Tuberculosis, Social Inequality, and the Hospital in Nineteenth-Century Scotland

by

Amy Walker Farnbach Pearson

A Dissertation Presented in Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

Approved November 2013 by the
Graduate Supervisory Committee:

Jane Buikstra, Chair
Rachel Fuchs
Alexandra Brewis Slade
Charlotte Roberts

ARIZONA STATE UNIVERSITY

December 2013

ABSTRACT

Medical practice surrounding tuberculosis (TB) treatment in two nineteenth-century Scottish charitable hospitals reveals that in developing empirically-positioned constructs of this and related diseases, medical practitioners drew upon social assumptions about women and the working classes, thus reinforcing rather than shedding cultural notions of who becomes ill and why. TB is a social disease, its distribution determined by relationships among human groups; primary among these is the patient-practitioner relationship, owing to the social role of medical treatment in restoring the ill to both health and society. To clarify the influence of cultural context upon the evolution of medical constructs of TB, I examined Glasgow Royal Infirmary (GRI) and Royal Infirmary of Edinburgh (RIE) ward journals, admissions registers, and institution management records from 1794 through 1905. Medical practice at the turn of the nineteenth century was dominated by observation and questioning of the patient, concordant with conceptions of physicians' labor as mental rather than physical. This changed with the introduction of the stethoscope in the 1820s, which together with the dissection of the poor allowed by the 1832 Anatomy Act ushered in disease concepts emphasizing pathological anatomy. Relationships between patient and practitioner also altered at this time, exhibiting distrust and medical dominance. The mid-Victorian era was notable for clinicians' increasing interest in immorality's contributions to ill health, absent in earlier practice and linked to conceptions of women and the working classes as inherently pathological. In 1882, discovery of the tubercle bacillus challenged existing nutritional, hereditary, and environmental explanations for TB. Although practitioners utilized bacteriological methods, this discovery did not revolutionize diagnosis or treatment. Rather, these older models were incorporated with perceived behavioral, environmental, and biological degradation of the working classes, rendering marginalized groups "soil" prepared

for the "seeds" of disease — at risk, but also to blame. This framework, in which marginalized groups contribute to their increased risk for disease through refusal to accord with hegemonically-established "healthy" behavior, persists. As a result, meaningful change in TB rates will need to address these longstanding contributions of social inequality to Western medical treatment.

For Faye, Bruce, and John, lost;

for Osbjorn and Frederick, found.

ACKNOWLEDGMENTS

Thanks are due to a great many people and organizations who made this dissertation possible. Jane Buikstra's unstinting support for my academic development and for this project kept me focused through numerous hurdles. Without her, I cannot say the project would have suffered; rather, without her, the project would not be. Rachel Fuchs's guidance in developing this dissertation's historical methodology and her kind but challenging critique of early drafts improved the final product immensely. Charlotte Roberts sparked my initial interest in tuberculosis in a presentation at the University of Colorado at Boulder; to her I also owe a debt of gratitude for her input in developing multiple iterations of my dissertation project, including of course its final form. Alexandra Brewis Slade's thoughtful feedback was instrumental in garnering funding as well as in framing and refining this culminating work. Thank you all.

I also owe thanks to a number of organizations and individuals in Glasgow and Edinburgh. Malcolm Nicolson secured my status as an honorary research associate at the University of Glasgow during my stay there and assisted me in obtaining a visa. Lydia Marshall and the other staff of the University of Glasgow's Centre for the History of Medicine were instrumental in smoothing the many details involved with settling me in Glasgow and establishing my affiliation with the university. The Centre for the History of Medicine further provided office space and computing resources during my time there. Both the University of Glasgow and the University of Edinburgh granted me access to their libraries for supplemental research. Alistair Tough and the National Health Service Greater Glasgow and Clyde Board Archives staff advised me on the records best suited for this project and were tireless in locating those I needed in Glasgow; Laura Gould and the Lothian Health Service Archive staff performed the same services for me at the University

of Edinburgh. Kate Dall provided housing upon my arrival in Glasgow. All of these individuals worked to make my proposed research a reality, and I am profoundly grateful.

The contributions of my friends and family have been significant as well. The Murray – Eastham family offered me their home and companionship during my stay in Glasgow. Good friends and mentors who have offered advice, insight, critique, and support throughout this process are too many to name, but most notably include Shamsi Daneshvari, Kristi Lewton, Carolynn Matras, Stephanie Meredith, Anne Stone, and the Spring 2007 proposal writing class. Family has offered the same: I thank all of the Farnbachs, Thomtons, and Pearsons for their unstinting support of my research and writing. My best thanks are due to Osbjorn and Frederick Pearson, for everything.

