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A Matter of Degree: The Normalisation of Hypertension, circa 1940 – 2000

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High blood pressure is a peculiar disorder. In most cases it is without symptoms, and patients are often diagnosed with hypertension, curiously, when they have no idea that they are ill. A quick series of easy measurements with the sphygmomanometer, a piece of laboratory technology that has become part of routine medical practice, delivers the diagnosis. Even though mild or moderate hypertension itself hardly causes any symptoms, most doctors and medical administrators agree that the disorder is 'one of the most important preventable causes of premature death worldwide' and treatment is advisable.[1] High blood pressure is framed today generally not as a disease that causes direct suffering, but as a 'risk factor' in stroke and heart disease, a quantifiable marker of potential disease.[2] However, the boundary that separates normal and pathological blood pressure remains disputed. A recent review article on the pathophysiology of hypertension avoids questions of classification and does not mention any such boundaries.[3] The general consensus, based on a number of long-term epidemiological studies, is that in terms of risk the lowest is the best possible blood pressure. Medical authors tend to be careful not to draw a clear line between physiological and pathological blood pressures, but government-appointed committees are willing to recommend thresholds for treatment, and these thresholds get

increasingly lower. In recent US guidelines on hypertension ‘high normal blood pressure’ (above 120/80 mm Hg) has been re-classified as ‘pre-hypertension’ and treatment is recommended.[4]

Well into the 1950s hypertension was quite a different matter from what we take it to be today. Hypertension was conceived of as a disease, and a pressure reading of, say, 140/80 mm Hg alone (classified as stage 1 hypertension in the new US guidelines) would definitely not have raised a doctor’s eyebrows. With only very few, drastic treatment options available, whether a patient was to receive treatment or not was a matter of judgement for the physician rather than the expected (and officially sanctioned) response to a series of sphygmomanometer readings. In general, the hypertensives who received treatment had malignant hypertension, severely increased blood pressure with manifest pathological effects, a disease which not only posed a long-term risk but led to clearly distinguishable, acute symptoms and possibly the death of the patient. According to one of the pioneers of hypertension research in Britain, Sir Colin Dollery, malignant hypertension has all but disappeared from the industrialised world since effective drug treatments became available in the 1950s.[5] Simultaneously hypertension was re-defined as a quantitative disease, the upper end of a bell-shaped normal distribution. In the absence of symptoms, hypertension has come to be framed by epidemiological data, notions of risk, and a succession of new drug treatments.

The transition from an acute, life-threatening disease into a matter of degree and the difficulty of defining boundaries makes hypertension an ideal test case for Georges Canguilhem’s classic essay on *The Normal and the*

Pathological. Hypertension is a disorder that is defined by modern medical science. In this chapter, after introducing Canguilhem's main arguments I will turn to the role that clinical science has played in the transition of high blood pressure. I will conclude by discussing the wider context of this transition during a time that has often been characterised as the 'golden age' of modern biomedicine, and some of its implications.

The normal and the pathological

Georges Canguilhem's book on *The Normal and the Pathological*, first conceived in 1943 and revised in the mid-1960s, has received a lot of attention recently, not least from cultural theorists, after being reissued in 1991 in the trendy 'Zone' cultural studies series, with an introduction by Michel Foucault.[6] The philosopher and physician Canguilhem owes much of this revived interest in the English-speaking world to the fact that Foucault named him as one of his major influences.[7] It should not be forgotten, though, that *The Normal and the Pathological* was initially above all a book about physiology and its role in the epistemology of medicine. Canguilhem's book is an expression of long-standing concerns, not only in France, about the meanings of scientificity in medicine. He challenges the notion (which he traces to Claude Bernard) that medicine can only be scientific if it is reduced to the application of physiology with its positivist assumption that life is governed by laws identical in kind to those that govern the world of inanimate objects. He points to the problems associated with Bernard's attempts to

identify the normal with a – quantifiable – ideal of organic function that can be assessed by way of rigorous experimentation. Physiology is where the laboratory and the clinic meet, and this leads to tensions. If the body is merely a complicated mechanism, whose functions and dysfunctions can be evaluated by way of laboratory technologies (such as the sphygmomanometer), there can be no qualitative difference between normal and pathological states. If, on the other hand, the normal is more or less identical with the 'healthy' and therefore attached with qualitative values, there can be no continuity between normal and pathological states.

Canguilhem not only questions the uncritical use of physiological concepts in practical medicine, he also rejects the other common approach to normality, that of statistics. Statistically obtained averages cannot provide a doctor with clear guidelines for judging the health of individuals. While the distinction between normal and pathological is clear for every individual, boundaries between normal and pathological on the level of populations are fuzzy. Furthermore, the real opposite of the normal is the abnormal and not the pathological, and an anomaly does not automatically lead to illness but may merely be a (potentially useful) variation.

Health, according to Canguilhem, depends on the ability of individuals to respond to different environments by adaptation, by adopting new norms, which in certain circumstances can lead to physiological parameters very different from those measured under the ideal laboratory conditions that Bernard aimed for (and these parameters may therefore well be abnormal). Disease, in turn, is caused by the inability to adapt. Malignant hypertension fulfils these requirements: it severely restricts patients' abilities to adapt to

new situations, and they know that they are not well. Malignant hypertension in the 1940s and 50s, according to Colin Dollery, was a 'death dealing disease', and most of the patients he encountered before effective drugs became available felt seriously ill.[8] They often had difficulties with breathing at night, were woken in the morning by a headache and troubled during the day by blurred vision.[9] Canguilhem's book emphasises the role of individual experience as the root of all medical science. Perceived illness is the basis of the science of pathology and of all meaningful knowledge on physiological processes, but physiology in turn does not provide us with reliable information on what is pathological. An increased blood pressure may be merely the attempt of the body to adapt to a special situation.

