succeed and fail shows that there is something more than our will involved in the production of scientific facts, even though it is us who choose what these recipes are.

Fleck too connects the passive element of knowledge with utility, and with inevitability and necessity. For instance, when describing the procedure for carrying out the Wassermann reaction he says:

“for every active element of knowledge there corresponds a connection that is passive and inevitable. We have already mentioned a few such linkages, for instance, that the mere use of alcohol for preparing extracts is an active element of knowledge, whereas the actual usefulness of such extracts is a passive one and therefore a necessary consequence” (Fleck 1979: 82).

The recipe for the Wassermann reaction is ours to choose, but whether or not this recipe is useful for the detection of patients with syphilis is not up to us (Fleck
1979: 79, 81). Given these choices, the usefulness of the test is inevitably and necessarily forced upon us. The necessity here is not a logical necessity. The choice to use alcohol in the recipe for the Wassermann reaction does not logically entail that the test should or should not be useful. It does not provide any a priori indication about the test’s utility at all. Nevertheless, the ability to foresee which patients have syphilis using the Wassermann reaction is an ability that those who know how to carry out the test have or do not have\(^\text{112}\), whether they want it or not. As passive associations are forced upon us against our individual and collective will, they cannot be explained by appealing to cultural history or collective psychology. By tying the passive element of knowledge to the ability to foresee, to predict, and therefore to be useful, Fleck marked the passive element of knowledge as a constraint that is greater than that which results from the active element of knowledge alone.

Another way of looking for the source of this additional constraint is to consider with what claiming that the atomic weights for oxygen and hydrogen are not 16 and 1.008 respectively would clash. Should researchers claim that the atomic weight of oxygen was not 16, this would immediately clash with the body of active associations already accepted by their peers. Specifically, it would directly contradict the active association between the atomic weight of oxygen and the number 16. In contrast to this, there was no conflict between the active associations made by the researchers investigating the atomic weight of hydrogen and the claim that this atomic weight was not 1.008. Indeed, many of these late nineteenth century researchers explicitly stated that they were carrying out their work to see whether or not the ratio of the atomic weights of hydrogen

\(^{112}\) Löwy (2004b) describes how researchers came to find that the Wassermann reaction was not an accurate test for syphilis. This makes my point, as there is a fact of the matter.
to oxygen was exactly 1 to 16 (Hamerla 2003; Cooke and Richards 1887: 149; Rayleigh 1887: 356; Morley 1900: 284; Richards’ Nobel lecture, 6th December 1919\(^\text{113}\)). So if the atomic weight of oxygen was exactly 16, it was a possibility for these researchers that the atomic weight of hydrogen was exactly 1, and therefore not 1.008. The loss of freedom these researchers felt in being unable to claim that the atomic weight of hydrogen was exactly 1, or indeed any number other than 1.008, did not arise solely from a clash with the active element of knowledge. A clash with something in addition to this is required to account for this constraint.

The same insights apply to Fleck’s examples concerning the active and passive elements of knowledge about syphilis. Consider the claim that “sometimes mercury does not cure the carnal scourge but makes it even worse" (Fleck 1979: 10). According to Fleck, this “restriction of the curative effect of mercury” was a passive element of knowledge (1979: 10), and therefore like the claim that the atomic weight of hydrogen is 1.008. As discussed in the last chapter, Fleck argued that it was impossible to produce this passive association between the patients with the carnal scourge and the poor efficacy of treatment with mercury without first making active associations. Specifically, Fleck referred to the active association of all patients with pathology of the genitals, which today would be seen as a collection of patients with different venereal diseases (1979: 10). Only having made this active association, it was possible to find that not all of these

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\(^{113}\) Richards received the Nobel Prize for chemistry in 1914 for measuring atomic weights of many elements with great accuracy. In his Nobel lecture he described his results as finding that “the atomic weight of hydrogen must not be far from 1.008 if oxygen is taken as 16.000” (a transcript of the lecture is available at www.nobelprize.org/nobel_prizes/chemistry/laureates/1914/richards-lecture.html). However, in his 1887 (p. 173) publication of this result he described it as if hydrogen was taken to be 1 then oxygen must not be far from 15.953. What he measured in his work was the atomic weight for oxygen. So this was the passive association in his work, and hydrogen being taken as exactly 1 was the active association.
patients recovered following treatment with mercury. But it was not the making of this active association that determined this result. Indeed, it is fair to speculate that the physicians attempting to treat the carnal scourge with mercury did hope, and possibly expect, that this therapy would work. The association of all patients with afflicted genitals does not contradict, and is perfectly compatible with, the possibility of cure following treatment with mercury. If there is a clash between these things, this clash is not with the active element of knowledge. This clash arises from the experience of trying to treat people with the carnal scourge using mercury, and failing to do so. Something in addition to the active associations is required to account for this additional constraint.

I find these insights from Fleck and Poincaré very helpful in this context. The active element of knowledge is necessary but not sufficient to produce the passive element of knowledge. After these researchers and physicians decided to make the active associations they did, they experienced a loss of freedom in addition to the constraint they imposed upon themselves. As they constrained themselves, they experienced an additional loss of freedom.

In pointing out this additional loss of freedom, I do not mean to imply that we are less free overall after we produce facts than before. Indeed, both Fleck and Poincaré viewed this additional loss of freedom as a good thing. Poincaré thought that science would become powerless without it, for we would be unable to use science to make predictions about what we will observe in specific circumstances in the future (2001a: 5; 2001b: 321). For Fleck the alternative to passive constraint was not complete freedom, but rather confusion and chaos:

“The work of the research scientist means that in the complex confusion and chaos which he faces, he must distinguish that which obeys his will from that which arises spontaneously and opposes it. This is the firm ground that
he, as representative of the thought collective, continuously seeks. These are the passive connections, as we have called them” (Fleck 1979: 95).

It is only by finding things that do not obey our will that we are able to ward off this confusion and chaos. It is because we are not free to claim that the atomic weight of hydrogen is anything other than 1.008 (given that oxygen is exactly 16) that we know the correct recipe for rocket fuel. It is because we know that “sometimes mercury does not cure the carnal scourge but makes it even worse” (Fleck 1979: 10) that we know to warn patients of the potential dangers of this therapy. It is by finding sure footing that we are enabled to climb.

Even if Fleck does connect the passive element of knowledge with the additional loss of freedom discussed above, it is wrong to identify Fleck’s account of the passive element of knowledge with this alone. Fleck also connected the passive element of knowledge with other sorts of constraint that are not so useful in developing an account of objectivity. Fleck therefore conflated several different accounts of the passive element of knowledge. These need untangling, and this is best done with reference to Fleck’s comments about the similarities between science and fiction. Having done this it will be possible to show how a reading of Fleck in the light of Poincaré’s conventionalism can be used to develop a valuable account of objectivity.

4 – Alternative accounts of the passive element of knowledge in Fleck’s work
Fleck frequently described passive associations as “inevitable”, “necessary”, “substantive” and “compulsory” connections that arose “spontaneously” following the adoption of active associations (1979: 10, 82-83, 101). I have discussed what these claims mean on the context of three of Fleck’s examples – the measurement of the atomic weight of hydrogen, the efficacy of mercury in the treatment of syphilis, and the diagnostic accuracy of the Wassermann reaction. The trouble is
that Fleck also said these inevitable, necessary, substantive, spontaneous and compulsory passive connections arise whenever a body of active associations are laid down, such in any work of fiction:

“The passive and the active elements cannot be separated from each other completely either logically or historically. Indeed it is not even possible to invent a fairy tale which does not contain some inevitable connections” (Fleck 1979: 95).

Fleck claimed that whenever active associations are made, passive associations will inevitably follow, even in fairy tales. If passive connections occur in any work of fiction, then it is unlikely the additional constraint experienced as the passive element of knowledge can be usefully connected with objectivity.

I am not suggesting that passive elements of knowledge in the sense that interests me never occur in works of fiction. I am not suggesting that the presence of the passive element of knowledge could function to demarcate science and fiction114. Indeed, Fleck goes on to argue that myth and science exist on a continuum:

“Myth differs from science in this respect only in style. Science seeks to include in its system a maximum of those passive elements irrespective of inherent lucidity. Myth contains only a few such passive elements, but they are artistically composed” (Fleck 1979: 95).

This is an intriguing claim with which I do not want to disagree. Fleck does not expand on this point, but I can make sense of it. Sometimes scientists make use

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114 Neither am I arguing here that the ratio of active to passive elements of knowledge could serve as a criterion for objectivity. Fleck does argue that myth contains relatively fewer passive associations than does science (1979: 95, 98); and it has been suggested that this property might serve to demarcate science and fiction (Sady 2012). Fleck also argues that, as time goes on, scientific knowledge becomes progressively more dense with passive associations, and researchers feel more and more constrained (1979: 83-84). In this thesis, I identify objectivity with a reduction in freedom to choose how things are. Therefore, progressively increasing resistance can be related to progressively increasing objectivity. This is a complex issue, not least because in my case study I would describe the transition from Hope to Mackenzie involves a significant reduction in the density of passive associations. And yet this is still a transition that I would want to describe as progressive. Developing an account of objectivity by degree, and of how progress can occur despite a reduction in the density of passive associations, is beyond the scope of this thesis, and will be left for future work.
of theoretical models that they know are inaccurate for ease of calculation. So, for instance, scientists working on the Apollo space program used Newtonian mechanics in their calculations, despite their knowing that Newtonian mechanics were strictly incorrect. They deliberately made use of what they knew was a little bit fictional to assist them in their work. Charles Dickens, when he wrote the fictional story *Oliver*, was perhaps doing something similar. As Dickens was making a social commentary on the plight of orphans in Victorian London, he was constrained (at least a little bit) by his observations of the way orphans were treated in this period. So, when Oliver asks the Beadle “Please Sir, I want some more”, Dickens cannot have the Beadle reply “Of course you can have more, there is plenty for all”. Dickens, then, is also using fiction as a device to help him in his work (which I take to be a social commentary on the plight of orphans in Victorian London). The Apollo scientists’ work is mostly constrained by observation, and a little bit fictional, whereas Dickens’ work is mostly fictional, and a little bit constrained by observation. Thus science and fiction may be thought to exist on a continuum. Many myths and works of fiction, like parables, fables and allegories, contain life lessons for the audience to reflect upon. As such, they can be informative about the world in which we live, and may well contain some passive elements of knowledge in the sense in which I am interested. I raise this issue because I wish to highlight that my objections to Fleck are focused only on his claim that any set of active associations on their own can produce inevitable (passive) connections like “the atomic weight of hydrogen is 1.008” (if the atomic weight of oxygen is 16), and not on the possibility that science and fiction exist on a continuum.