A number of organizations have supported financially my research and writing. This material is based upon work supported by the National Science Foundation under Grant No. BCS-0852610: any opinions, findings, and conclusions or recommendations expressed in this material are those of the author and do not necessarily reflect the views of the National Science Foundation. The School of Human Evolution and Social Change at Arizona State University provided institutional and financial support. The Graduate and Professional Students Association of Arizona State University provided conference travel grant funding, which assisted me in presenting early versions of portions of this work for feedback. Cake Fetish and the University of New Mexico Department of Anthropology employed me during my data analysis and writing. This dissertation would not have been possible without these organizations' assistance.

TABLE OF CONTENTS

Page

LIST OF TABLES....xi

LIST OF FIGURES....xiv

LIST OF ABBREVIATIONS....xv

CHAPTER

1 INTRODUCTION....1

Social Responses to Disease....3

Illness as Deviance and the Role of Medical Treatment....5

Social Structure and Subjectivity in Medicine....8

Tuberculosis and Society....10

Histories of British Tuberculosis....15

Overview of the Dissertation....17

2 METHODS....23

Data Collection....27

Case Notes....27

Admission Registers....34

Administrative Records....37

Analysis....38

Qualitative Methods....38

Quantitative Methods....40

CHAPTER Page

3 ESSENTIALISM, 1794 – 1820 44

Disease Concepts 46

Essentialism 52

Moral Influences 53

Gender Influences 54

Etiology 55

Specificity of Diagnosis 57

Care and Treatment 60

The Role of Physicians 66

Conclusions 70

4 THE RISE OF PATHOLOGY, 1821 – 1840 71

Dissection and the Anatomy Act 73

The British Introduction of Percussion and Auscultation 77

The Stethoscope in Glasgow 83

Dissection, Physical Examination, and Charitable Medical Practice 96

Conclusions 102

5 JOHN HUGHES BENNETT, PHYSICAL DIAGNOSIS, AND THE POSSIBILITY OF CURE 103

Medical Theory, Training, and Practice 106

TB as a Nutritional Disorder 120

Cod Liver Oil and the Question of Curability 123

Conclusions 132

CHAPTER Page

6 PHYSICAL DISEASE AND SOCIAL PATHOLOGY, 1841 – 1880 134

Patients, Practitioners, and the Role of Medicine 135

The Importance of Medical Care 136

The Practitioner-Patient Relationship 139

Medical Authority 144

Authority, Technology, and Diagnosis 147

Patient Narratives 148

Signs, Symptoms, and Diagnosis 150

Technology and Medical Perception 161

The Persistence of Essentialism 168

Nutritional Etiology in Practice 168

Heredity, Environment, Behavior, and Disease 175

Disease in the Social Body 184

Conclusions 189

7 DEGENERATION AND THE TUBERCLE BACILLUS, 1881 – 1905 190

The TB Bacillus in Britain 192

Bacteriology Comes to GRI 198

Diagnostic Continuity in the Bacteriological Era 201

The Introduction of Bacteriological Methods 213

Contagion, Curability, and Treatment 223

Degeneration, Improvement, and Tubercular Disease 235

"Congenial Soil" 240

Conclusions 245

CHAPTER Page

8 CONCLUSIONS 246

WORKS CITED 256

Unpublished Primary Sources 256

Published Primary Sources 256

Published Contemporary Sources 266

APPENDIX

A DOCUMENTATION OF IRB EXEMPTION 276

B FREQUENCIES OF DIAGNOSES CLASSIFIED AS TB-RELATED 278

C SIGNS AND SYMPTOMS RECORDED IN SAMPLED CASES FOR THE ESSENTIALIST PERIOD, 1794 – 1820 281

D SIGNS AND SYMPTOMS RECORDED IN SAMPLED CASES FOR THE PATHOLOGICAL PERIOD I, 1821 – 1840 289

E SIGNS AND SYMPTOMS RECORDED IN SAMPLED CASES FOR THE PATHOLOGICAL PERIOD II, 1841 – 1880, AT GLASGOW ROYAL INFIRMARY 301

F SIGNS AND SYMPTOMS RECORDED IN SAMPLED CASES FOR THE PATHOLOGICAL PERIOD II, 1841 – 1880, AT THE ROYAL INFIRMARY OF EDINBURGH 315

G SIGNS AND SYMPTOMS RECORDED IN SAMPLED CASES FOR THE BACTERIOLOGICAL PERIOD, 1881 – 1905 336

H TREATMENTS RECORDED IN SAMPLED CASES FOR THE ESSENTIALIST PERIOD, 1794 – 1820 359

APPENDIX Page

I TREATMENTS RECORDED IN SAMPLED CASES FOR THE PATHOLOGICAL PERIOD I, 1821 – 1840 362

J TREATMENTS RECORDED IN SAMPLED CASES FOR THE PATHOLOGICAL PERIOD II, 1841 – 1880, AT GLASGOW ROYAL INFIRMARY 368

K TREATMENTS RECORDED IN SAMPLED CASES FOR THE PATHOLOGICAL PERIOD II, 1841 – 1880, AT THE ROYAL INFIRMARY OF EDINBURGH 375