Canguilhem locates the origins of the notions he analyses (and criticises), of the pathological as merely a quantitative variation of health, in the nineteenth century. While well established in physiology, however, these concepts were implemented in medical practice only in the twentieth century. In the following sections of this chapter I will look at the role that the establishment of an infrastructure for clinical research played in this process (in Britain roughly between the end of World War I and the 1950s), along with the development of new, 'physiological' means of medical intervention - biological and chemical therapeutic agents that were highly visible symbols of medical progress. I will offer possible explanations for the success of the concept of a quantitative disease in a medical landscape shaped by the new clinical sciences as well as new administrative concerns, by contextualising a well-publicised debate over the re-framing of high blood pressure in Britain in the mid-twentieth century. The dispute between two influential British

clinicians, George White Pickering and Robert Platt, over the nature of essential hypertension, provides me with a lens to study this transition.[10]

Platt, Pickering and clinical science in Britain

Both Platt and Pickering were prominent clinical scientists, but as we will see they represented slightly different traditions. Compared to France and Germany, Britain was late to establish an infrastructure for clinical science. Christopher Lawrence has argued in two important articles that until well into the twentieth century, the British medical élite were rather sceptical about the new institutions of clinical science.[11] Established British clinicians were not opposed to medical innovations *per se*. However, wedded firmly to a medical market place where the part-time affiliation with a medical school provided them with competitive advantages, élite doctors felt uneasy about the growing state intervention in medicine. As Christopher Booth has shown, prior to World War I England had no clinical science tradition to speak of. There were no full-time professors in clinical subjects, for example, and the London medical schools had no clinical laboratories, hardly any links with the universities, and no paid staff.[12] This changed after the war, not least due to the activities of the new Medical Research Council (MRC).[13]

To establish an infrastructure for medical research to the Council meant both the training of young researchers and the provision of posts at MRC units in hospitals around the country. Initially, in the absence of an existing clinical research landscape, this was partly an export of Cambridge physiology into

clinical settings, and partly an attempt to copy German and US models.[14] The research units of Thomas Lewis and Thomas Renton Elliot at University College Hospital (UCH) were dedicated to the translation of themes such as cardiovascular regulation, pioneered by Cambridge physiologists, into clinical research.[15] The colleague of Platt and Pickering and professor at the Postgraduate Medical School at Hammersmith Hospital, John McMichael, highlighted this in 1952: 'Progress in understanding disease processes is determined by the availability and applicability of laboratory techniques. Clinical investigation closely follows physiology, while surgery and pharmacology provide its therapeutic "experiments".'[16]

George Pickering's work, like McMichael's, was initially informed by the research schools of Lewis and Elliot. Much of Pickering's training was geared towards a career in full-time medical research, and he never worked in private practice. Throughout his career he was based in the 'Golden Triangle' of Oxford, Cambridge, and London. He studied at Pembroke College, Cambridge, and pursued his clinical studies at St Thomas's Medical School in London, from where he graduated in 1930. After resident appointments at St Thomas's he entered Thomas Lewis's clinical research unit at University College London.[17] In 1939 Pickering was appointed professor of medicine at St Mary's Hospital, London, where he assembled a group of clinicians and scientists working on the study of blood pressure, employing a wide range of approaches, from biochemistry to epidemiology. In 1956, Oxford University appointed Pickering as Regius Professor of Medicine.

Robert Platt's career unfolded in the provinces and was initially not as focused on research as Pickering's. Platt studied medicine in Sheffield and

began his teaching career as a part-time lecturer at the University of Sheffield, also running a successful private practice.[18] After World War II, he accepted an offer to become the first full-time, salaried Professor of Medicine at the University of Manchester, appointed to the first chair of this kind outside the capital.[19] Under his leadership the Manchester department specialised in nephrology, the physiology and pathology of the kidney.[20] Platt's career and his move from part-time university appointment and a flourishing private practice in Sheffield to a full-time university chair in post-war Manchester is representative of the larger changes in British twentieth-century academic medicine, namely the move from part-time to full-time clinical research posts.

In the following section we will examine how their different backgrounds, with Platt more wedded to an older, individualist model of clinical practice than Pickering, found their reflections in different concepts of the nature of high blood pressure.

Hypertensive disease

How was high blood pressure framed when Platt and Pickering started their careers? In the 1930s and 40s high blood pressure – if no other obvious causes could be found – marked a distinct and specific disease, essential hypertension or hypertensive disease. According to this paradigm, which Platt continued to defend, there was also a distinct group of people, the hypertensives. While they may not know this when young, their blood pressure would inevitably go up later in life if not treated in time. In younger

years, these hypertensives would not even necessarily have high blood pressure.

Platt and his co-workers in their work on high blood pressure followed up patients who were treated at the Manchester Royal Infirmary for malignant hypertension. Significant for Platt's approach, and distinguishing it from Pickering's, as we will hear, was that he studied patients who were undergoing treatment in his hypertension clinic, and their relatives. In Canguilhem's terms, Platt's patients had already lost their innocence and lived their lives under the new, narrower conditions that the disease was imposing on them. The treatment, in many cases surgical sympathectomies with their serious side effects, kept patients alive but would never return them to the state that Canguilhem calls normative.[21] Platt was looking for family links and believed to have found the cause of the hypertensive disease of his patients in their genes.