Fortunately, the examples Fleck uses to illustrate his claim that passive associations (like “if oxygen is 16 then hydrogen is 1.008”) occur in any fairy tale
or myth do not support this claim. According to Fleck, passive associations occur in fantasies. “As any poet knows, a web of fantasy spun for long enough always produces inevitable, spontaneous and formal connections” (Fleck 1979: 101). This may be taken to suggest that as a story becomes more detailed and elaborate, the freedom of the characters in the story to do certain things that have not already been discussed in the story becomes restricted. So for instance if an author writes that a fictional character is very large, then this will mean that the character will be unable to fit into small spaces that we are told are only just large enough for a much smaller character. As the author makes some decisions about the size of the character, he or she loses the freedom to make other decisions about what the character can fit into. This has some superficial resemblance to the loss of freedom experience by researchers when they make decisions about the atomic weight of oxygen and then find that they lose the freedom to decide what the atomic weight of hydrogen is. But this resemblance is only superficial. Recall that in Fleck’s chemical example the stipulation of the atomic weight of oxygen was not by itself sufficient to determine whether or not the atomic weight of hydrogen was 1.008. The claim that the atomic weight of hydrogen is something else is perfectly consistent and compatible with the claim that the atomic weight of oxygen is 16. However, in the case of whether or not this fictional character can fit into a small space this is not so. The claim that the character can fit into small spaces conflicts with the claim that the character is very large. The conflict here is with the decisions already made by the author, and therefore with the active associations laid down in the work of fiction. There is no loss of freedom in addition to that imposed by the active associations laid down by the author.

Another way of looking at the difference between science and fairy tales is to consider a fictional group of scientists trying to measure the atomic weight of
hydrogen. The author of this tale can set everything up in the story just as it is in real life. The same apparatus can be used, the same recipes for making oxygen and hydrogen gas followed, the same procedures for weighing the gasses carried out. The atomic weight of oxygen can be stipulated as exactly 16. All this being given, is the author of such a tale now limited as to what the atomic weight of hydrogen can be? Is the author now not free to make the fictional research scientists find that the atomic weight of hydrogen is, for instance exactly 1? No. The author is still quite free to choose what the results of this fictional experiment are. Finding that the atomic weight of hydrogen is exactly 1 may even make a neater, more easily understood and therefore better story, as this conforms to Prout’s hypothesis. Indeed, authors have created worlds far more fantastic than a world where the only difference from ours was that if the atomic weight of oxygen is 16, then atomic weight of hydrogen is 1. In fiction, a few flashing lights and spinning discs makes time travel possible. In fiction, there is a wardrobe somewhere in England that acts as a portal to another world. In fiction, people can be fired from a gun barrel with sufficient velocity to reach the moon without being killed by the forces that produce the necessary acceleration. When writing fiction the authors do not necessarily experience the same loss of freedom as researchers do in real life.

Even if the logical consequences of adopting active associations are not sufficient to account for the loss of freedom experienced as the passive element of knowledge, Fleck argues that the constraining effects of the active associations are not limited to their logical consequences. Perhaps this is the additional constraint we have been seeking? So Fleck says that in Greek myth “Aphrodite cannot but be the wife of Hephaistos and the lover of Ares” (1979: 101). He also says that “[i]n a romance about chivalry, one cannot simply write “horse” instead
of “steed”, although these words are logical synonyms differing only in style” (Fleck 1979: 101). Fleck says the same applies to artistic work. “This we can easily demonstrate by placing part of a second painting over a good painting executed in a different style. The two parts would clash with each other, even if the two paintings were matched in content” (Fleck 1979: 101). Saying that Aphrodite was in a monogamous relationship with Hephaistos does not strictly contradict what is written in the rest of Greek myth. Using the word horse in a chivalric romance would not contradict what is written in the rest of this body of work. Overlaying a realistic painting with part of an impressionistic one of the same scene would not make the subject of the painting incoherent, as overlaying this realistic painting with another realistic painting of a different subject would. Nevertheless, Fleck is right to say that in each case there would be a clash with the character of what has gone before.

Even so, none of these examples capture the additional loss of freedom connected with the passive element of knowledge above. In the case of Greek myth, it would indeed be out of character for Aphrodite not to take a lover\textsuperscript{115}. Aphrodite was forced to marry Hephaistos by her father Zeus. To have her not take a lover would be to condemn the goddess of love to a loveless marriage. Nevertheless, the constraint storytellers feel that prevents them from saying that Aphrodite did not take Ares as a lover (given the character of Greek myth) is not the same as the constraint that researchers feel that prevents them from finding

\textsuperscript{115} Other readings of Fleck’s use of Greek myth are possible. For example, the myth of Aphrodite, Hephaistos and Ares might reflect an observation that beautiful young women who are compelled by their fathers to marry the boring village craftsman, tend to run off with a glamorous soldier. To the extent that this myth reflects this life experience, it contains passive associations like ‘if O = 16, the H = 16’. However, I struggle to read Fleck’s use of this myth in this way. Fleck does not discuss this myth in any detail, only referring to it as one of many possible examples of a “web of fantasy” (1979: 101). He refers to it in the same paragraph as his examples of chivalric romance and painting, which are concerned respectively with the character of the language and brushwork of these forms of art. Consequently, I read Fleck as being focused on conflicts with the character of the canon of Greek myth.
that the atomic weight of hydrogen is 1.008 (given that the atomic weight of oxygen is 16). As discussed above, for hydrogen to have an atomic weight of something other than 1.008 would not have conflicted with the character of the rest of the chemical research that had been done up until the late nineteenth century. Indeed, for those chemists who expected Prout’s hypothesis to be true it was the finding that the atomic weight of hydrogen was 1.008 clashed with the character of chemistry in this time. In Fleck’s Greek myth example, the conflict is still with the active associations already accepted as canon, even if it is not with the logical consequences of those active associations.

In the chivalric and artistic examples the situation is slightly different. To deny that Aphrodite took a lover is to change the content of Greek myth. According to Fleck, to use the word horse in a chivalric romance, or to change from realism to impressionism, is not to change the content of these pieces of work, but rather to change their style. The same content is being expressed in a different way\textsuperscript{116}. This is equivalent to changing how the results of a scientific experiment are reported, rather than changing what these results are. So, for instance, instead of reporting the results of the experiment to measure atomic weights as (1)\textsuperscript{117} “if the atomic weight of oxygen is 16 then the atomic weight of hydrogen is 1.008”, this result is reported as (2) “if the atomic weight of hydrogen is 1 then the atomic weight for oxygen is 15.873”. There is a clash here because the values for the

\textsuperscript{116} I am using the controversial distinction between content and language here because Fleck makes use of it. I want to argue that even if this distinction is adopted the passive element of knowledge that Fleck identifies in fairy tales and myth is still not the same as the passive element of knowledge he identifies in science. I also think that this is actually quite a good way to describe the discussion over whether hydrogen or oxygen should be the standard for atomic weight. The historical actors involved in this argument understood it as an argument over what language to use to express the same empirical findings (see section 6 below).

\textsuperscript{117} For clarity I will number the claims discussed in this paragraph. These numbers apply to this paragraph and the next paragraph only.
atomic weights of oxygen and hydrogen differ. However, the clash is not the same as that described as the passive element of knowledge above.

Above I argued that in Fleck’s chemical example researchers were not free to make claims like (3): “if the atomic weight of oxygen is 16 then the atomic weight of hydrogen is 1”, as this conflicted with the empirical finding that (1) “if the atomic weight of oxygen is 16 then the atomic weight of hydrogen is 1.008”. The conflict between claim 1 and claim 2, which is equivalent to the sort of conflict that Fleck identified in fiction, is not the same as the conflict between claim 1 and claim 3, which Fleck identified in science. This is not to say that conflicts like that between claim 1 and claim 2 do not happen in science. The late nineteenth and early twentieth century researchers into atomic weights did have long and protracted arguments about whether oxygen or hydrogen should be used as the standard atomic weight. In the last chapter I described how Poincaré thought that historical arguments about whether the earth turned around were similarly content free. I am happy to concede conflicts like that between claims 1 and 2 may well occur in science. I am also happy to concede to both Fleck and Poincaré that the researchers having arguments like these often feel as though they are disagreeing about the content of science, rather than about the style or language in which this content is experienced or expressed. Even so, in situations where the content of what is claimed is judged to be the same, a disagreement about how to express those claims is solely a disagreement about the active associations used to produce these claims. Such disagreements only feel passive and “substantial” because the researchers having them are not aware of the active element of knowledge that constrains them through the collective will of their research community. In contrast to this, the passive element of knowledge identified in Fleck’s chemical example and discussed in section 1.2 above is more
than a feeling, and does not result solely from the collective will of the research community. Conflicts like that between claim 1 and claim 3 are not the result of simply adopting active associations, and consequently do not have to occur in fictional work.

Fleck therefore developed (and conflated) several different accounts of the passive element of knowledge, not all of which are of value to me in this context. Sometimes he referred to passive associations as things that arise solely from the adoption of active associations. This sort of passive association is therefore fully determined by the collective will of a community of researchers, and is fully explicable in terms of cultural history and collective psychology. As I have said, the sort of constraint that I am looking for is in addition to that provided by the active element of knowledge. In contrast to Fleck, Poincaré never argued that producing a system of conventions on its own was sufficient to produce scientific facts. Poincaré argued, for instance, that there were no facts about whether or not the laws of a game were true, because all the laws of a game are stipulated by people (Poincaré 2001b: 320). “[I]t is clear that if all the laws had been transformed into principles nothing would be left of science” (Poincaré 2001b: 332). Poincaré never argued that scientific facts could be produced in the absence of the additional loss of freedom described. As I have shown above, Fleck did identify an account of the passive element of knowledge that is accompanied by this additional loss of freedom. Nevertheless, he did not distinguish this account of the passive element of knowledge from the others that he suggested. Neither did he argue that this account of the passive element of knowledge could be understood as a form of objectivity. Poincaré, however, did

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118 Poincaré says that the rules of “tric-trac” (a game like backgammon) “rely on proof by universal consent” (2001b: 320). They are whatever the community of people who play the game agree that they are.
suggest how the passive element of knowledge can be understood as a form of objectivity.

5 – The passive element of knowledge and objectivity
Fleck held that passive associations were not explicable in terms of cultural history or collective psychology. “For this very reason these seem to be “real”, “objective” and “true” relations” (Fleck 1979: 10). I want to highlight that for Fleck the passive element of knowledge only seemed to be objective, true and real, as opposed to actually being objective, true and real. This is because Fleck held that passive elements of knowledge could only be produced following the adoption of active elements of knowledge, and therefore that all passive facts were conditioned by cultural history and collective psychology. “Members of different scientific communities live in their own scientific and also professional reality” (Fleck 1986a: 49). As discussed in the previous chapter, Fleck regarded the notion of “things in themselves”, as they are apart from observers, as unintelligible (Fleck 1990: 267; 1979: 28). Consequently, he regarded all talk of an objective reality (or the nature of the world in itself) as unintelligible, and only to be excused for grammatical reasons (1990: 267). Nevertheless, Poincaré did propose an account of objectivity that can be applied to Fleck’s account of the passive element of knowledge discussed above, and which is valuable to me here.