L TREATMENTS RECORDED IN SAMPLED CASES FOR THE BACTERIOLOGICAL PERIOD, 1881 – 1905 384

M COMPARISON OF STATISTICAL SIGNIFICANCE ACROSS SYMPTOM CATEGORIES, PERIODS, AND INSTITUTIONS 393

N COMPARISON OF STATISTICAL SIGNIFICANCE ACROSS TREATMENT CATEGORIES, PERIODS, AND INSTITUTIONS 395

LIST OF TABLES

Table		Page
2.1	Classification of Diagnoses	24
2.2	Standard Case Results Recorded by GRI and RIE Medical Staff	28
2.3	Sampled Years for Case Notes, Amended Protocol	30
2.4	Cases Comprising the Complete and Amended Samples	33
2.5	Sampled Years for Admissions Registers	36
3.1	GRI Admission Registers and Case Notes Samples, 1794 – 1820	46
3.2	Expectations Under the Models of Galen, Paracelsus, Cullen, and Brown	52
3.3	P-values for Fisher's Exact Tests of Differences in Clinical Indicators, 1794 – 1820	56
3.4	P-values for Fisher's Exact Tests of Differences in Treatments, 1794 – 1820	63
3.5	Outcomes, 1794 – 1820	65
4.1	GRI Admission Registers and Case Notes Samples, 1821 – 1840	73
4.2	Auscultation and Percussion Examinations, 1835 – 1840	86
4.3	P-Values for Fisher's Exact Tests of Differences in Clinical Indicators, 1821 – 1840	88
4.4	Constitutional and Humoral Remarks	92
4.5	P-Values for Fisher's Exact Tests of Differences in Treatments, 1821 – 1840	94
4.6	Outcomes, 1821 – 1840	96
5.1	Private and Charity Cases Published by J.H Bennett under full or obscured names	117
5.2	J.H. Bennett's and Other Physicians' Treatment of TB-Related Disorders with Cod Liver Oil	131

Table Page

6.1 GRI and RIE Admission Registers and Case Notes Samples, 1841 – 1880 136

6.2 P-values for Fisher's Exact Tests of Differences in Clinical Indicators at GRI, 1841 – 1880 151

6.3 P-values for Fisher's Exact Tests of Differences in Clinical Indicators at RIE, 1841 – 1880 152

6.4 Amenorrhea, 1794 – 1880 155

6.5 Clinical Indicator Category Popularity at GRI and RIE, 1841 – 1880 156

6.6 Urinalysis Beyond Visual Inspection 164

6.7 P-values for Fisher's Exact Tests of Differences in Treatments at GRI, 1841 – 1880 171

6.8 P-values for Fisher's Exact Tests of Differences in Treatments at RIE, 1841 – 1880 172

6.9 Cod Liver Oil Prescriptions, 1841 – 1880 173

6.10 Outcomes, 1841 – 1880 175

6.11 Family Histories of Disease, 1841 – 1880 177

7.1 GRI Admission Registers and Case Notes Samples, 1881 – 1905 192

7.2 P-Values for Fisher's Exact Tests of Differences in Clinical Indicators, 1881 – 1905 203

7.3 Amenorrhea, 1794 – 1905 206

7.4 Strumous History 208

7.5 Family Histories of Disease, 1841 – 1905 210

7.6 Bowel Function 211

7.7 Bacteriological Examination 215

Table Page

7.8 Single and Multiple Bacteriological Examinations 216

7.9 P-Values for Fisher's Exact Tests of Differences in Treatments, 1881 – 1905 227

7.10 Alcohol and Opium Prescriptions 228

7.11 Cod Liver Oil Prescriptions, 1841 – 1905 231

7.12 Outcomes, 1881 – 1905 232

7.13 Histories of Immoral Behavior 244

LIST OF FIGURES

Figure		Page
1.1	Mortality in Great Britain from Consumption, Phthisis, and Pulmonary Tuberculosis per 100,000 population	19
2.1	Distribution of Diagnosis Categories, Complete Sample	32
2.2	Distribution of Diagnosis Categories, Amended Sample	34
2.3	Diagnosis Category Proportions, Admissions Sample	37
7.1	Schematic Representation of Single and Multiple Bacteriological Examinations	218
7.2	Proportions of Patients Dismissed Cured, Relieved, or Neither	233

LIST OF ABBREVIATIONS

BMJ	*British Medical Journal*
EMJ	*Edinburgh Medical Journal*
EMSJ	*Edinburgh Medical and Surgical Journal*
GMJ	*Glasgow Medical Journal*
GRI	Glasgow Royal Infirmary
RIE	Royal Infirmary of Edinburgh
TB	Tuberculosis
UK	United Kingdom