The paper in which Platt summarised the results of his study on heredity and hypertension was published in the *Quarterly Journal of Medicine* in 1947.[22] Drawing on his work with the Manchester patients, Platt suggested that 'essential hypertension is the heterozygous (or occasionally homozygous) expression of a dominant Mendelian characteristic', or in other words, he proposed the existence of a specific hypertension gene.[23] This suggestion was in line with much of the contemporary literature. Platt's article does not explicitly state where he expected the genetic defect to be located, but it is likely that he was thinking about a gene associated with aspects of kidney function. Platt argued for the necessity of long-term follow-up studies into the natural history of hypertension.[24] He hypothesised that these studies were

going to reveal that with regard to blood pressure the population was divided into two distinct groups: a normal majority, and a distinct group of hypertensives, whose blood pressure was going to reach pathological levels in middle age.

In the 1940s, Pickering may have agreed with Platt. Based on a review he published in 1952, it is quite plausible to assume that Pickering expected the results of a study he and his co-workers were then undertaking at St Mary's Hospital to be in line with Platt's suggestions and the dominant hypotheses regarding the nature of essential hypertension (although he already discusses the difficulty of determining upper limits of normal blood pressure).[25] The debate between the two began two years later when Pickering and his co-authors, Michael Hamilton, John Alexander Fraser Roberts, and Clive Sowry, published the results of this study in a series of articles in the journal *Clinical Science*, entitled 'The Ætiology of Essential Hypertension'.[26] The conclusions of Pickering and his co-authors were different from Platt's in a number of significant points. Above all, they disputed his evidence for the existence of two distinct groups, one normal and one hypertensive. Hypertension to Pickering was now merely a quantitative phenomenon, the upper end of a normal distribution of blood pressures. The distinction between physiological and pathological was unclear, and where two groups seemed to show in the data, this was an artefact of measurement. 'Hypertension is,' Pickering wrote in 1974, '... as I pointed out in 1955, a new type of disease in which the deviation from the norm is one of degree and not of kind. It is a quantitative disease.'[27]

Pickering and his colleagues, in contrast to Platt, did not study a group of hypertensives but surveyed the blood pressures of outpatients at St Mary's Hospital who were treated for conditions, mostly surgical, that had nothing to do with hypertension. In regard to blood pressure, they were studying apparently healthy people. The study was the attempt to find the normal distribution of the markers of a potential disease in a healthy population. This approach was subsequently taken much further by large epidemiological studies such as the Framingham Heart Study and the smaller-scale follow-up studies undertaken by the MRC Epidemiological Research Unit in South Wales (for which Pickering acted as an advisor).[28] These studies were designed to not only establish distributions in normal populations, but also look at the emergence of pathological problems over time. They aimed at calculating the statistical links between physiological parameters in healthy people and subsequent illness, statistical entities which today we call risk factors.[29]

Pickering's quantitative concept of hypertension was the product of surveys, and the difference between Platt and Pickering could also be interpreted as an expression of the transition proposed by David Armstrong, from 'hospital medicine' to 'surveillance medicine'.[30] In Platt's eyes, hypertension was an essential reality – either you had it or you did not. According to Pickering, blood pressure was distributed along a continuum. Everybody was to be considered normal, but that did not mean that they were necessarily also healthy. While treatment may be appropriate, this had to be decided for every individual patient, taking into account other parameters. A

clear distinction between hypertensives and non-hypertensives was impossible.

Surveys, statistics and genetics

The main factor shaping Pickering's new outlook was his wholehearted embrace of surveys and statistical methods, brought about by his collaboration with John Alexander Fraser Roberts, an expert on statistics and human genetics.[31] Fraser Roberts, the son of a Welsh farmer, had started his career in agricultural genetics in the 1920s, studying inherited characteristics in Welsh mountain sheep. In the 1930s he turned to human biology, and in 1943 he obtained his MD. At the time of the collaboration with Pickering he held three different posts. He was Director of the Burdon Mental Research Department at Stoke Park Colony, Bristol; Director of Research at the Royal Eastern Counties Institution in Colchester (site of Lionel Penrose's work on Phenyl Ketone Urea); and Lecturer in Medical Genetics at the London School of Hygiene and Tropical Medicine (home institution of the pioneering biostatisticians and epidemiologists, Sir Austin Bradford Hill and Major Greenwood). Fraser Roberts had also established a genetic counselling clinic at the Great Ormond Street Children's Hospital. He was Pickering's link to reformed eugenics, new thinking in psychology, and an increasingly more sophisticated body of knowledge in medical statistics and population genetics.[32]

Platt nurtured what he himself called an ‘amateur interest’ in genetics, and his knowledge of statistics was limited.[33] Statistics was not strictly speaking Pickering’s specialty either, but he could draw on the expertise of Fraser Roberts. Increasingly central to the debate was the question if Pickering’s bell curve was really a bell curve, or a composite curve with humps that moved towards higher blood pressures in older populations and represented the carriers of one, or several hypertension genes.[34] Pickering compared the distribution of blood pressures to Francis Galton’s (the pioneer of eugenics) findings on height distribution in Britain, and the distribution of intelligence, which was one of Fraser Roberts’ main fields of expertise. The Platt camp, in contrast, compared hypertension with phenylketonuria, a disorder caused by a mutation in a single gene.

The debate between Platt and Pickering triggered a series of letters to the *Lancet,* and informed much British research on high blood pressure and the epidemiology of cardiovascular disease in the 1950s and 1960s. Well-known examples were the studies by Morrison and Morris on London bus drivers and conductors, and those already mentioned, by William Miall and his co-workers, on the inhabitants of mining villages in South Wales, both conducted within MRC research units.[35] Miall and his colleagues designed their study in collaboration with Pickering and Fraser Roberts, while Morris and Morrison supported Platt’s hypotheses.[36] A team of epidemiologists at the London School of Hygiene and Tropical Medicine, meanwhile, worked on ways of achieving non-biased blood pressure measurements.[37] The debate died down in the mid-1960s, when Platt moved away from his single gene hypothesis, while Pickering conceded that the bell curve may well

accommodate pathologies caused by single gene mutations. After all, nobody challenged the statistical distribution of intelligence or body height, despite the existence of disorders such as phenylketonuria which caused mental deficiencies and others that affected body height.