As discussed above, Poincaré tried to protect himself against the charge of extreme relativism by developing an account of crude facts, which are forced upon observers by the way the world is. I, along with Fleck, have abandoned this prospect. And yet, Poincaré also suggested other ways in which an account of objectivity can be defended. Instead of understanding objectivity as capturing the true nature of things, Poincaré suggested that objectivity should be understood as capturing the true relations of things.
Poincaré agreed with Fleck that science does not capture how the world is in itself and apart from the knower, as conventions always have to be adopted to produce scientific knowledge. He viewed all knowledge of scientific objects as knowledge of things as they appear to us through the use of conventions, and not as knowledge of things in themselves. Consequently, Poincaré viewed questions about “the true nature of things” as unintelligible:

“To the first question, no one would hesitate to reply no; but I think we may go further; not only science cannot teach us the nature of things; but nothing is capable of teaching it to us and if any god knew it, he could not find words to express it. Not only can we not divine the response, but even if it were given to us, we could understand nothing of it; I ask myself even whether we could understand the question.

When, therefore, a scientific theory pretends to teach us what heat is, or what is electricity, or life, it is condemned beforehand; all it can give us is only a crude image. It is, therefore, provisional and crumbling” (Poincaré 2001b: 347-348).

Nevertheless, even if questions about the true nature of things are unintelligible, this does not rule out the possibility of asking a second and slightly different question about how these conventionally defined objects relate to one another:

“The first question being out of reason, the second remains. Can science teach us the true relations of things? What it joins together should that be put asunder, what it puts asunder should that be joined together?” (Poincaré 2001b: 347-348).

Applying this to Fleck’s example of syphilis, I agree that the question “what is the true nature of syphilis?” cannot be answered, because the conventions/active associations we use to define the group of patients with syphilis are chosen by us and can change. I also agree that this question makes no sense, because it is equivalent to asking for a description of how syphilis appears to us that does not appeal to descriptions of how syphilis appears to us. And yet, even if asking about the true nature of an object (like a group of patients with a disease) is impossible,
we can still ask how these objects *relate* to each other once they have been conventionally defined. Once those patients with syphilis are defined, and once those patients who are cured following treatment with mercury are defined, it becomes possible to ask how these groups of patients relate to one another. Are they the same group of patients, or not? If researchers define which patients have syphilis, and define the procedure for the Wassermann reaction, it becomes possible to ask whether or not the Wassermann reaction accurately detects those patients with syphilis. Even if questions about the true nature of objects (like those patients with syphilis, those patients that respond to treatment with mercury, and those patients that produce a positive Wassermann reaction) are not intelligible or answerable, questions about how these objects of appearance relate to one another are perfectly intelligible and answerable.

Furthermore, Poincaré saw such relations as a source of *objectivity*. Even though he argued that claims about the nature of conventionally defined objects could not be objectively true or false, this did not apply to scientific facts about how these objects related to one another. In contrast to conventions, Poincaré argued that scientific facts could be objectively true:

> “[T]he enunciation of a fact can only be *true or false*. This is not so for any proposition; if this proposition is the enunciation of a convention, it cannot be said that this enunciation is true, in the proper sense of the word, since it could not be true apart from me and is only true because I wish it to be” (Poincaré 2001b: 324).

This way of understanding truth and falsehood is worth highlighting. A convention cannot be true or false because conventions are *both* not true apart from the knower and *are* only accepted because the knower wishes them to be. The key point here is that the status of being true apart from me and that of being true because I wish it to be are independent of each other. It is possible for knowledge
not to be true apart from the knower, and yet for it also not to be true because the knower wishes that it is. As discussed above, this is exactly the status of scientific facts and passive associations (when Fleck is read as a conventionalist). Conventions/active associations are necessary for the production of scientific facts/passive associations. Therefore, scientific facts/passive associations are not true apart from the knower. Nevertheless, the wishes of researchers as captured in conventions/active associations are not sufficient to account for the loss of freedom experienced as scientific facts/passive associations. Therefore, scientific facts/passive associations are not true because the knower wishes them to be. It is precisely because scientific facts/passive associations resist the will of the knower (as Fleck (1979: 95) puts it) that they have a right to be called objective truths. The relations between objects, then, can be a source of objectivity, even if objects themselves cannot:

“Therefore, when we ask what is the objective value of science, that does not mean: Does science teach us the true nature of things? But it means: Does it teach us the true relations of things?” (Poincaré 2001b: 347).

When read like this Fleck’s account of the passive element of knowledge not only seems to be an account of objective knowledge, but really is an account of objective knowledge. However, as Fleck produced and conflated a number of different accounts of the passive element of knowledge (as discussed above), his account of the passive element of knowledge must be read selectively in order to reveal this account of objectivity. It is only when Fleck is read as a conventionalist that this account of objectivity becomes apparent.

This account of objectivity can be used to show how Hope and Mackenzie’s work was at once historically contingent without thereby collapsing into an extreme form of relativism. Before I apply this conventionalism to Hope and Mackenzie, I
should address a potential objection to the conventionalist position I have outlined above. It is possible that this conventionalism is *trivial*, and does not matter in *practice*, and I need to argue that this is not the case.

6 – This conventionalism is not trivial: the choice of active associations is more than a choice of language in which to express facts

Even if doctors do have a choice to make about which active associations to adopt, this does not necessarily mean that adopting different sets of active associations will have an important effect on the *practice* of medicine. Indeed, conventionalists like Poincaré and Mach both thought that there was an important sense in which it did not really matter which conventions (or in Fleckian terms active associations) were used to produce scientific facts (or passive associations), because the choice of different conventions only resulted in the same facts being presented in a different language. According to Poincaré “*all the scientist creates in a fact is the language in which he enunciates it*” (Poincaré 2001b: 328). If this is the case then adopting a different set of active associations may only affect how patients are *talked about*, rather than how they are *managed*. **In my view this is not the case**, but the claim that the adoption of different active associations/conventions is equivalent to the adoption of a new language needs unpacking before I can defend this view.

The particular example that Poincaré had in mind when he developed the view that conventions and language were equivalent was the choice of whether to adopt Euclidean or non-Euclidean geometry in order to produce facts about the science of mechanics (Poincaré 2001a: 35-70). Poincaré maintained that although it was impossible to describe the facts of mechanics without adopting one form of geometry as a convention, it was nevertheless the case that all the results of a mechanical experiment could be presented using a different geometry
without any loss of content. “I challenge anyone to give me a concrete experiment which can be interpreted in the Euclidean system, and which cannot be interpreted in the system of Lobatschewsky. As I am well aware this challenge will never be accepted” (Poincaré 2001a: 62). Poincaré argued that the choice about which geometry to use was akin to the choice about whether to use the metric system or the imperial system of weights and measures, and akin to the choice of whether to use the Cartesian system of co-ordinates or the system of polar co-ordinates (Poincaré 2001a: 45). Weights, distances and locations can be equally well described no matter what choice is made, and Poincaré thought the same went for the choice of geometry (2001a: 45). The choice of convention could be better or worse only in the sense that it made the necessary calculation more or less simple to carry out. Scientists were supposed to choose the most convenient language in which to conduct their research, but their choice did not matter beyond issues of convenience (2001a: 45). I could illustrate what Poincaré meant by this by discussing the translation of mechanical observations from one geometry to another, but it is simpler for me to do this with reference to the chemical example Fleck used, as this has already been described.

As discussed above, the chemical example that Fleck used to illustrate the relationship between the active and passive elements of knowledge was “if 16 is assumed as the atomic weight for O, oxygen, of necessity the atomic weight of H, hydrogen, will inevitably be 1.008” (Fleck 1979: 83). Even if oxygen was used as the standard for the measurement of atomic weight in Fleck’s time, this was not inevitable. In the late nineteenth and early twentieth century researchers debated whether hydrogen should be used instead of oxygen as the standard, with the atomic weight of hydrogen stipulated to be exactly 1 (Holden 2004; Richards 1900). As discussed above, if the atomic weight of hydrogen is assumed
to be exactly equal to 1, then the measured atomic weight of oxygen will inevitably be 15.88 (see Clarke 1903: 4-5). Even though the stated results of the measurement of these atomic weights are different, there is an important sense in which they are the same result reported in a different language. Both are expressions of the relative weight of an atom of hydrogen to that of an atom of oxygen, but this result is expressed in each case using different conventions. As Fleck would have it, the content of these two claims is the same.

This view that the choice of active associations is equivalent to the choice of a language in which to express the same content is reflected in the nature of the early twentieth century debate over whether to use oxygen or hydrogen as the standard. The participants in this debate who favoured the use one standard did not argue about the accuracy of the results obtained by researchers using the other standard were inaccurate. Those researchers who favoured one standard did not accuse those who favoured the other of poor experimental technique or of being poor observers. Instead, each side argued that the use of the others’ standard would be inconvenient, just as Poincaré said they should.

Those who favoured the hydrogen standard argued that making the lightest substance (hydrogen) exactly equal to 1, and therefore making all the other measurement of atomic weights expressions of how many time heavier they were than this lightest substance, would be simpler for new students to understand (Richards 1900: 173-174; Holden 2004). Those who favoured the oxygen standard argued that, because the atomic weights of many elements were measured using reactions between these elements and oxygen, the use of oxygen as a standard was more convenient for the researcher (Richards 1900: 173; Holden 2004). If hydrogen was used as the standard, then researchers would have to know the relative weights of oxygen and hydrogen in addition to
the relative weights of oxygen and the element in question in order to work out the relative weight of the element to hydrogen. This would mean that every time the relative weight of oxygen to hydrogen was revised, a great many other atomic weights would also have to be revised at the same time (Richards 1900: 173; Holden 2004). Additionally, it just so happened that a greater proportion of measured atomic weights were close to whole numbers if oxygen was used as the standard instead of hydrogen, which made calculations easier to carry out (Richards 1900: 173). Thus, the arguments over whether to use oxygen or hydrogen as the standard for the measurement of atomic weights turned on considerations of convenience for teachers and convenience for researchers (Holden 2004).

At first neither standard commanded universal consent (Clarke 1903: 1). In the early twentieth century results for the atomic weights of elements were often published in both formats, with results of the same empirical work expressed in terms of the oxygen standard and then expressed in terms of the hydrogen standard appearing side by side (Clarke 1903: 4-5). It was left to practising chemists to figure out over time which standard was most convenient. “That standard which best serves to coördinate chemical and physical knowledge will ultimately be chosen, and the other will gradually fall into disuse” (Clarke 1903: 1-2). Eventually the oxygen standard gained acceptance as the most convenient way to express these empirical results, but this choice was not based on considerations of truth, but rather on considerations of convenience of expression and calculation. The example of the debate about how to report the measurements of atomic weight illustrates nicely why some conventionalists (like Poincaré and Mach) viewed the choice of active associations as a choice about
the language in which to express the same empirical content, to be made only with consideration of convenience of expression and the economy of thought.

In my view, talking about the difference between claims made using the hydrogen standard and claims made using the oxygen standard as a difference in language rather than a difference in content is reasonable. With it in mind, I can see the value in the distinction between language and content that was developed and used by some conventionalists. Nevertheless, changes to conventions are not always equivalent to changes in language, because changes to conventions often result in changes to the practice of science.