CHAPTER 1

INTRODUCTION

"If the number of victims which a disease claims is the measure of its significance," wrote Robert Koch in his 1882 description of the tubercle bacillus, "then all diseases … must rank far behind tuberculosis." (221; translation from Koch 1882b, 23) The disease is ancient, appearing in the archaeological record at least 6,000 years before the present, in a Neolithic skeleton from Liguria, Italy (Formicola et al. 1987). Beginning in the seventeenth century, analogous diseases like consumption were recorded among the commonest causes of death throughout much of Western Europe (Dubos and Dubos 1952, 8). However, Koch's (1882a) discovery at the close of the nineteenth century ushered in a period of optimism regarding tuberculosis (TB) treatment and control. This culminated in the 1943 discovery of streptomycin, the first antibiotic effective against the disease; John Crofton's subsequent introduction of the Edinburgh Method combining streptomycin with para-aminosalicylic acid and isoniazid to guard against antibiotic resistance (Crofton 1959) has shaped treatment up to the present day.

Despite such advances, though, TB's toll on human populations remains staggering. After decades in decline, in the mid-1980s TB cases in England and Wales (Spence 1993) and elsewhere began to increase again. By 1990, the TB resurgence reached an estimated 7.5 million new cases per year and 2.5 million deaths, prompting the World Health Organization to declare the disease a global emergency in 1993 (Dolin et al. 1993). Twenty years later, the emergency continues, exacerbated by HIV confection and drug resistance: there were an estimated 8.6 million new cases of the disease worldwide in 2012, and 1.3 million TB deaths (World Health Organization 2013).

In 2009, there were an estimated 9.4 million new cases of TB globally (World Health Organization 2010), and as a result the 2012 incidence rates represent a marked improvement. Although the epidemic is far from resolved, the World Health Organization remains optimistic that spread of the disease will be reversed by 2015 (World Health Organization 2013). Of course, coarse-grained figures for the incidence and prevalence of TB mask a great deal of heterogeneity of risk among population subgroups. The disease can be maintained at endemic or epidemic levels within a segregated subgroup although rates remain low for the population as a whole, hindering control efforts by creating reservoirs of the disease that also pose a risk to the entire population while reducing its visibility outside of the high-risk communities (Acevedo-Garcia 2000, 1147). Thus, for example, in the United States at the start of the global TB emergency nearly seven out of ten adult TB cases and over 85 percent of cases in children occurred among minorities (Centers for Disease Control 1992); in 2009, TB in White, non-Hispanic individuals accounted for only 16 percent of United States cases overall (Centers for Disease Control 2010).

TB has long occurred most frequently among the least privileged, famously leading René Dubos and Jean Dubos (1952) to term it a social disease (xxxvii). Poverty, overcrowded and under-ventilated housing, poor nutrition, drug and alcohol use, and certain occupations have all been linked with increased risk for the disease in addition to poverty (Acevedo-Garcia 2000; Rosenman and Hall 1998; Walpola et al. 2003). Observers have noted increased rates of TB among low-income and otherwise marginalized groups consistently from the pre-antibiotic era (e.g., Chadwick 1842; Gavin 1850; Stevenson 1928) to the present day (Acevedo-Garcia 2000; Das 2006; Dievler and Pappas 1999; Farmer 1999; Gandy and Zumla 2002a; Krieger and Gruskin 2001; McMichael 1995; Rubel and Garro 1992).

Although antibiotic therapy is highly cost-effective and boasts a cure rate greater than 90 percent, it is clear that the groups most at risk for TB have the least access to appropriate treatment regimens (Farmer 1999; Gandy and Zumla 2002a). To remedy this, social scientists are increasingly examining sociocultural barriers to treatment. A wealth of research analyzes local health beliefs about TB in order to more effectively promote Western treatments (Long et al. 1999; Menegoni 1996; Nichter 1994; Promtussananon and Peltzer 2005; Rubel and Garro 1992). Other researchers, most notably Paul Farmer (2004), link unequal risk for TB and access to treatment with structural violence, a larger pattern in which life expectancy is reduced among marginalized groups: that is, the structure of society causes early death among the disadvantaged.

To Dubos and Dubos (1952), TB is a social disease not only because its distribution is socially determined, but also because it "modifies in a peculiar manner the emotional and intellectual climate of the societies that it attacks." (xxxvii) The cost of TB in human populations is morbidity and death, as well as a social malaise, the destabilization and demoralization that accompany uncontrolled or uncontrollable infectious disease. Sociocultural barriers to treatment and structural violence both arise from and feed upon such social malaise, in which circular iterations of marginalization and blame render disease the perceived-natural state of affected groups, both untreatable and recalcitrant to treatment. It is this nexus among culture, disease, and medical practice that I take as my focus in this work.