Physiological norms and administrative change

It is comparatively easy to explain what shaped Pickering's new approach to the nature of high blood pressure, but it is difficult to analyse the changing attitudes to what counted as medical mainstream within the changing social and moral economies of modern medicine. Steve Sturdy and Roger Cooter have attempted to do this for laboratory medicine, arguing that the increasingly central role of the laboratory in modern medicine since the late nineteenth century in Britain was closely associated with new administrative demands growing out of the rationalisation of health systems.[38] They suggest that

> the academicization of leading sectors of hospital medicine, and the introduction of laboratories and other scientific investigative techniques into clinical research, teaching and practice, did much to favour the growth of an administrative as opposed to an individualized way of knowing in medicine. This way of knowing was well suited to the demands of administering a corporate system of mass health care organized around a hierarchical division of medical labour. Shaped by the need to regulate and

> standardize diagnostic and therapeutic practice, it was closely linked to the pursuit of efficiency both in hospital medicine and in the health care system as a whole.[39]

Sturdy and Cooter do not discuss the central roles of statistics and genetics, which, as we have seen, complemented laboratory approaches in Pickering's new take on high blood pressure. It seems that the implementation of physiological concepts in clinical practice in Britain went along with another epistemic transition in medicine, associated with the rise of the welfare state.

Since the late nineteenth century, as David Armstrong has pointed out, the dispensary played an increasingly central role in medical epistemology. Similarly, the survey became a crucial tool in medicine, and Armstrong suggests that this paved the way for a new epistemic system, which he calls 'surveillance medicine'.[40] The objects of surveillance medicine are not individual bodies, as in hospital medicine, but populations. The normal came to be located not in the individual body (as for Bernard) but in the social body. In the course of the transition from hospital medicine to a preventive paradigm organised around the results of surveys, statisticians and geneticists felt that they had something to offer to clinicians. Population genetics combined the survey with the laboratory and, by way of genetic counselling, even with the individual clinical encounter. Fraser Roberts, in the 1940 edition of his textbook, saw the main significance of genetic analysis in its scientific forecasting ability that could potentially help to meet the emerging need for focused prophylaxis in medicine: 'A clear recognition of genetic susceptibility,' he argued, 'might be the best approach to the identification of controllable factors.' And, well within the paradigms of reformed eugenics, the 'knowledge

that a special hereditary susceptibility existed might sometimes lead to the institution of earlier treatment than would otherwise be the case'.[41] It may hardly be necessary to point out (as Cooter and Sturdy argued for the laboratory) that academic epidemiology, too, had its roots in administrative concerns. The MRC Units for Social Medicine and for Epidemiology had conceptual and institutional links with welfare administration and occupational medicine. The South Wales Unit was initially dedicated to pneumoconiosis, miner's disease. Jerry Morris, the founder of the Social Medicine Unit was drawn to social medicine through his friendship with Richard Titmuss, the statistician and pioneer of the post-war welfare state.[42]

The late 1940s and 1950s were a time when infectious disease seemed to be defeated and epidemiologists turned to chronic and degenerative diseases, the ailments of middle and old age such as cardiovascular disorders and cancer. The risk factor concept was born in the life insurance industry in the early twentieth century and found its way into mainstream medical science and practice in the 1950s.[43] Epidemiological studies pointed to the association of high blood pressure with cardiac heart disease and turned risk factors into a serious scientific concept.[44] Platt's single gene hypothesis matched the older clinical paradigm of hospital medicine, which looked at specific patients that could be identified and treated. Pickering's quantitative concept was informed by a focus on populations rather than individual patients, and ultimately allowed the treatment of a risk, a potential problem, rather than an identifiable, specific disease.

Therapy defines disease

The post-World War II period saw not only the rise of administrative concerns in medicine, but also an increasingly central role for the pharmaceutical industry. New therapies for chronic diseases confronted western health bureaucracies with new cost pressures. Today drugs for high blood pressure are big sellers. The cost of antihypertensive drugs in the United States, for example, amounts to currently about 15 billion US Dollars, accounting for 10 percent of the country's total spending on drugs.[45]

Drug treatments for hypertension were developed around the same time when the debate between Platt and Pickering enlivened the pages of the *Lancet* and informed much British research on high blood pressure. The new drugs led to the disappearance of malignant hypertension, but, as side effects became less drastic, they also led to continuing debates over the treatment of mild and moderate hypertension, imposing greatly increased costs on the health system.[46] In the absence of clear notions of where the physiological ended and the pathological started, therapy was no longer just reactive. Rather, the availability and expected success of a therapy began to determine the diagnosis.[47] 'While there is no natural dividing line between what is normal and what is abnormal', Pickering argued in 1974, 'something is known about the levels of arterial pressure above which treatment is beneficial'.[48]

In the 1940s, high blood pressure was treated surgically by sympathectomy in a minority of patients suffering from life-threatening malignant hypertension. Both Platt and Pickering treated patients in this way.[49] The side effects of the operation could be drastic and it went out of

fashion in the 1950s. Surgical sympathectomy was replaced by a class of drugs, the ganglion blockers, which were thought to block the nerve endings of the sympathetic nerve system in what resembled a chemical sympathectomy and whose side effects were almost as drastic as those of the surgical procedure.[50] Pickering studied the effects of these drugs in the early 1950s.[51] Other drugs followed, which all lowered blood pressures but in many cases caused what resembled a new disease in turn.