A key reason that some conventionalists (like Poincaré and Mach) thought that a change to conventions was equivalent to a change in language was that they thought that there was an important sense in which changing the conventions or language used to express scientific facts did not matter to the practice of science. Fleck’s chemical example can again be used to illustrate this. Many rockets are fuelled by burning hydrogen and oxygen together to produce water vapour. Say that I am making a rocket that will carry one metric tonne of liquid hydrogen fuel, and I need to work out how much liquid oxygen fuel I need. To make this calculation it is helpful to work out the weight of liquid oxygen that contains the same number of oxygen atoms as there are hydrogen atoms in the one tonne of liquid hydrogen (this result can then be halved to calculate the weight of liquid oxygen I need, as to produce a molecule of water (H₂O) I need two atoms of hydrogen to combine with one atom of oxygen). This is equal to one tonne multiplied by the number of times heavier an oxygen atom is than a hydrogen atom. The key point is that is does not matter whether I carry out this calculation with results expressed using oxygen as a standard or hydrogen as a standard. Using oxygen as a standard, my working would be 1 times 16 divided by 1.008.
Using the hydrogen standard, my working would be 1 times 15.88 divided by 1. Either way I find that 15.88 tonnes of liquid oxygen has the same number of atoms as 1 tonne of hydrogen, and therefore that I need 7.94 tonnes of liquid oxygen to fuel my rocket. Whether I do this calculation using the oxygen or hydrogen standard matters no more to the final result than does doing this calculation using pounds instead of tonnes to measure the weight of the fuel. The result in either case is the same, so long as I am consistent with my use of convention throughout my calculation.

The view that conventions do not matter to the practice of science is expressed very clearly by Mach. Mach pondered the question of why it is that physicists treat electricity as a *substance*, whereas they treat heat as a *motion*. He argued that this was because of the conventions that physicists had come to adopt for historical reasons, and that they could just as well treat electricity as a motion, and heat as a substance (Mach 1910: 44-46):

“What, now, is the reason of this difference of view in our treatment of heat and of electricity? The reason is purely historical, wholly conventional, and what is still more important, wholly indifferent” (Mach 1910: 44 [my emphasis]).

My goal here is not to defend Mach’s (and Poincaré's) view that our understanding of heat as a motion, and electricity as a substance, is conventional. I only want to draw attention to the view (held by Mach, and with which I disagree) that changing conventions/active associations is *wholly indifferent* to the practice of science.

It is not *prima facie* unreasonable to think that this might be the case in medicine. Consider the toy example of a group of patients with a disease – X. These patients (X) receive a treatment for their disease – A. Now consider the same
group of patients (X), but described in a different way. Split this group (X) up into
two separate groups – Y and Z. Let this be equivalent to adopting a different set
of diagnostic conventions, so that instead of having one group (X), all of whom
have the same disease, instead we have two smaller groups (Y and Z), who have
two different diseases. This change in diagnostic convention has apparently
produced a change to the management of the patients. And yet, this need not be
the case. If the patients in Y and Z still all receive treatment A, then there is a
sense in which this change in diagnostic convention has not led to a change in
the management of the patients. All that has happened is that the language used
to describe the management of these patients has changed, as the same patients
still get the same treatment. If this is the only consequence of adopting different
sets of conventions/active associations, then doctors need not worry very much
about these choices. All that need be done is for someone to occasionally review
diagnostic terminology and try to find the most important means of expressing the
same clinical information.

Even if it might be the case that a change to the active associations/ conventions
used to produce disease categories does not affect the management of patients,
this is not necessarily so. Consider a situation where the patients are diagnosed
with two separate diseases and split into groups Y and Z, but this time it is only
patients in Y that receive treatment A. Let the patients in Z get a different
treatment – B. In this new scenario the patients are managed differently depending on whether they are diagnosed as members of group X, or diagnosed
as members of group Y or Z. In this new scenario, the adoption of different sets
of diagnostic conventions does affect the management of patients. Therefore, it
is important that doctors pay attention to their diagnostic conventions in this
scenario. I would argue that the transition from Hope’s understanding of heart
disease to Mackenzie’s understanding of heart failure is like this second scenario, and unlike the first.

7 – The objectivity in Hope and Mackenzie’s work
Recall from the last chapter that Hope thought that the claim that auscultation could be used to identify valvular heart disease with “almost complete certainty” was a brute fact (Hope 1833: 336). Recall also that this fact could only be produced following the active association of heart disease and lesions of the heart valves found at post mortem examination. So, for Hope, to find pathology of the valves post mortem was to find heart disease. Consequently, doctors who accepted this active association between heart disease and valvular lesions lost the freedom to say that heart disease was not associated with valvular lesions.

Hope claimed to be able to detect heart disease by detecting murmurs heard at auscultation. As discussed in chapter 4, Hope went to some lengths to demonstrate that he could in fact predict which patients had valvular pathology (and therefore heart disease), and that he could even predict the specific type of pathology that would be found at a particular valve, from the sounds he heard at auscultation. If Hope could really do this, then he had found that heart disease and sounds heard at auscultation were associated, and those physicians who were sceptical about the diagnostic power of the stethoscope lost the freedom to claim that there was no such association.

It was only following a self-imposed constraint, in the form of the association of heart disease and valvular lesions, that Hope was able to experience a further constraint, in the form of the association of heart disease and the sounds heard at auscultation. Although this active association is necessary for the production of the fact that auscultation was a powerful diagnostic tool, it is not sufficient to account for this further constraint. The finding that auscultation could not be used...
to predict the valvular pathology discovered post mortem does not contradict and is not incompatible with the active association of heart disease and post-mortem findings. Indeed, this is what Hope’s opponents expected to be the case, and what Laennec actually found to be the case. This further constraint cannot be accounted for by the active associations alone. If this constraint is as Hope describes it, and he can do what he said he could do, then he has every right to call it a fact.

The same is true for Mackenzie’s claim that the view that “the sounds of a healthy heart must be clearly struck and free from murmurs is not based on accurate observation” (Mackenzie 1916: 101). Recall from the previous chapter that Mackenzie could only produce this fact following his acceptance of the active association between heart disease and a poor prognosis. So for Mackenzie, if the patient did not have a poor prognosis then the patient did not have a heart disease. Consequently, Mackenzie lost the freedom to claim that heart disease was not associated with a poor prognosis. Mackenzie claimed that he had encountered many patients of all ages who, despite having heart murmurs and doing strenuous work for many years, never became ill with the symptoms of heart disease (e.g. with breathlessness and fluid accumulation). If this was the case, and there really were (and perhaps are) many patients who had murmurs and never become ill, despite years of strenuous work, then Mackenzie lost the freedom to claim that patients with murmurs had heart disease. At the same time, if there really were many such patients, those physicians that claimed that a patient with a heart murmur would almost invariably become ill with the symptoms

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119 Recall from chapter 4, section 3, that Laennec was trying to associate particular pathologies with particular sounds. Hope was not trying to do this, and thus came to a different conclusion.
of heart disease if they did not keep their circulation “tranquil” lost the freedom to make this claim.

It was only following a self-imposed constraint, in the form of the association of heart disease and a poor prognosis, that Mackenzie was able to experience a further constraint, in the form of the lack of association between heart disease and heart murmurs heard at auscultation. Although this active association was necessary for the production of the fact that auscultation was not a powerful diagnostic tool, it is not sufficient to account for this further constraint. The finding that the detection of murmurs at auscultation could not be used to predict which patients had a poor prognosis does not contradict and is not incompatible with the active association of heart disease and a poor prognosis. Indeed, in Mackenzie’s time, the view that patients with murmurs would almost invariably go on to develop breathlessness and fluid accumulation if they did not keep their circulation tranquil was commonly held, not least by very influential doctors like Clifford Allbutt. This further constraint cannot be accounted for by the active associations alone. If this constraint is as Mackenzie describes it, and there really were many patients with heart murmurs who never became ill with the symptoms of heart disease despite many years of strenuous work, then he had every right to call it a fact.

Hitherto I have concentrated on the production of facts about the diagnostic power of auscultation, but this insight about how the making of individual or collective decisions (the active element of knowledge) produces an additional loss of freedom (in the form of the passive element of knowledge) applies to all the other fact Hope and Mackenzie produced as well. It is worth discussing the production of two further facts to substantiate this claim.
As discussed in chapter 4, Hope found that heart disease was associated with the symptoms of breathlessness and fluid accumulation (Hope 1833: 24, 205, 215, 346). Hope produced this fact by examining symptomatic patients in a hospital setting shortly before they died, and then carrying out a post-mortem examination. As has also been discussed, Corvisart argued that it was possible to predict the presence of valvular lesions of the heart using the symptoms and signs with which the patient presented, including the presence of breathlessness and fluid accumulation. These early nineteenth century researchers were engaged in research that attempted to correlate the results of the ante-mortem physical examination with the results of the post-mortem examination. They found that many of the patients with heart lesions, including enlargement of the heart and valvular lesions, had breathlessness and fluid accumulation. But this association between heart lesions and the symptoms of breathlessness and fluid accumulation does not in and of itself establish that heart disease and the symptoms of breathlessness and fluid accumulation are associated. And yet, as discussed in the last chapter, Hope (and Corvisart) had the strong intuition that to have a valvular lesion of the heart simply was to have a heart disease. Only after these early nineteenth century researchers had made the active association between valvular lesions and heart disease, was it possible to find that heart disease and the symptoms of breathlessness and fluid accumulation were passively associated.

These researchers made an active association between heart disease and valvular lesions of the heart, and as they did so they lost the freedom to claim that heart disease was not associated with valvular lesions of the heart. Following this self-imposed constraint, they then could experience an additional constraint - that many of the patients with heart disease (at least in a hospital setting)
presented with breathlessness and fluid accumulation. Although this active association was necessary for the production of this additional constraint, it is not sufficient to account for it. Holding the view that patients with heart disease do not commonly present with breathlessness and fluid accumulation does not clash with the view that patients with valvular lesions have heart disease. It would be quite possible to write a work of fiction where this is true. This further constraint cannot be accounted for by the active associations alone. If it is as Hope describes it, and most patients who presented to hospitals with heart disease (e.g. lesions of the heart obstructing the circulation) were “short winded” and/or had “dropsy”, then he had every right to say that the claim that breathlessness and fluid accumulation were signs of heart disease was a fact.

In chapter 2, and chapter 5 section 4, I described how Mackenzie produced the fact that there must be occult heart disease, where “seemingly healthy heart muscle is the seat of changes too subtle for our present methods to detect” (Mackenzie 1913: 7-8). Mackenzie also produced this fact by examining symptomatic patients before they died, and using the post-mortem examination to look for lesions to the heart. Mackenzie found that many patients with symptoms such as breathlessness, fluid accumulation and chest pain, did not have heart lesions that were detectable at post-mortem examination. He argued that this finding forced us all to conclude that there must be heart disease that could not be detected in his time. But this lack of association between heart lesions and the symptoms of breathlessness and fluid accumulation does not in and of itself establish that heart disease and the symptoms of breathlessness and fluid accumulation are associated. And yet, as discussed in a previous chapter, Mackenzie had the strong intuition that to have breathlessness and fluid accumulation (particularly in the absence of any pathology of the kidney or liver
that could explain these symptoms) simply was to have a heart disease. Only after Mackenzie had made the active association between these symptoms and heart disease, was it possible to find that heart disease and absence of heart lesions were passively associated.