SOCIAL RESPONSES TO DISEASE

In the now-usual definition established by Kleinman et al. (1978), disease is biological. The purview of Western medical practitioners, disease comprises "abnormalities

in the structure and function of body organs and systems" in contrast to illness, the "experiences of disvalued changes in states of being and in social function; the human experience of sickness" (251). Made explicit in Kleinman and colleagues' segregation of biological disease from social illness is the distinction between empirical diagnosis and subjective experience: the varied experience of sickness in the broader social context does not accord exactly with the physiological normalities or abnormalities identified by Western medicine. In emphasizing these biological attributes, medical practice and medical science align themselves with the natural rather than the social. Nonetheless, medical conceptions of normality and abnormality are culture-laden. In identifying disease with excursions beyond what has been defined as acceptable human variation, the diagnosis of disease-as-abnormality rests upon "social as well as physical and biological facts" (Taussig 1980, 5), acting to reify a constructed normalcy by delineating its bounds with reference to an underlying natural rather than social reality.

The naturalization of disease as biological abnormality has taken place within the cultural structure its objective positioning seeks to escape. For Jean Comaroff (1982), the construction of medicine as cultural intervention in natural processes is an outgrowth from the dominance of empiricism and rationalism in the modern West. The hegemony of an "empirical objectivity … centered upon man as self-determining, biologically contrived individual … regulated by impersonal 'natural' laws" (57) serves to minimize the importance of social relationships and social structures to human lifeways and to emphasize individualism. This tends, Comaroff argues, to remove sickness from its social context, as in the significance in nineteenth- and twentieth-century germ theory of interactions between pathogen and host rather than "the interaction of the 'host' and his social and material context" (60) — although it must be noted that research interest in biosocial models of

disease has more recently increased (e.g., Acevedo-Garcia 2000; Farmer 2004). Medicine, then, has been framed as a struggle of culture — specifically, scientific culture — against nature, rather than as interpersonal conflict or the result of culturally structured interrelations (Comaroff 1982, 63).

This, like the reification of normalcy through an oppositional definition of disease, characterizes disease as a natural rather than a social phenomenon. This naturalization of disease tends to obscure rather than weaken its relationship to the social, however; naturalized as an external threat, disease parallels Mary Douglas' (1966) formulation of the perceived consequences of pollution. As Douglas implies, pollution invites negative consequences originating *outside* the social order such as naturalized disease, whether the polluting element is transgressive behavior or matter out of place. Because the consequence is natural, the transgression may also be considered such, and as a result, "pollution" may be considered the violation of naturalized boundaries. The threat of disease, externalized through disease's construction as natural, thus regains entry as a result of naturalized social transgression.

ILLNESS AS DEVIANCE AND THE ROLE OF MEDICAL TREATMENT

Despite the naturalization of such boundaries, however, groups might employ sanctions within the social order to defray the risk from transgression: hence, the naturalization of hegemonic behavior as healthy. For the Victorians, "the sum of one's transactions with the environment" (Rosenberg 1997, 29) determined one's health status. "[U]ncleanliness of the mind and body act and react, and perfect health of one is incompatible with an unhealthy state of the other," J. Milner Fothergill's (1874, 24–25)

health guide prescribed, linking physical health with moral restraint. The link is not just historic: in Peter Conrad's (1994) study of adherence to wellness recommendations regarding diet and exercise, he concludes, "even apart from any health outcomes, the pursuit of virtue and a moral life is fundamentally an aspect of the pursuit of wellness." (385) In both the Victorian era (e.g., Haley 1978; Showalter 1980; Poovey 1986) and today, the relationship between virtue and health is rendered recursive, with virtue promoting health and health-promoting behavior constructed as virtuous.

As a result, aspects of culture beyond medical practice come to be regarded as a natural barrier between human and nature, and by extension, between health and illness. A consequence of this conflation of wellness with virtue is the construction of health as intrinsically right, with both illness and disease, in contrast, as "wrong." Medically, to be diseased is to be physiologically distinct, biologically other than what one is expected to be. Medical pathology is thus both naturalized — defined by reference to an underlying natural rather than social reality — and excluded from the "normal." Because physiological normalcy is itself culture-laden, social constructs of sickness as alien or transgressive echo definitions of disease as abnormality, serving to emphasize the underlying social influence upon constructs of health as well as both illness and disease.

If health reflects virtue, the conflation of sickness with wrongness or difference similarly constructs illness as deviance. This formulation originates with Talcott Parsons (1951), for whom illness constitutes passive deviance: passive because he considers the ill to be blameless for their condition, and deviant owing to the exemption from hegemonic social expectations illness confers. More recent writing has detailed the overlap between illness and deviance, with Sally Gadow (1980), Comaroff (1982) and Linda C. Garro (1992) describing the subversion caused by illness as productive of disorder. For Gadow (1980) and Garro

(1992), illness subverts the self to the body; for Comaroff (1982), as I discussed above, naturalized disease subverts human culture to nature. Illness, in disrupting the orthodoxy of the social order and the lived body (cf. Gadow 1980), becomes heterodox. It is thus that illness is rendered deviance requiring remedial action. If illness is deviant, then health by extension becomes orthodox; orthodoxy by reflection becomes healthy, and heterodoxy unhealthy. Indeed, it is by this path that illness and disease become metaphors for social ills (Crawford 1994; Gilman 1985; Sontag 1978), both signifier of and signified by other forms of deviance.