In 1948, a low-salt rice diet promoted by the emigré Walter Kempner at Duke University (inspired by the teachings of the German lifestyle reform movement), showed an unexpected antihypertensive effect and was tested by an MRC working group that included Platt.[52] It also led to the work on the thiazide diuretics in the laboratories of Sharp & Dohme, drugs that make patients urinate more and lead to a reduction of the amount of fluid and salt in the body. The thiazide diuretics, first marketed by Merck Sharpe & Dohme in 1959, had few side effects and were the first drugs that allowed the mass treatment of high blood pressure patients, even for milder forms of hypertension.[53] It was now justifiable to treat a mere risk factor, a blood pressure at the upper end of a normal distribution, without first having to identify a distinct and specific pathology.

There are parallels with psychiatric disorders, which also became normalised due to the growing influence of what Armstrong calls the 'community gaze' and the availability of new drug treatments in the post-war period. According to Armstrong:

> In essence, the post-war psychiatric perception was a normalizing gaze: not, as in the Panopticon, a normalizing gaze over an

> enclosed and inherently ‘abnormal’ population, but over an entire domain. This normalizing gaze over the whole tended to obliterate the legitimacy of the distinction between normal and abnormal and tended to create one community where before there had been two.[54]

Normalisation had consequences for members of both former ‘communities’. The boundaries between healthy and ill, between normal and abnormal (and also between somatic and psychological) became blurred.

Conclusion

Armstrong’s approach helps us to understand where the quantitative approach to high blood pressure and the risk factor approach had their origins and why they were so successful in the context of post-war medicine, but this does not automatically mean that Canguilhem’s concerns have lost their validity. Surveillance medicine may be part of medical reality today, but it is not the whole story. Medical encounters still take place between individuals, and it is difficult to argue that there is not some essential reality to illness. What Armstrong calls surveillance medicine is merely an additional layer of medical reality and, as John Pickstone has argued, the history of medicine is not ideally told as a story of successions, in which the new completely replaces older layers.[55] In fact, the hospital medicine of Foucault’s *The Birth of the Clinic* (Pickstone calls this analytical medicine) and the older, individualist model of patronage medicine (Pickstone identifies it as biographical

medicine), both continue to play important roles in certain realms of modern medical practice.[56]

Canguilhem's book, *The Normal and the Pathological*, was conceived at the beginning of the rise of the risk factor model. His criticism is mostly directed towards the growing influence of physiological concepts in medical practice. *The Normal and the Pathological* was partly a contribution to the debate over the old question if medicine is more of an art or a science. There are clear links and continuities between Canguilhem's thinking and the holist criticism of mainstream medicine that flourished in the interwar period: Canguilhem cites Kurt Goldstein, for example, as a major influence.[57] Another influence was Henry Sigerist, who is best known in the English-speaking world as a historian of medicine, but who also wrote extensively on the theory of medicine and was among the more outspoken participants in debates over a crisis of modern medicine in Weimar Germany.[58] This chapter has focused on Britain, and here criticisms analogous to those voiced by Canguilhem found their expression in long-standing concerns over the incommunicability of clinical knowledge, as analysed by Christopher Lawrence.[59]

The debate between Platt and Pickering partly had its origins in the different meanings that the notions of the 'normal' and the 'pathological' have acquired in the different realms of clinical science, medical practice and health administration. The relatively new field of geriatrics, as Armstrong shows, is one where the blurring of the boundary between normal and pathological is especially noticeable. The variation of physiological parameters becomes broader with age, and may pass into abnormality. This can (but does not have to) lead to states that are best described as pathological. But what makes

these states pathological? Here we are back with Canguilhem. It depends on the living subject and his or her environment whether a variation is perceived as unbearable. We know when we feel ill (and when we don't). According to the geriatrician Bernard Isaacs: 'the ability to define the "normal" becomes neither a matter of semantics nor statistics, but a burning issue to be decided afresh at every clinical intervention.'[60]

The notion of a risk factor has added a new dimension to the question of what is healthy and what pathological. Being identified as 'at higher than average risk' (to suffer a stroke, for example) reconfigures a subject as not quite healthy, but not quite ill either. A risk factor is not automatically a disease, neither is it a clearly identifiable cause of disease. It is, for example, a behavioural pattern (such as smoking) or a physiological parameter (such as blood pressure) associated statistically with the development over time of disease in a population. But what does this mean for individuals? If, as Canguilhem argues, disease is 'not merely the disappearance of a physiological order but the appearance of a new vital order', so is the assurance that an individual may be at risk of premature death or disability.[61] Whether blood pressure is normal or not is a fairly theoretical question. But when an individual, who may feel perfectly healthy at the time, is told that his or her high blood pressure may affect her life expectancy or quality of life, and when he or she is advised to undergo treatment to lower this blood pressure, this affects what Canguilhem calls normativity and turns the individual into a patient.

Sociological studies have shown that in individual clinical encounters both medical staff and patients tend to translate statistical risk into binary

categories of normal or abnormal, sick or healthy, which are more easily grasped.[62] While people with high blood pressure may not feel ill, they are nevertheless entering a new stage in their lives when they are told that they are ‘at risk’ and are prescribed drugs to treat this risk. Being treated provides them with a new identity. In cases where the side effects of the drugs are worse than the symptoms caused by the increased blood pressure, this makes it inevitable to accept the new ‘patient’ identity. Ironically, experts and textbook authors such as Pickering have been very aware of this, warning their readers not to frighten their patients unnecessarily or bother them with unnecessary treatments. However, in practice things often look different. The increasing costs, which chronic diseases impose on the welfare system, have politicised risk factors and created an incentive to turn physiological into political norms. The regulation of physiological functions has become an important issue for the regulation of the economy, and the population approach of surveillance medicine provides a means of mediation between individual bodies, the physiological laboratory, and the administrative bodies of the welfare state.