Mackenzie made an active association between heart disease and symptoms like breathlessness and fluid accumulation, and as he did so he lost the freedom to claim that heart disease was not associated with these symptoms. Following this self-imposed constraint, he then could experience an additional constraint – that many of the patients with heart disease did not have detectable heart lesions. Although this active association was necessary for the production of this additional constraint, it is not sufficient to account for it. Holding the view that patients with heart disease do always have detectable heart lesions does not clash with the view that patients with symptoms like breathlessness and fluid accumulation have heart disease. It would be quite possible to write a work of fiction where this is true. This further constraint cannot be accounted for by the active associations alone. If Mackenzie was correct to say that many patients with heart failure (diagnosed from the presence of symptoms like breathlessness and fluid accumulation) did not have detectable heart lesions at post-mortem examination, then he had every right to say that the claim that these patients had occult heart disease was a fact.

Both Hope’s work and Mackenzie’s work also show that this conventionalism is not trivial. Recall that Hope (1833: 326, 367) advised that patients with an obstruction of their circulation (as evidenced by murmurs detected at auscultation) should lead a “scrupulously tranquil life”. Hope warned that if these patients did not do this, their hearts would inevitably enlarge, they would develop symptoms of breathlessness and fluid accumulation, and their disease would
quickly lead to a premature death (1833: 367). Recall also that late nineteenth century doctors gave very similar advice. As discussed in a previous chapter, Broadbent admitted that he was proud to have “liberated” many patients with valvular lesions from orders not to play games or take exercise, or even to go upstairs or walk uphill (Broadbent 1897: 90-91). Broadbent himself advised patients with murmurs to avoid prolonged or violent exertion, as he believed this would damage their hearts further (1897: 47). Mackenzie also reported that people with were being denied the opportunity to serve in the military, made to pay higher premiums for their life insurance, and advised to restrict their level of physical exertion whilst at work or at play (1916: 99). The point is that the active association of heart disease with valvular lesions of the heart had consequences for patients, because these patients were understood to be a group in need of treatment.

Recall that Mackenzie objected to the association of heart disease with valvular lesions (1913: 8). He argued that many patients with valvular lesions and murmurs did not in fact have heart disease (1911a: 795; 1916: 101). Instead he argued that patients with heart disease should be detected by attending to the patient’s symptoms (particularly progressively worsening breathlessness and fluid accumulation) (Mackenzie 1916: 44-45). In other words, Mackenzie changed the active associations he used to diagnose heart disease. Notice also that the patients Mackenzie argued had heart disease were not the same patients as many of his contemporaries argued had heart disease. Many late nineteenth century doctors thought that patients with murmurs but without progressively worsening breathlessness had heart disease, whereas Mackenzie did not. Mackenzie thought that many patients with progressively worsening breathlessness had heart disease, and he argued that many of his
contemporaries did not (1913: 18; 1916: 49-50). What is key here is that Mackenzie felt that he needed to make this change precisely because he was unhappy with the way patients with murmurs were managed in his time. Mackenzie thought that many patients with murmurs had their lives unnecessarily restricted because his contemporaries associated heart disease with valvular lesions and murmurs. He associated heart disease with progressively worsening breathlessness because he thought this allowed for more accurate prognostication (Mackenzie 1916: 45), and therefore focused attention on the patients that actually needed treatment. He changed the active associations he adopted precisely because he wanted to change how patients were managed.

In the longue durée history, other changes to active association also affect the management of patients. Harrison (1962: 1379) argued that heart failure should not be defined as a condition resulting from disease of the heart muscle itself, to the exclusion of other cardiac causes of the syndrome of breathlessness and fluid accumulation. He did this on the grounds that some patients with congestive symptoms due to structural heart disease of the endocardium (the inside lining of the heart) or pericardium (the sac in which the heart sits) would not be diagnosed with heart failure if this definition was adopted. The key point is that Harrison (1962: 1379) was concerned that this change to the definition of heart failure (and therefore a change in active associations) would lead to the confusion of doctors and the mismanagement of patients.

Recall also Vasan and Levy’s (2000) objections to the European Society of Cardiology (ESC) criteria for the diagnosis of heart failure. They discussed the hypothetical example of an elderly lady, who presented with breathlessness and fluid accumulation, but without echocardiographic evidence of cardiac dysfunction (Vasan and Levy 2000). They argued that the application of the
ESC’s criteria, which required evidence of cardiac dysfunction in addition to symptoms and signs, would fail to diagnose this woman with heart failure and thus lead to her mismanagement. They argued that “a normal LVEF should not be used to reject a diagnosis of CHF [congestive heart failure] if the clinical presentation is convincing” (Vasan and Levy 2000). In Fleckian terms, they argued that making an active association between heart failure and certain measures of cardiac dysfunction would lead to the mismanagement of patients.

As these examples show, changes to active associations often do have important consequences for the management of patients. In opposition to Mach (1910: 44), I argue that changes made to the conventions adopted are not wholly indifferent to the practice of medicine, as they often matter a lot. A change to the active associations that are adopted by a group of practitioners is often very significant to the practice of medicine, and therefore quite unlike presenting the same medical knowledge in a different way. In opposition to Poincaré’s (2001b: 328) view, by changing active associations/conventions researchers create much more in a fact than the language in which they enunciate it.

**Conclusion**
Several scholars have argued that Fleck advocated an extreme form of relativism, where facts are whatever researchers decide they are. In such an epistemology, knowledge is fully determined by social, cultural and historical factors. Fleck denied this, but his critics have found his counter-argument unconvincing. Fleck’s critics do not see how the claim that facts are completely determined within a thought style is any different to the claim that facts are completely determined by social, cultural, and historical factors alone. Even though Fleck explicitly says that any attempt to account for facts using these factors alone always leaves out “something inevitable, steadfast and inexplicable by historical development”
(Fleck 1979: 79), his critics argue that he thereby contradicts himself. Fleck’s critics simply do not see how Fleck can consistently maintain that all facts are at once historically contingent whilst at the same time claiming that they are determined by something other than historical, cultural and social factors. In other words, Fleck’s critics cannot see how facts can at once be invented and discovered.

I have argued that Fleck’s epistemology does show how facts can at once be invented and discovered. The key to seeing this is to read Fleck as a kind of conventionalist (in the vein of Henri Poincaré), and thereby to attend closely to the relationship between the active and passive elements of knowledge. I have paid particular attention to the different ways in which knowledge production is constrained in each of these elements of knowledge. Using Fleck’s example of the measurement of the atomic weight of hydrogen, I have argued that the constraint provided by the active element of knowledge is entirely self-imposed. This means that the researchers measuring the atomic weight of hydrogen in the late nineteenth century chose to stipulate that the atomic weight of oxygen should be exactly 16 atomic weight units. As a result of this stipulation, these researchers lost the freedom to say that the atomic weight of oxygen was a different number, like 14 or 15. Nevertheless, this loss of freedom was of their own choosing. The same can be said for the collective (and unconscious) decision made by sixteenth century physicians to define syphilis as the carnal scourge, as opposed to any of the other definitions of syphilis that have been used historically. Whether researchers are aware of it or not, the glue that holds things in the active element of knowledge together is the researchers’ collective will.

This is not the case for many of the things Fleck refers to as passive associations. Without stipulating that the atomic weight of oxygen was exactly 16 (or at least
without making some other active association), it would have been impossible for these late nineteenth century to measure the atomic weight of hydrogen. Without defining syphilis as the carnal scourge (or at least without defining syphilis in some way), it would have been impossible to make observations about how patients with syphilis responded to treatment with mercury. As Fleck says, without making active associations, it is not possible to produce passive associations. Furthermore, adopting different active associations produces different passive associations. Active associations are necessary for the production of passive association. However, they are not sufficient. It is not the stipulation that the atomic weight of oxygen is 16 that determined the result that the atomic weight of hydrogen was 1.008 (as opposed to 1 or another number). It was not the stipulation that syphilis was the carnal scourge that determined the finding that sometimes treatment with mercury make the carnal scourge even worse (as opposed to always making the condition better). In addition to the constraints these researchers imposed upon themselves, they also experienced an additional constraint that does not result from the social, historical and cultural factors than account for the active element of knowledge. Both Fleck and Poincaré argue that without this additional constraint, science and medicine could not prove useful. As we constrain ourselves, we experience an additional loss of freedom. When read as a conventionalist, Fleck’s passive element of knowledge is this additional loss of freedom.

However, Fleck is not always consistent in his account of the passive element of knowledge. He sometimes claimed that the passive element of knowledge would arise solely as a result of laying down active associations. So, for instance, he said that the passive element of knowledge must arise even in fairy tales. Fleck also says that the adulterous nature of Aphrodite, the vocabulary available to
authors of chivalric romance, and the inability to change artistic style in the middle of painting a picture, are all also examples of things that are constrained in the same way as the passive element of knowledge in the sciences. I have argued that this is not true. Although these are constraints imposed upon the people working in these disciplines, these constraints arise solely from the active associations already adopted in these disciplines. There is no additional constraint in these examples. The additional loss of freedom that is experienced in science and medicine need not arise in fiction.

So it is only when Fleck is read selectively, and as a conventionalist, that an account of the passive element of knowledge as an additional loss of freedom becomes available. Another advantage of reading Fleck as a conventionalist is that this allows the passive element of knowledge to be read as a version of objectivity. Even though the use of conventions to produce scientific laws meant that they were not true apart from the knower, Poincaré argued that they should still count as objective because they were not true simply because the knower wished them to be. Conventions are necessary, but not sufficient, for the production of scientific laws. In exactly the same way, active associations are necessary, but not sufficient, for the production of facts. When Fleck is read as a conventionalist, the passive element of knowledge does not obey the researcher’s will, and hence should count as a version of objectivity.

As the active element of knowledge is necessary for the production of facts about disease in the passive element of knowledge, historical, social and cultural factors are necessary for the production of facts about disease. All facts about disease are therefore historically contingent, and at least partly invented. And yet, as the active element of knowledge is not sufficient for the production of facts. The additional constraint experienced as the passive element of knowledge is not fully
determined by the historical, cultural and social factors that determine the active element of knowledge. As the passive element of knowledge does not obey researchers' will, all facts about disease are therefore at least partly discovered. Facts about disease are therefore simultaneously invented and discovered. Fleck’s critics are therefore wrong to say that Fleck contradicts himself when he claims that all facts are at once historically contingent whilst at the same time claiming that they are determined by something other than historical, cultural and social factors.

I have also argued that the conventionalist epistemology discussed here is not trivial. The changes to facts produced caused by changes to conventions/active associations adopted do not reduce to trivial changes in language used to describe those facts, because these changes result in changes to scientific practice. I have argued (using work from Hope, Mackenzie and other researchers from the longue durée history) that in medicine adopting different conventions/active associations leads to changes to how patients are managed. Such a conventionalism should not be considered trivial.