A notable result of wellness as virtue and illness as deviance is the implication of agency through conferral of a personal responsibility to maximize one's health using socially- and physiologically-defined proper living. This is emphasized in recent work in medical sociology (e.g., Michailakis and Schirmer 2010). As Adele E. Clarke et al. (2003) summarize, "health becomes an individual goal, a social and moral responsibility … the focus is no longer on illness, disability, and disease as matters of fate, but on health as a matter [of] ongoing moral self-transformation" (171–72). This echoes Conrad's (1994) recursive conception of medicalization and "healthicization," the former "turn[ing] the moral into the medical" while the latter "turns health into the moral" (223). In consequence of such an interrelationship between constructs of illness and disease, together with individual agency in maintaining health, illness acquires blame (cf. Lorber 1967), particularly for those violating social norms. As Michailakis and Schirmer (2010) summarize, "If they had lived a better life, they would not have become ill" (943), a sentiment affecting not only the social interactions of the ill but also their medical treatment through the enshrinement of blame in medical policy, and perhaps medical attitudes, as this work will examine.

Indeed, medical treatment and practitioner-patient interactions play a dual role in the social experience of sickness. For Parsons (1951), the sick role's exemption from hegemonic social expectations bears in return an expectation for those occupying the sick role to seek and comply with medical treatment. In Judith Lorber's (1967) work, medical practitioners in diagnosing disease confirm or deny the patient's self-identification as ill, thus granting or barring exemption from social expectations. Medical practice further serves a reparative function: Michael T. Taussig (1980) argues that the doctor-patient relationship brings "the patient's conventional understandings and social personality … back into the fold of society" (4) by reaffirming — and in the case of Western medicine, reifying — social ideology (cf. Comaroff 1982). The responsibility of the sick to seek and comply with treatment, then, arises from the social need to authoritatively confirm access to the sick role and to restore ill individuals to normalcy not only biologically but also socially, once more connecting medical and social constructs of sickness in recursive interdependence.

SOCIAL STRUCTURE AND SUBJECTIVITY IN MEDICINE

Despite the myriad interactions between social and medical constructs of sickness, the overarching narrative of Western medicine has traditionally been one in which subjective illness constructs are shed in favor of asocial objectivity. In this narrative, the rise of pathologically- and bacteriologically-based Western medicine in the nineteenth century ushered in an increasingly rational medicine rooted in empirical observation and unbiased evidence. Changing ideas about disease and diagnosis occurred throughout the Western world during this period, which was marked by increasingly specialized knowledge of anatomy and pharmacology (e.g., Bennett 1841; Bennett 1853), a greater ability to infer pathology within the body from without due to new technologies like the stethoscope

(Laënnec 1819; Nicolson 1993), and a greater sense of professionalism and of the importance of medical science in society (Atkinson 1992; Brown 2010; Jordanova 1996; Lawrence 1998; Marshall 1995; Nenadic 2010; Poovey 1986; Richardson 1988).

For Michel Foucault (1963), such developments signified a fundamental shift in the relationship between practitioner and patient. The medical mapping of the human body made possible by the large-scale teaching clinics of the eighteenth and nineteenth centuries subjected the patient to an all-seeing medical gaze, through which the practitioner could penetrate the (dehumanized) body's surface to arrive at the hidden truth of a patient's condition, often hidden from the patient him- or herself. The practitioner's privileged knowledge was accompanied by increased power over the patient, but also in society as a whole, as arbiters of the ways in which disease could be conquered.

At the same time, however, social explanations for disease continued across the boundary between subjective and objective concepts of disease in both medical and social practice. Typical of this is the eighteenth- and nineteenth-century concept of diathesis, a variably heritable, occult predisposition for diseases such as TB, cancer, or mental illness necessitating careful self-management — "obey[ing] the whole physical and moral code," as one American doctor phrased it (Alcott 1857, 13) — to prevent the development of disease. That sociomedical concepts like diathesis persisted alongside the increasing empiricism of nineteenth-century medicine emphasizes the extent to which social constructs are embedded in Western medical perceptions of disease. Indeed, an environment of pervasive, concealed risk of illness has persisted to the modern day, although researchers largely downplayed or ignored this aspect of medical practice until the last quarter of the twentieth century. Since that time, however, it has been rediscovered as an element of biomedicalization (e.g., Clarke et al. 2003).