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[1] Bryan Williams, 'Drug Treatment of Hypertension: Most patients will need a treatment cocktail – including a thiazide diuretic', *British Medical Journal*, 2003, 326, 61-62.

[2] On the history of risk factors, see William G. Rothstein, *Public Health and the Risk Factor: A History of an Uneven Medical Revolution*, Rochester: University of Rochester Press, 2003; Robert A. Aronowitz, *Making Sense of Illness: Science, Society, and Disease*, Cambridge: Cambridge University Press, 1998.

[3] Gareth Beevers, Gregory Y.H. Lip & Eoin O'Brian, 'ABC of Hypertension: The pathophysiology of hypertension', *British Medical Journal*, 2001, 322, 912-916.

[4] The guidelines issued in 2003 in the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure distinguish between normal blood pressure (<120/80 mm Hg), pre-hypertension (120/80 to 139/89), stage 1 hypertension (140/90 to 159/99), and stage 2 hypertension (160/100 and higher). See Aram V. Chobanian et al., 'The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: The JNC 7 Report', *Journal of the American Medical Association*, 2003, 289, 2560-2572. See also Janice Hopkins Tanne, 'US Guidelines say blood pressure of 120/80 mm HG is not "normal"', *British Medical Journal*, 2003, 326, 1104.

[5] C. T. Dollery, 'A Clinician Looks at the Future,' *British Journal of Clinical Pharmacology*, 1982, 13, 127-32; interview with Professor Sir Colin Dollery, conducted by the author on 3 July 2002.

[6] Georges Canguilhem, *The Normal and the Pathological*, New York: Zone Books, 1991. A useful summary and critical evaluation of the main arguments can be found in Mary Tiles, 'The Normal and Pathological: The Concept of a Scientific Medicine', *British Journal for the Philosophy of Science*, 1993, 44, 729-742. For a discussion of the book in the context of contemporary debates over the social construction of scientific knowledge, see Malcolm Nicolson, 'The Social and the Cognitive: Resources for the Sociology of Scientific Knowledge', *Studies in the History and Philosophy of Science*, 1991, 22, 347-369.

[7] For a recent appraisal and a discussion of the links in their work, see Christiane Sinding, 'The Power of Norms: Georges Canguilhem, Michel Foucault, and the History of Medicine', in Frank Huisman and John Harley Warner, eds, *Locating Medical History: The Stories and their Meanings*, Baltimore: Johns Hopkins University Press, 2004, 262-284. Some of the links are discussed by Tiles, 'The Normal and Pathological'. See also Gary Gutting, *Michel Foucault's Archaeology of Scientific Reason*, Cambridge: Cambridge University Press, 1989. For further appraisals, see the papers in a double-issue of *Economy and Society* dedicated to Canguilhem's work (volume 27, 1998, issues 2 & 3, 151-331).

[8] Dollery, 'A Clinician looks at the Future', p. 127.

[9] For the clinical symptoms, see also George W. Pickering, *High Blood Pressure*, London: Churchill, 1955, pp. 241-312; Frederick H. Smirk, *High Arterial Pressure*, Oxford: Blackwell, 1957, pp. 83-116

[10] The dispute is documented in J. D. Swales, ed., *Platt Versus Pickering: An Episode in Recent Medical History*, London: The Keynes Press & British Medical Association, 1985.

[11] Christopher Lawrence, 'Incommunicable Knowledge: Science, Technology and the Clinical Art in Britain 1850-1914', *Journal of Contemporary History*, 1985, 20, 503-20; idem, 'Still Incommunicable: Clinical Holists and Medical Knowledge in Interwar Britain', in idem and George Weisz, eds, *Greater than the Parts. Holism in Biomedicine 1920-1950*, New York & Oxford: Oxford University Press, 1998, 94-111.

[12] Christopher C. Booth, 'Clinical Research', in: Joan Austoker and Linda Bryder, eds, *Historical Perspectives on the Role of the MRC*, Oxford: Oxford University Press, 1989, 205-41. For the rise of laboratory medicine see also Steve Sturdy and Roger Cooter, 'Science, Scientific Management and the Transformation of Medicine in Britain c. 1870-1950', *History of Science*, 1998, 36, 421-466.

[13] On the history of the MRC, see Austoker and Bryder, eds., *Historical Perspectives on the Role of the MRC*; A. Landsborough Thomson, *Half a Century of Medical Research*, Volume One: *Origins and Policy of the Medical Research Council (UK)*, London: HMSO, 1973, and Volume Two: *The Programme of the Medical Research Council (UK)*, London: HMSO, 1975.

[14] In the US this was above all the successful model of Johns Hopkins Medical School, which in itself was an attempt to adapt German models for the US.

[15] Cf Booth, 'Clinical Research', Joan Austoker and Linda Bryder, 'The National Institute for Medical Research and Related Activities of the MRC,' in Austoker and Bryder, eds., *Historical Perspectives on the Role of the MRC*, 35-57; Henry Dale, 'Thomas Renton Elliot, 1877-1961', *Biographical Memoirs of Fellows of the Royal Society*, 1961, 7, 53-74; Arthur Hollman, *Sir Thomas Lewis: Pioneer Cardiologist and Clinical Scientist*, London: Springer, 1997. Helen Valier, *The Politics of Scientific Medicine in Manchester*, Unpublished PhD dissertation, Manchester: University of Manchester, 2002. Lewis, who was on the payroll of the Medical Research Committee since 1916 was also greatly influenced by James MacKenzie. See also Joel D. Howell, '"Soldier's Heart": the Redefinition of Heart Disease and Speciality Formation in Early Twentieth Century Great Britain', *Medical History* Supplement No 5, 1985, 34-52.