I have applied these insights to two facts produced by Hope and a further two produced by Mackenzie. In Hope’s case I focused on the facts that the stethoscope is a powerful diagnostic instrument, and that breathlessness and fluid accumulation are signs of heart disease. In Mackenzie’s case, I focused on the facts that the stethoscope is not a powerful diagnostic instrument, and that there must be occult heart disease. In each case, these facts could not be produced without first adopting active associations. Hope made the active association between heart disease and valvular lesions of the heart, whereas Mackenzie made the active association between heart disease (or more properly, heart failure) and a syndrome of breathlessness and fluid accumulation. Having
constrained themselves by making these active associations, these doctors experienced an additional loss of freedom with respect to facts about whether or not breathlessness and fluid accumulation were signs of heart disease (in Hope’s case), about whether or not there was occult heart disease (in Mackenzie’s case), and whether or not the stethoscope was a powerful diagnostic instrument (in both cases). These facts can be understood as invented and discovered, historically contingent and yet objective, and there is no contradiction in saying this.
Conclusion

The methods employed in this thesis have been successful. Paying careful attention to medical practice has revealed that the commonplace assumptions about the function of the heart are problematic. Using the medical literature to trace the development of present day knowledge of heart failure has produced an historical account of this disease that is quite different to the two already available (Cowie and Holland 2002; Jarcho 1980). Rather than being an account of how this knowledge has developed towards present day views, which are taken to be timelessly correct, my work emphasises the historical contingency of medical knowledge of heart failure. I thus bring heart failure into the fold of diseases that have been treated using historicist-conceptualist historiography (Wilson 2000). In doing this I have drawn attention to the interest many early nineteenth century doctors, including Hope, had in what Corvisart called “pathological physiology”; and to the role this played in developing explanations for their patient’s symptoms, in prognostication, and in the production of therapeutic advice. I have argued that Mackenzie’s occupational role as a general practitioner and his early interest in the study of pain, were of central importance to his research program. This thesis has contributed to the scholarship on James Hope and James Mackenzie.

In addition to historicizing heart failure, I have also been sensitive to the concerns of many scholars that recognising the historical contingency of knowledge leads to an extreme, and therefore unacceptable, relativism (Boghossian 2006; Seidel 2014). To assuage these concerns, I have appealed to Ludwik Fleck’s epistemology, which I have argued is capable of showing how medical knowledge is at once invented and discovered. I have not, however, simply used Fleck’s work to address these epistemic concerns about the consequences of historical
contingency. I have also used this historical work to inform my reading of Fleck’s work. The meaning of Fleck’s account of the active and passive elements of knowledge has hitherto been obscure to many scholars, and I have used this historical work to draw out a valuable epistemology from Fleck’s work. In this thesis, philosophical reflection has informed my account of what happened historically, just as history has been used to inform my philosophical work.

It has not been possible to directly address the concerns of absolutist philosophers like Boghossian (2006) and Seidel (2014) in this thesis. Even so, my arguments that medical knowledge is at once invented and discovered point to a way in which relativist epistemologies need not collapse into extreme and unhelpful forms of relativism, as these philosophers argue. My thesis may also be able to support the arguments made by philosophers such as Helen Longino, who also tries to stake out an epistemic position between “empiricist” and “wholist” poles (1990: 20-37). Longino says that her aim is “to show both how social and cultural values play a role in scientific inquiry and how broadening our conception of that inquiry from an individual to a social activity enables us to see that the sciences are not, nevertheless, hopelessly subjective” (Longino 1990: 37). Longino’s argument is that the social practice of intersubjective and transformative criticism of the background assumptions that permits objectivity, and prevents her epistemology slipping into an extreme relativism (1990: 64-82).

This may be complemented by my argument that it is attention to an additional loss of freedom over and above the constraint imposed by social and cultural factors that prevents this slippage. Future avenues for research therefore emerge from my thesis.

In the introduction to this thesis, I ask three questions about the epistemic status of medical knowledge. 1) Is medical knowledge about the diagnosis of heart
failure historically contingent? 2) Does the historical contingency of this medical knowledge mean that it is impossible to make progress in medical practice? 3) How does the historical contingency of medical knowledge affect discussions about the development and evaluation of medical knowledge? These questions have framed the historical and philosophical investigations carried out in this thesis. By way of conclusion, it is useful to address them explicitly.

I have drawn attention to a number of philosophers and historians who hold that at least some aspects of medical knowledge are not historically contingent. In the introduction, I discussed Paul Thagard’s work on the development of medical knowledge. Thagard describes himself as a medical realist, who holds that diseases and their causes are mind independent entities, of which researchers can gain knowledge (1999: 81). He therefore holds that medical knowledge is entirely discovered, and independent of any mental or social constructs, and not at all historically contingent. Thagard is not alone in holding this view. In chapter 3, I discussed the views on naturalist philosophers of medicine, such as Christopher Boorse (1977), who hold that the functions of patients’ organs can be objectively discovered, and that ‘disease’ is equivalent to a reduction in the functional ability of that organ. I also drew attention to several normativist philosophers of medicine who, even though they do not accept that disease status or biological function can be objectively determined, do hold that at least some biological facts relevant to disease status can be discovered. The view that at least some aspect of medical knowledge is not historically contingent is well represented in contemporary philosophy of medicine.

I have argued against this view, particularly in the case of knowledge about heart failure. In the longue durée history I present in chapter 2, I track the development of knowledge of heart failure over two centuries. At no point do I find that
researchers had no choice but to accept certain conclusions about what heart failure was and which patients were suffering with it. This history is presented in eight stages, with each stage representing a different way in which heart failure was understood. Differences in understanding might be due to different patients being taken as having the disease, the disease being diagnosed in a different way, or what was taken to be its pathophysiology being changed. In all but one case (the transition between stage 4 and stage 5) each of the transitions between these stages were precipitated by unexpected observations – by conflict with experience. Even so, I have shown that researchers were never forced to make the changes to how heart failure was understood that they did. There were always plausible alternative ways to resolve these conflicts with experience. Knowledge of heart failure, as we currently understand it, is not the inevitable result of the way that the world is. Rather, this knowledge appears to be historically contingent.

My argument that knowledge of heart failure is historically contingent is perhaps prescient of knowledge of other diseases. As discussed in chapter 1, it is widely accepted amongst philosophers that the function of the heart is to pump blood around the body. Indeed, the heart is the most widely used example of an organ with a supposedly obvious function in the philosophical literature on biological function (Wouters 2005). By attending carefully to medical practice, I have shown that this is not the case. Doctors in the present day do not claim that the function of the heart is simply to pump blood around the body, or even to pump blood around the body at a rate that meets metabolic demands. Rather, the function of the heart is commonly taken by doctors to be to pump an adequate volume of blood around the body at a normal filling pressure. The discrepancy between what doctors and philosophers think the function of the heart is shows, at the very
least, that the function of the heart is not obvious. In addition to arguing that the function of the heart is not obvious, I have argued that it is historically contingent. Finding that medical knowledge which is widely taken to be obvious is actually historically contingent perhaps – *a fortiori* – calls into question the epistemic status of knowledge about other organ systems and other areas of medicine. If facts about how the heart contributes to the survival of patients, and about which patients are suffering with heart trouble, are historically contingent, then the epistemic status of present day knowledge of kidney, liver and lung disease are also perhaps called into question.

Future research could be aimed at exploring the historical contingency of other areas of medical knowledge, and of other diseases. It may be profitable to re-examine the case studies that have led other scholars to conclude that medical knowledge is not historically contingent (such as Thagard’s 1999 study of stomach ulcers). In the case of heart failure, philosophers may want to disagree with me and argue that present day knowledge of this disease is discovered, and the inevitable result of the way the world is. It is incumbent upon such philosophers to identify the place in the *longue durée* historical development of present day ways of understanding heart disease that this ultimate truth was discovered, and explain why they believe this to be the case. I suspect no such arguments will be forthcoming, and suggest that recognising the historical contingency of medical knowledge of heart failure will better suit the study of medical epistemology in many instances.

The historiography of medicine is also informed by this thesis. As discussed in chapter 3, some historians adopt what Adrian Wilson (2000) has called a ‘naturalist-realist’ historiographical stance, and hold that there are timeless facts about disease status, which are unaffected by changing cultures. This stance is
often adopted when historians write *longue durée* histories of disease. As discussed in the introduction, Mark Harrison (2015) claims that it is important to make use of timeless facts about disease in order to write historical accounts of disease that are not restricted to a particular time and place. The problem is that if there is nothing about a disease that is timeless and ahistorical, then it appears as though there is nothing to unite different ways of understanding sick people together apart from the present centred choices of historians. Historians wishing to write extended accounts of disease may thus feel that it is necessary to take some aspect of disease (usually some aspect of the disease’s biology) to be timeless in order to write an extended account of it. If historians want to track the spread of a particular sort of pathogen around the world, as Harrison does, then this may be the case. Nevertheless, my thesis shows that it is possible to write *longue durée* history of disease without taking any aspect of the disease to be timeless and ahistorical. Continuity between different ways of understanding sick patients can be established by the historical actors themselves, without needing to appeal to timeless biological facts. Historians may wish to adopt naturalist-realist stance with respect to some aspect of disease, but should not feel that this must be done in order to write an extended account of a particular disease. It is possible to write *longue durée* history of disease whilst acknowledging the radical historical contingency of medical knowledge.

The historical contingency of medical knowledge may come as a surprise to many philosophers of medicine in the present day. It will come as no surprise at all, however, to those philosophers and historians of medicine who have argued for some time that medical knowledge is historically contingent (Engelhardt 1985a; Wilson (2000); Cunningham (2002); Cooter (2013); Stein (2014)). However, these philosophers and historians do not show how medical knowledge can be
discovered as well as invented. It is not acceptable to reduce medical knowledge to pure invention, as accepting this would undercut the project of trying to find more effective ways of relieving the suffering of sick people. Accepting the view that patients “die from what their doctor’s (or bystanders) say they die of. And that’s that” (Cunningham 2002) would mean medical knowledge is whatever people agree that it is, and that diagnoses can never be mistaken.

I have used Ludwik Fleck’s work to produce an epistemology that can cope with medical knowledge being historically contingent whilst at the same time not collapsing into an extreme form of relativism. I have followed other philosophers (particularly Jerzy Giedymin) in drawing attention to the connections between Fleck’s epistemology, Kant’s ‘Copernican revolution in thought’ and conventionalism. I have argued that Fleck’s epistemology can usefully be read as a form of conventionalism, by seeing Fleck’s active element of knowledge as corresponding to Poincaré’s conventions, and seeing Fleck’s passive element of knowledge as corresponding to Poincaré’s laws. Fleck’s conventionalism is more useful than Poincaré’s for my purposes; not least because Fleck incorporated skills, experience, training and practices into his account of the active element of knowledge, whereas Poincaré only incorporated statements into his account of conventions. Crucially, Fleck also managed to articulate his conventionalism without needing to appeal to any account of crude facts. This allows all facts to be fully historically contingent and provides another advantage over Poincaré, whose epistemology requires an account of crude facts that he could not provide.