The social influences on medical thought I have discussed here — etiologies of social transgression, stigma, and blame, as well as the crystallization of biological definitions of disease without shedding social models — are particularly apparent for TB, perhaps exacerbated by its indolent and somewhat obscure course from infection to disease. For Dubos and Dubos (1952), TB was a social disease, dependent upon interactions among individuals as much as interactions between the pathogen and host. It remains so sixty years later. In recognition of the impact of sociocultural context on the distribution and experience of disease, it has become common in recent years to call for historical investigation of such contemporary health disparities (e.g., Farmer 2004; Gandy and Zumla 2002a; Gandy and Zumla 2002b; McMichael 1995; Parker and Aggleton 2003; Porter and Ogden 2002; Taussig 1980). Indeed, such concerns are today re-emergent within an environment cognizant of the longstanding interplay among social Otherness and medical and social constructs of sickness, from the stigma associated with disability and illness (e.g., Das 2001; Crawford 1994; Gregg 2011; Lawrence et al. 2008; Muenning 2008; Parker and Aggleton 2003; Scambler 2009) to the emphasis on personal agency in maintaining health (Michailakis and Schirmer 2010). Indeed, these contemporary sociomedical problems still bear ideas about health and sickness established prior to the germ theory "revolution" in the nineteenth-century context within which the contemporary practice of Western medicine evolved.

TUBERCULOSIS AND SOCIETY

Both disease and illness are laden with social meaning. What can it signify, then, to term TB a social disease? "Captain of all these men of death," (Bunyan 1680, 244) TB has long loomed large in the Western imagination, altering and being altered by the societies

within which it has flourished. The modern global TB emergency arrived weighted with centuries of human experience; coming to grips with this iteration of the disease will mean grappling with the disease's history in human populations.

TB is a bacterial infection caused by *Mycobacterium tuberculosis* or other strains of the ancient and closely-related *M. tuberculosis* complex. Traditionally, medical researchers have thought *M. tuberculosis* to have emerged as a human adaptation of the cattle strain *M. bovis* following that species' domestication, a view that still finds adherents (e.g., Wolfe et al. 2007). However, recent analyses of the patterning of genetic variation among *M. tuberculosis* complex strains indicate that their common ancestor was a human pathogen — or at least more closely related to the human strains *M. canettii* and *M. tuberculosis* than *M. bovis* — approximately 40,000 years before the present (Gutierrez et al. 2005; Brosch et al. 2002; Wirth et al. 2008), when anatomically modern humans were first beginning to migrate throughout Eurasia.

As we understand it today, the primary mode of TB transmission is via the inhalation of bacteria-laden sputum droplets. In up to 90 percent of individuals infected (Sundareshan and Evans 2010, 74), latent infection results when the immune system forms granulomas, structures that contain the bacilli at the initial site of infection and the tubercles for which the disease is named. Initial or subsequent failure of this containment results in proliferation of destructive lesions, progressive infection, and TB disease.[1] Although the site of disease is most often the lungs (historically termed consumption and phthisis), it can also affect the glands (scrofula), digestive system (tabes mesenterica), skeleton (Pott's disease of the spine, strumous or scrofulous joints), skin (lupus vulgaris), and nervous system (tubercular meningitis).

[1] For a combined overview of this process expressed allegorically to World War II, see Wilmer (1943).

The symptoms of TB or TB-like diseases in humans were familiar even in antiquity — Chinese texts ca. 2700 BCE reference a "consumption" not incompatible with TB, while Mesopotamian sources ca. 675 BCE describe hemoptysis and fever consistent with the disease (Aufderheide and Rodriguez-Martín 1998, 127). The Hippocratic corpus (ca. 400 BCE) addresses both pulmonary and disseminated TB (ibid.). Scrofula (king's evil) is noted in records from the fifth through eighteenth centuries CE in France and eleventh through eighteenth centuries CE in England, owing to the belief that the king could heal cervical lymphadenitis with a touch and attendant ceremonies in which he purported to do so (Dubos and Dubos 1952, 6–8; Keers 1978, 18). By the seventeenth century, the seemingly disparate syndromes of phthisis, scrofula, and other extrapulmonary forms of TB began to be linked, originating with Franciscus Sylvius' association between scrofula and phthisis (Myers 1974, 214).

The disorders historically analogous to TB — consumption, phthisis, and tabes — are all synonymous with wasting, and the disease that would be known as TB was once only one of many different wasting diseases (e.g., bloody flux, blood loss, and diabetes) named alike according to this symptom (cf. Morton 1720). It was during the eighteenth century, however, that medical writers redefined other conditions away from wasting terms, leaving TB as the only consumption recognized by the British as such; the symptoms and course of pulmonary TB in the modern view — the appearance of pulmonary tubercles, their rupture to form abscesses, and the subsequent hemoptysis and systematic disease — are furthermore described in texts from this time (e.g., Moore 1782; Sayre 1790). Although the causality of TB remained poorly understood from the modern point of view, both Richard Morton, in his encyclopedic *Phthisiologia* (1720, 67), and Benjamin Marten, in his *A New Theory of Consumptions* (1720), asserted that the disease was contagious during this period.