[16] John McMichael, 'Cardiovascular Research: Introduction,' *British Medical Bulletin*, 1952, 8, 301-303.

[17] John McMichael and W. Stanley Peart, 'George White Pickering 26 June 1904 - 3 September 1980,' *Biographical Memoirs of Fellows of the Royal Society*, 1982, 28, 431-449.

[18] See Platt's memoirs: Robert Platt, *Private and Controversial*, London: Cassell, 1972; and the obituary: 'Lord Platt of Grindleford, Bt, M.D. Sheff., M.Sc. Manc., F.R.C.P.', *Lancet*, 1978, i, 114-5.

[19] For the significance of this development, see Valier, *The Politics of Scientific Medicine in Manchester*.

[20] Cf ibid., pp. 294-303.

[21] Robert Platt and S. W. Stanbury, 'Sympathectomy in Hypertension', *Lancet*, 1950, i, 651-9. In this class of operations, surgeons removed sections of the so-called sympathetic ganglia, nerves that run on both sides of the vertebral column and that control the automatic responses of the body to all sorts of environmental stimuli. See F. H. Smirk, *High Arterial Pressure*, Oxford: Blackwell, 1957, pp. 401-428.

[22] Robert Platt, 'Heredity in Hypertension', *Quarterly Journal of Medicine*, 1947, 16, 111-33. We should beware of the temptation to see molecular genetics as the main origin of medical genetics, as many authors on modern medicine seem to do. See Peter A. Coventry and John V. Pickstone, 'From what and why did genetics emerge as a medical specialism in the 1970s in the UK? A case history of research, policy and services in the Manchester region of the NHS', *Social Science and Medicine*, 1999, 49, 1227-1238.

[23] Platt, 'Heredity in Hypertension', quoted after Swales, *Platt Versus Pickering*, p. 8.

[24] Ibid., p. 15.

[25] George W. Pickering, 'The Natural History of Hypertension', *British Medical Bulletin*, 1952, 8, 305-309.

[26] M. Hamilton, George W. Pickering, J. A. Fraser Roberts, and G. S. C. Sowry, 'The *Æ*tiology of Essential Hypertension. 1. The Arterial Pressure in the General Population', *Clinical Science*, 1954, 13, 11-35; idem, 'The *Æ*tiology of Essential Hypertension. 2. Scores for Arterial Blood Pressures Adjusted for Differences in Age and Sex', *Clinical Science*, 1954, 13, 37-49; George W. Pickering, J. A. Fraser Roberts, and G. S. C. Sowry, 'The *Æ*tiology

of Essential Hypertension. 3. The Effect of Correcting for Arm Circumference on the Growth Rate of Arterial Pressure with Age', *Clinical Science*, 13, 1954, 267-71; M. Hamilton, George White Pickering, J. A. Fraser Roberts, and G. S. C. Sowry, 'The Ætiology of Essential Hypertension. 4. The Role of Inheritance,' *Clinical Science*, 1954, 13, 273-304.

[27] George W. Pickering, *Hypertension: Causes, Consequences and Management*, Edinburgh & London: Churchill Livingstone, 1974, p. 33.

[28] Thomas R. Dawber, *The Framingham Study: The Epidemiology of Atherosclerotic Disease*, Cambridge, Mass. & London: Harvard University Press, 1980. W. E. Miall, 'Follow-Up Study of Arterial Pressure in the Population of a Welsh Mining Valley', *British Medical Journal*, 1959, ii, 1204-10.

[29] William B. Kannel, Thomas R. Dawber, Abraham Kagan, Nicholas Revotskie, and Joseph Stokes, 'Factors of Risk in the Development of Coronary Heart Disease - Six-Year Follow-up Experience', *Annals of Internal Medicine*, 1961, 55, 33-50.

[30] David Armstrong, 'The Rise of Surveillance Medicine', *Sociology of Health and Illness*, 1995, 17, 393-404; idem, *Political Anatomy of the Body: Medical knowledge in Britain in the Twentieth Century*, Cambridge: Cambridge University Press, 1983.

[31] This is indicated in a lecture draft by Pickering on 'The Genetic Factor in Essential Hypertension', Wellcome Library, PP/GWP/D.2. For the history of medical genetics in Britain and the important role of Fraser Roberts, see Coventry and Pickstone, 'From what and why did genetics emerge as a

medical specialism in the 1970s in the UK?'; Peter A. Coventry, *The Dynamics of Medical Genetics: the Development and Articulation of Clinical and Technical Services under the NHS, especially at Manchester*, Unpublished PhD dissertation, Manchester: University of Manchester, 2000. On Fraser Roberts, see also P. E. Polani, 'John Alexander Fraser Roberts, 8 September 1899 - 15 January 1987', *Biographical Memoirs of Fellows of the Royal Society*, 1992, 38, 306-22. On the rise of statistical thinking and its implications, see Ian Hacking, *The Taming of Chance*, Cambridge: Cambridge University Press, 1990.

[32] On eugenics and its transformations in the light of the Nazi atrocities in Germany, see Daniel J. Kevles, 'Out of Eugenics: The historical politics of the human genome', in Daniel J. Kevles and Leroy Hood, *The Code of Codes: Scientific and Social Issues in the Human Genome Project*, Cambridge, Mass: Harvard University Press, 1992, 3-36; Diane Paul, *The Politics of Heredity: Essays on Eugenics, Biomedicine, and the Nature-Nurture Debate*, Albany: State University of New York Press, 1998.