I have argued that by paying attention to the relationship between Fleck’s active and passive elements of knowledge, it is possible to see how medical knowledge can at once be invented and discovered. I have explained how active associations are produced and sustained by historical, social and cultural factors.
The active element of knowledge is necessary to produce passive facts about disease, and the facts produced will change depending on which active elements are adopted. Thus, all facts are historically contingent, and at least partly invented according to the (individual or collective) will of researchers. And yet, the active element of knowledge is not sufficient for the production of the passive element of knowledge. Hope’s adoption of the association between heart disease and lesions of the heart found at post mortem examination did not determine that the stethoscope should be a powerful diagnostic tool. Mackenzie’s adoption of the association between heart disease and a poor prognosis, and between heart disease and the syndrome of breathlessness and fluid accumulation, did not determine that the stethoscope was not a powerful diagnostic instrument. Nor did it determine that patients with valvular lesions did not necessarily have heart disease, or that there must be occult heart disease. Empirical experience was also necessary to produce these facts. Even though these active associations made the experience of these facts possible and influenced what these facts were, the active element of knowledge did not determine what these experiences would be. The active element of knowledge is a constraint that researchers (individually or collectively, consciously or subconsciously) impose upon themselves. Having constrained themselves, researchers then experience the passive element of knowledge as an additional loss of freedom. This passive element of knowledge resists the will of historical actors, and cannot be explained by historical, social or psychological factors alone. The passive element of knowledge is at least partly discovered.

Fleck, however, is not entirely consistent in his account of the passive element of knowledge. He sometimes does present the passive element of knowledge as being fully determined by the active associations adopted. This is illustrated by
his claim that any web of fantasy, if “spun for long enough”, will give rise to passive associations (Fleck 1979: 101; see chapter 7, section 4). The useful aspects of Fleck’s conventionalism are buried in his work, and tangled up with what are for my purposes less useful positions. Poincare, however, was always very clear (particularly in his polemic about Édouard LeRoy), that conventions or principles (corresponding to active associations) are not sufficient to determine the laws (corresponding to passive associations) that are produced. Following the adoption of conventions, laws are imposed upon researchers. Poincaré goes so far as to argue that this imposition is a form of objectivity. Even though objects may be conventionally defined, once this is done the relationships between these objects will no longer be for the researchers to choose. By reading Fleck as a conventionalist, it is possible to distil from his work an account of the passive element of knowledge that corresponds to Poincaré’s laws. This is an account where the passive element of knowledge resists researchers’ will, and therefore (following Poincaré) can be looked at as a form of objective knowledge.

Given the different active elements of knowledge that they adopted, both Hope and Mackenzie managed to experience something that resisted their will. Once they adopted the active associations they did, it was no longer up to them to decide whether or not the stethoscope was a powerful diagnostic instrument. In this conventionalist light, the facts produced by Hope and Mackenzie can properly be called objective.

In light of these reflections on how medical knowledge is at once invented and discovered, it becomes possible to make progress in the face of radical historical contingency. I have argued that all aspects of medical knowledge of heart failure are at least partly invented. Consequently, I cannot argue that progress is made as knowledge comes closer to capturing the way the world is in itself. However,
progress can be made in a different sense. Instead of thinking about discovery in terms of capturing the way the world is in itself, discovery can be thought of as finding a way of being resisted, by recognising the additional constraint of the passive element of knowledge. When Mackenzie pointed out that patients with heart murmurs very often did not become ill in the expected way, this finding was an important medical discovery. No matter how much other researchers might have wanted to believe that just about every patient with a heart murmur would become ill with breathlessness and/or fluid accumulation unless their circulation was kept tranquil, Mackenzie discovered resistance to this belief in his experience of this sort of patient. Having constrained himself in the active element of knowledge, which permitted the identification of patients with valvular lesions and patients who became ill as a result of a heart condition, Mackenzie was able to experience the additional constraint that patients with valvular lesions very often did not become ill in the expected way. This resistance was useful to medical practice, as it diminished the needless restriction of the activity of patients. This discovery, and this change in medical practice, should count as progress, even though it is not progress towards an ultimate truth about heart disease.

This progress is should not be thought of as progress away from erroneous ways of understanding heart disease either. Hope’s suggestion that valvular lesions caused breathlessness and fluid accumulation, and that consequently just about all patients with valvular lesions would eventually become ill in the expected way if their circulation was not kept tranquil, did turn out to be wrong. Even so, this does not mean that Hope’s way of understanding sick patients was not valuable, and did not lead to important medical discoveries. Many patients today are fortunate that Hope’s way of understanding sick patients has persisted despite Mackenzie’s aggressive rejection of it. Surgeons today do operate to repair the
heart valves of many patients with valvular lesions. Many symptomatic patients do experience a dramatic improvement of their symptoms following surgery, and the average life span of cohorts of selected asymptomatic patients is also known to improve following surgery (Goldstone et al 2015). Consequently, valvular lesions are still referred to as a cause of heart failure. The finding that valvular lesions are associated with breathlessness, fluid accumulation and reduced survival in many patients was an important discovery made by Hope and other early eighteenth century doctors. To say that Mackenzie was correct to point out deficiencies in Hope’s way of understanding sick patients does not mean that Hope’s way was inferior to Mackenzie’s, because Hope’s understanding produced useful passive associations which Mackenzie’s understanding did not.

Making the active association between valvular lesions and disease, enabled Hope to make the passive association between valvular disease and breathlessness and fluid accumulation, and to identify valvular lesions as a cause of these symptoms. This enabled doctors in the twentieth century to develop surgical techniques to treat this disease, to relieve suffering and prolong life. As surgical techniques have improved, the view that patients with valvular disease should undergo early valvular repair before they become symptomatic has become more prevalent. This approach entails that patients who would never have developed heart failure would receive surgical treatment of their valvular disease. This is deemed acceptable because the operative risks are so low that outcomes for the cohort of patients managed like this are improved. This is an important finding, which is a valuable factor to take into account in patient management. It would not be possible to make this finding unless patients with valvular diseases were taken to be the same sort of patient, even if they are asymptomatic. Mackenzie’s way of understanding heart disease, which required
that patients be symptomatic in order to be diseased, would not generate this passive resistance. The point is that both ways of understanding sick people produce resistance which is useful, and which the other way does not. Whether a way of understanding sick patients is progressive is a function of whether it generates useful ways of being resisted. I expect this can happen in a many different ways.

Following my conventionalist reading of Fleck, the acquisition of objective knowledge need no longer be understood as acquisition of knowledge of the ‘world in itself’. Instead, this can be understood as having experiences that resist our will, that are not up to us, and recognising that this is the case. This shift in how objectivity is understood has important consequences for how the diagnostic practices are assessed, and for how heart failure is diagnosed in practice. Researchers are free to give up a search for universal and timeless truths about which diagnostic tests are most accurate of all, without having to worry that this means that they have to give up the search for objective knowledge of disease altogether. Instead of worrying about universal truth, researchers can concentrate their efforts on finding interesting and useful ways of being resisted.

My argument that all aspects of medical knowledge are historically contingent relies on showing that researchers could always respond to conflicts with experience in multiple ways. This does not mean, however, that researchers resolved conflicts with experience by entirely rejecting everything that had previously been accepted as true, and starting again from scratch. Recall from chapter 2 that each transition from one stage to another did not simply involve the rejection of knowledge from the stage before it. Each transition also involved the preservation and use of knowledge from the stage before it. Whilst Mackenzie rejected so much of Hope’s way of understanding heart disease, he accepted
that heart disease was associated with the syndrome of breathlessness and fluid accumulation. Mackenzie may have explained the production of breathlessness (and pain) in a different way to Hope, but he still inherited these associations from Hope’s time. The relevant difference here between Hope’s and Mackenzie’s ways of understanding heart disease is that for Hope the association between heart disease and the syndrome of breathlessness and fluid accumulation was an empirical finding and passive, whereas for Mackenzie it was an active association that allowed empirical findings to be produced.\footnote{Fleck discussed how passive associations can be elevated in status to active ones over time through sheer weight of tradition: “The greater the distance in time or space from the esoteric circle, the longer a thought has been conveyed within the same thought collective, the more certain it appears. If the bonds consist in mental training during childhood years or, better still, in a tradition several generations old, they will be indissoluble” (Fleck 1979: 106-107).}

The active associations adopted were often so strong that many of these historical actors did not feel that they had a choice about how to respond to the unexpected observations that they made. Mackenzie felt that he was “forced” to conclude that there must be occult heart disease, because patients with the syndrome of breathlessness and fluid accumulation did not have obvious lesions to their hearts (Mackenzie 1913: 7-8; see chapter 7, section 7). Harrison argued that because patients with this syndrome did not all have a reduced cardiac output, it was logical to conclude that the “forward failure’ theory is not acceptable” (Harrison 1935: 69; see chapter 2, section 3). The active associations made by researchers were so strong that they could not entertain alternative ways of resolving the conflicts they experienced. The reaction of these historical actors to new observations was underdetermined, but it was also profoundly influenced by the active associations they adopted. In order to explain how these
researchers reacted to new observations, it is necessary to explain why they
adopted the active associations they did.

In order to explain why Mackenzie adopted the active associations he did it is
necessary to look back to Hope’s time. Without *longue durée* history, it is not
even possible to understand why Mackenzie faced the conflict with experience
that he did, let alone why he resolved it in the way that he did. Mackenzie was
surprised to find that many patients with murmurs never became ill. Without
expectation there can be no surprises. So, to explain this surprise, it is necessary
to explain the expectation. This expectation originated in the early nineteenth
century, and cannot be explained without looking at least this far back in time. In
order to understand why Mackenzie adopted the active associations he did, and
why he diagnosed heart failure in the way that he did, it is important to look at
medical research and practice well before Mackenzie’s time. As Mackenzie once
said “If we would really seek to find the true reasons for many accepted beliefs,
we must first consider those who propounded them” (Mackenzie 1911a: 794).

The value of *longue durée* history becomes even more apparent if the rest of the
history presented in chapter 2 is discussed in Fleckian terms of active and
passive associations. Consider Harrison’s work (chapter 2, stage 3). Can this be
understood without understanding Hope’s and Mackenzie’s work? I would argue
that it cannot. Harrison explicitly set out to test Mackenzie’s view that heart failure
was caused by a reduced output from the heart. Harrison argued that he had
showed that this view must be incorrect, because many patients with heart failure
had higher than average cardiac output (taking metabolic rate into account). As
discussed, this conclusion betrays the view that heart failure could be identified
from the presence of the syndrome of breathlessness and fluid accumulation. In
order to explain why it was that Harrison was so sure that patients with heart
failure could be identified using this syndrome, it is necessary to acknowledge that Harrison inherited this active association from Mackenzie, who in turn had inherited this association (in passive form) from Hope’s time.

It is also impossible to explain how Harrison resolved his conflict with experience without appealing to history. As discussed in the previous chapter, Harrison could have simply claimed that many patients with the syndrome of breathlessness and fluid accumulation did not have heart failure. This solution does not appear to even have occurred to him, as for him the active association between this syndrome and heart failure was so strong. As far as Harrison was concerned, he had falsified Mackenzie’s forward failure theory. If we want to understand why Harrison’s conclusions were reasonable to him, it is necessary to understand why he took these things for granted, and this requires a long historical view. Furthermore, Harrison actually resolved this conflict by explicitly appealing both to Starling’s work on “the law of the heart” from the early 1900s (Patterson et al 1914), and to Hope’s back-pressure theory from the early 1800s (Hope 1833). Thus it is not possible to explain either the conflict with experience that Harrison encountered, or how he resolved it, without looking at the development of knowledge of heart disease over the previous century.