[33] Letter, R. Platt to H. Harris, quoted after Coventry and Pickstone, 'From what and why did genetics emerge as a medical specialism in the 1970s in the UK?', p. 1232.

[34] On the normalising power of graphical representations, see David Gugerli and Barbara Orland, eds, *Ganz normale Bilder: Historische Beiträge zur visuellen Herstellung von Selbstverständlichkeit*, Zurich: Chronos, 2002.

[35] S. L. Morrison and Jerry N. Morris, 'Epidemiological Observations on High Blood-Pressure Without Evident Causes', *Lancet*, 1959, ii, 864-70; William E.

Miall, ‘Follow-Up Study of Arterial Pressure in the Population of a Welsh Mining Valley’, *British Medical Journal*, 1959, 1204-10. For the history of these units, see A.R. Ness, L.A. Reynolds and E.M. Tansey, eds, *Population-based Research in South Wales: the MRC Pneumoconiosis Research Unit and the MRC Epidemiology Unit*, London: Wellcome Trust, 2002; Shaun Murphy, ‘The early days of the MRC Social Medicine Unit’, *Social History of Medicine*, 1999, 12, 389-406; Virginia Berridge, ‘Celebration: Jerry Morris’, *International Journal of Epidemiology*, 2001, 30, 1141-5.

[36] For correspondence on the preparation for the South Wales study, see George W. Pickering papers, Wellcome Library London, PP/GWP/C.6/51.

[37] Interview with Professor Walter Holland, London, 15 April 2002.

[38] Sturdy and Cooter, ‘Science, Scientific Management and the Transformation of Medicine in Britain’.

[39] Ibid., p. 446.

[40] Armstrong, ‘The Rise of Surveillance Medicine’, idem, *Political Anatomy of the Body*.

[41] Quoted after Polani, ‘John Alexander Fraser Roberts’, p. 319.

[42] See Ness, Reynolds and Tansey, eds, *Population-based Research in South*; Murphy, ‘The early days of the MRC Social Medicine Unit’; Berridge, ‘Celebration: Jerry Morris’.

[43] Rothstein, *Public Health and the Risk Factor*.

[44] See *Measuring the Risk of Coronary Heart Disease: A Symposium*, Supplement to *American Journal of Public Health*, April 1957, 47.

[45] David Spurgeon, 'NIH promotes use of lower cost drugs for hypertension', *British Medical Journal*, 2004, 328, 539.

[46] W. S. Peart, 'The Problem of Treatment in Mild Hypertension', *British Journal of Clinical Pharmacology*, 1982, 13, 82-90; Medical Research Council Working Party, 'MRC trial of treatment of mild hypertension: principal results', *British Medical Journal*, 291, 1985, 97-104.

[47] For an insightful analysis of the links between technological innovation and the framing of disease, see Keith Wailoo, *Drawing Blood: Technology and Disease Identity in Twentieth-Century America*, Baltimore: Johns Hopkins University Press, 1997.

[48] Pickering, *Hypertension: Causes, Consequences and Management*, p. 33.

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[50] Austin E. Doyle, 'The Introduction of Ganglion Blocking Drugs for the Treatment of Hypertension', *British Journal of Clinical Pharmacology*, 1982, 13, 63-65; William D. M. Paton, 'Hexamethonium', *British Journal of Clinical Pharmacology*, 1982, 13, 7-14; F. H. Smirk, 'Hypotensive Actions of Hexamethonium Bromide and some of its Homologues: Their Use in High Blood-Pressure', *Lancet*, 1952, ii, 1002-1005; Edward D. Freis, 'Recent Developments in the Treatment of Hypertension', *Medical Clinics of North America*, 1954, 38, 363-374.

[51] George W. Pickering papers, Wellcome Library London, PP/GWP/C.6/69.

[52] 'Hypertension, food rationing advisory committee: rice diet; 1948-1950', MRC Papers, UK National Archives, FD1/396.

[53] Robert M. Kaiser, 'The Introduction of the Thiazides: A Case Study in Twentieth-Century Therapeutics,' in Gregory J. Higby and Elaine C. Stroud, eds, *The Inside Stroy of Medicines: A Symposium*, Madison, WI: American Instiute of the History of Pharmacy, 1997, 121-37; Karl H. Beyer, 'Discovery of the Thiazides: Where Biology and Chemistry Meet', *Perspectives in Biology and Medicine*, 1977, 20, 410-20; idem, 'Chlorothiazide,' *British Journal of Clinical Pharmacology*, 13, 1982, 15-24.

[54] Armstrong, *Political Anatomy of the Body*, p. 67.

[55] John V. Pickstone, 'The Biographical and the Analytical: Towards a Historical Model of Science and Practice in Modern Medicine', in Ilana Löwy, ed., *Medicine and Change: Historical and Sociological Studies of Medical Innovation*, Paris: Les Editions INSERM - John Libbey, 1993, 23-46. For the displacement model, see N. D. Jewson, 'The Disappearance of the Sick Man From Medical Cosmology, 1770-1870', *Sociology*, 1976, 10, 225-44.

[56] Michel Foucault, *The Birth of the Clinic*, London: Tavistock, 1973.

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[59] Lawrence, 'Incommunicable Knowledge'; idem, 'Still Incommunicable'.

[60] Bernard Isaacs, 'Has Geriatrics advanced?' in idem, ed., *Recent Advances in Geriatrics*, London: Churchill Livingstone, 1978, 1-5, p. 2.

[61] Canguilhem, *The Normal and the Pathological*, p. 193.

[62] Sonja Olin Lauritzen and Lisbeth Sachs, 'Normality, risk and the future: implicit communication of threat in health surveillance', *Sociology of Health and Illness*, 2001, 23, 497-516.