The same is true of Merrill’s work (chapter 2, stage 4). Merrill was surprised to find, amongst other things, that patients in heart failure typically had less concentrated blood, not more concentrated blood as was expected from Harrison’s backwards failure theory. In order make this surprising observation Merrill needed to be able to identify those patients with heart failure, which he did using the syndrome of breathlessness and fluid accumulation as Harrison and Mackenzie had before him. Merrill also inherited his conception of heart failure from earlier generations of researchers. This legacy was key in structuring
Merrill’s response to his conflict with experience. So strong was the connection between heart failure and the syndrome of breathlessness and fluid accumulation for Merrill that he did not consider that the patients who had dilute blood did not have a heart disease. Rather, he argued that the kidneys must be involved in the pathophysiology of heart failure. Merrill suggested that the pathophysiology of heart failure is the low output from the left side of the heart leads to decreased blood supply to the kidneys, which leads to systemic fluid accumulation; and pressure build-up from behind the left side of the heart leads to the pressing out of fluid into the lung tissue leading to breathlessness. Even though Merrill, by adopting this new theory, rejected Harrison’s explanation for systemic fluid accumulation, he did not reject everything that Harrison had suggested. This new theory was a perfectly reasonable modification of Harrison’s suggestions, made in the light of Harrison’s understanding of how breathlessness was produced, in the light of Merrill’s understanding of renal physiology, and in the light of traditionally accepted knowledge of the connection between heart failure and the syndrome of breathlessness and fluid accumulation. Again, in order to understand why Merrill’s actions were reasonable for him, it is necessary to understand why he made the active association between heart failure and this syndrome, and to do this it is necessary to take a long historical view.

Braunwald (chapter 2, stage 5) argued that the best way to investigate heart failure was to view the disease as a reduction of contractility of the heart muscle, and not as a syndrome of breathlessness and fluid accumulation. Even patients with this syndrome as a result of valvular and pericardial lesions were no longer seen as patients in heart failure, unless they also had a reduction in the contractility of their heart muscle. Braunwald transformed the association between this syndrome and heart failure from an active element of knowledge to
a passive one. Even so, Braunwald’s work was still heavily influenced by the views of researchers that came before him. Braunwald inherited his notion of contractility from Harrison, who himself drew on Mackenzie’s and Starling’s work on this notion. Harrison’s notion of contractility was not simply the amount of force developed by the heart muscle, but the force for a given preload\(^\text{121}\). A heart with reduced contractility would develop less force for the same amount of preload, and thus would eject a smaller percentage of the blood in the left ventricle at the end of filling – its ejection fraction would be less. Harrison and Braunwald use the notions of contractility and ejection fraction differently. For Harrison, contractility was part of a proposed explanation for the symptoms of some patients with heart failure. Harrison recognised heart failure from its symptoms, and used these symptoms to connect this notion of contractility to heart failure. For Harrison, it was the association between the syndrome of breathlessness and fluid retention that was an active element of knowledge. For Braunwald, by contrast, it was the reduction of contractility that was actively associated with heart failure, and key to the identification of heart failure. The connection between reduced contractility and heart failure was there in both cases, but the role it played in each case was different. Nevertheless, it was Harrison’s understanding of contractility that made Braunwald’s understanding of contractility possible. It is impossible to understand why Braunwald made the active associations he did without understanding Harrison’s views, and it is impossible to understand Harrison without looking at Mackenzie, and so on.

The elevation of the status of the connection between contractility and heart failure may seem like an insignificant change. At this time (around 1960), both

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\(^{121}\) Recall from chapter 2 that preload is the amount by which the heart muscle is stretched at the end of diastole just before it contracts.
Harrison and Braunwald thought that the patients with the syndrome of breathlessness and fluid accumulation and the patients with reduced contractility were (with a few exceptions) the same. If the presence of this syndrome and reduced contractility are closely correlated, then it does not really matter which is used to define heart failure. The patients diagnosed in either case would have been the same. Even though this difference would not have affected medical practice greatly at the time, it certainly did have an effect on how medical practice developed from that point.

When the researchers in stage 6 (see chapter 2, stage 6) found that reduced contractility and the syndrome of breathlessness and fluid retention were not correlated, they concluded that the presence of this syndrome could not be used to detect heart failure accurately. This confirms that reduced contractility was actively associated with heart failure, and also shows the dramatic effect that this changing an element of knowledge from passive to active (or vice versa) can have on the future development of medical knowledge. If Braunwald had not elevated the association between heart failure and contractility to an active association, and demoted the association between heart failure and the syndrome of breathlessness and fluid accumulation to a passive association, then the reaction of the researchers in stage 6 may well have been very different.

If the association between heart failure and the syndrome of breathlessness and fluid accumulation was an active element of knowledge, then the finding that the patients with this syndrome did not necessarily have reduced contractility of their heart muscle may well have been taken to demonstrate that reduced contractility was not the pathophysiology of heart failure.

This is not a wild piece of counterfactual speculation. When Harrison was confronted with the finding that patients with this syndrome did not necessarily
have a reduced cardiac output, did not reject the view that patients with this syndrome had heart failure. Rather, he rejected the view that reduced cardiac output was the pathophysiology of heart failure. When confronted with a mismatch between the patients with this syndrome and the patients with the expected pathophysiological explanation, Harrison rejected the pathophysiological explanation. The researchers in stage 6, by contrast, when confronted with a similar mismatch between patients with the syndrome and patients with the expected pathophysiological explanation, rejected the view that many patients with the syndrome had heart failure. Changes to the organization of elements of knowledge, from passive to active or vice versa, may not have a dramatic effect on practice at the time the change is made. Even so, these changes can have a profound influence to how researchers react to conflicts with experience, and thus on how medical practice develops.

The same observation carries through to the present day. Recall from chapter 2 (stage 7; and the introduction) that studies like that carried out by Soufer and colleagues (1985) played an important role in identifying heart failure with a preserved ejection fraction. Such studies argued that heart failure with a preserved ejection fraction existed because many patients with the syndrome of breathlessness and fluid accumulation did not have a reduced left ventricular ejection fraction. In these studies, the association between heart failure and this syndrome was an active association, the adoption of which allowed the fact of the existence of heart failure with a preserved ejection fraction to be produced. The authors of these studies, like Soufer and colleagues (1985), did not develop this active association themselves out of nothing. They inherited this active association from earlier generations of researchers, and ultimately from Mackenzie. In order to understand the reaction of present day researchers’ to the
observation that many patients with the syndrome of breathlessness and fluid accumulation do not have a reduced left ventricular ejection fraction, the reasons they actively associate this syndrome with heart failure must be understood. To do this, it is necessary to understand why Mackenzie made this active association, and to do that, it is necessary to look back to the early nineteenth century. As Fleck says, “[c]oncepts are not spontaneously created but are determined by their “ancestors”. That which has occurred in the past is a greater cause of insecurity – rather, it only becomes a cause of insecurity – when our ties with it remain unconscious and unknown” (Fleck 1979: 20). Longue durée history makes researchers aware of the reasons that certain active associations are adopted in the present day, and thus allows these active associations to be evaluated.

To exemplify the effect that history can have on how researchers react to new findings, consider the claim discussed in chapter 1 (section 3), that it is renal insufficiency that makes the difference between symptomatic and asymptomatic patients with diastolic dysfunction (Victor and Barron 2010). This study found that patients with diastolic heart failure tended to have renal insufficiency, whereas patients with asymptomatic diastolic dysfunction did not. Using these results, these researchers come close to arguing that patients in “heart failure” actually have a kidney problem: “The results of this study support the hypothesis that patients with normal left ventricular ejection fractions but diastolic dysfunction develop congestive heart failure because of underlying renal insufficiency” (Victor and Barron 2010: 770). They stop short of this radical claim, preferring instead to think in terms of kidney problems causing heart failure, or vice versa: “A prospective and larger study is needed to determine if renal insufficiency is a causative mechanism of heart failure or a secondary effect of heart failure” (Victor
and Barron 2010: 773). If this “heart failure” might be caused by kidney problems, and might be treated by improving renal function (as these authors suggest (2010: 773; see also Damman et al 2007), then why are these heart failure patients’ with a preserved ejection fraction not seen as patients with kidney problems? Why is disease not diagnosed by detecting renal insufficiency in patients who are accumulating fluid?

The reasons for this are in part historical. As discussed in chapter 4 (section 6), in the 1860s Walshe suggested that fluid accumulation (or “dropsy”) in patients with heart lesions might have an extra-cardiac cause (1862: 240). The association between Bright’s disease (which affected the kidney), fluid accumulation was widely recognised at the end of the nineteenth century (Peitzman 2007). Mackenzie himself recognised the association between Bright’s disease and hypertrophy of the heart (Mackenzie 1913: 317). Despite this, by end of the nineteenth century the active association between the syndrome of breathlessness and fluid accumulation and heart disease had developed. Consequently, patients with this syndrome were not seen as patients with kidney problems, even when there was no obvious pathology of the heart. In order to assess the possibility that patients with the syndrome of heart failure actually have kidney disease, one must understand the reasons for adopting the active association between this syndrome and heart problems. This requires looking back through medical history to find the reasons that this association was made, and the reasons why it was sustained.

One might even question why researchers are searching for ways of diagnosing diseases of particular organs at all. If the development of symptoms depends on aspects of the functioning of both organ systems, then why are heart failure and kidney failure the object of research, rather than failure of some system that
integrates aspects of both organ systems? Again, the reasons for this are partly historical. Researchers in the early nineteenth century investigated lesions of organs, and it followed from this that patients would be classified according to disease of particular organs. Mackenzie rejected the association between disease and organic lesions, but he still accepted the association between disease and particular organs, as he understood disease in terms of the malfunction of particular organs. Mackenzie inherited this association from nineteenth century researchers, and has passed it down to present day researchers. In order to assess the possibility that patients might not have heart or kidney trouble, and instead might have a disease that is distributed over several organs, it is helpful to understand how diseases and particular organ systems came to be associated. This requires looking back through medical history, at least to the nineteenth century. My thesis supports the views of several historians (Jones et al 2015), that medical history can be used to inform medical decision making in the present day.

I believe the general effect that medical history can have on medical decision making can be understood with the help of Kant’s analogy of the judge and the witness (discussed in chapter 6, section 2). The judge (representing researchers) interrogates the witness, supplying the active element of knowledge. The witness’s answers are framed by how the judge asked the questions, and together the judge and the witness produce testimony, which represents the passive element of knowledge. As the longue durée history reveals, the testimony so produced can surprise the judge, who may change how future questions are framed in the light of this testimony. Historical researchers have reacted to surprising results by changing the active associations they adopt. The interaction between the judge and the witness is not a collection of isolated events, but rather
an iterative process, rather like a conversation. Should the judge retire, and need to be replaced by a second judge who takes on the responsibility of interrogating the witness, it is useful for this second judge to be informed about how the conversation has proceeded thus far. Without some knowledge of the history of the conversation to date, the second judge may not fully understand the questions he or she is required to ask, nor the answers that are given, because he or she will not understand why the conversation has been framed in the way that it has. A researcher ignorant of medical history is at the same disadvantage as someone walking in half way through a conversation.
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