Despite these early British advocates for a contagious TB, existing historiography indicates that the British medical establishment continued to espouse non-contagious etiologies for TB until late in the nineteenth century. The Western nineteenth-century climate of increasingly scientific ideas about health and medicine spurred academic discourse in Scotland specifically (Lawrence 1988) and Britain more generally (Lawlor 2006; Smith 1988) as investigators sought to align new findings about TB and its analogues with established etiologies of heredity and morality. Both medical and social commentators in the nineteenth-century UK closely associated health with morality (e.g., Conrad 1994; Crawford 1994; Tesh 1988), and transgressive behaviors were counted among the causes of TB and analogous conditions; according to F. B. Smith (1988), diagnoses of these conditions relied on moral etiologies such as sexual incontinence and alcohol consumption until at least the 1860s (27–31).

Through such explanations, researchers made sense of consumption's distribution among individuals, while conceptually distancing healthy Self from consumptive Other (cf. Barker 2002; Broom and Woodward 1996; Gilman 1995b; Gilman 1995a; Gilman 1985; Lawlor and Suzuki 2000; Sontag 1978). At the same time, however, a conceptual shift is indicated by the lexical transition from wasting terms to tuberculosis, coined in Germany in 1839 (Keers 1978, 49). The conversion to a diagnostic category descriptive of the disease's internal signs — the development of tubercles rather than externally visible wasting — suggests that medical scientists and practitioners placed increased confidence in their ability to infer pathology within the body from signs and symptoms perceptible on the outside, made possible by new technologies like the stethoscope.

In the last third of the nineteenth century, however, exogenous etiologies such as environmental contagion and inflammation were incorporated into the British medical

construct of TB and its analogues, according to Michael Worboys (2001, 87). The accompanying recognition that not all consumptives were Other — some had the disease imposed upon them from the outside, and any might someday be cured — would seem to require changes to prevailing notions of who was consumptive and why. Nonetheless, Worboys (2001; 2000) argues that practitioners' solution was not abandonment of essentialist concepts of the consumptive. Rather, similarly to Tammy Duerden Comeau's (2007) observations on the tenacity of gendered constructs of cancer in nineteenth-century Britain, older hereditary etiologies were reconstructed to encompass newer idiopathic and exogenous causes.

Although Koch's (1882a) research associated tubercles with causative bacteria, researchers noted that exposure to tuberculous individuals did not appear to cause TB in healthy individuals. Accordingly, medical practitioners and researchers debated in what sense the disease was contagious, or indeed whether Koch's bacilli were truly associated with the disease at all. If the contagion theory were correct, wrote the American practitioner N. W. to *Science* in 1886, "the conclusion would seem self-evident that floating particles of dried sputa, or at least when freshly thrown off from the diseased subject, might easily enter the lungs of healthy persons, and reproduce the disease. Unfortunately clinical evidence does not support this *a priori* deduction." (86) Another American practitioner, G. W. Hambleton, agreed, declaring four years later that "accepted theories of consumption [including Koch's] must be rejected, because they … are incapable of offering an adequate explanation of all the known facts." (1890, 258)

After the first quarter of the twentieth century, however, Western medical practitioners and researchers widely accepted the contagion theory, and both the medical establishment and any of the public who availed themselves of various layperson's guides to

the disease (e.g., Brown 1928; Potts 1930; Wilmer 1943) understood the modern-day concept of TB. Despite this, however, control of the disease has escaped us, not for lack of effective treatments — although those we have are losing ground to antibiotic resistance daily (Sharma and Yoder 2011) — but because of our failure to understand the ways in which our contemporary concepts of this disease have been shaped by the social context within which they developed.

HISTORIES OF BRITISH TUBERCULOSIS

This work is far from the first to examine the history of TB; a catalogue of other such works, giving each the attention it deserves, would be prohibitively long. I accordingly present here a sketch of TB historiography, and have elaborated on those works formative to my later discussion in the pertinent chapters. A wealth of research explores TB's significance in Western culture. Perhaps most prominently, Susan Sontag's *Illness as Metaphor* (1978) outlined the cultural weight TB acquired during the eighteenth and nineteenth centuries, rendering the disease a metaphor for transcendence and passion — in consequence of which, Sontag argues, the human experience of disease is transformed into a form of social theater at the expense of individuals who are suffering. David S. Barnes (1995) elaborated on Sontag's themes, examining in *The Making of a Social Disease* TB's social significance in nineteenth-century France, in which uniquely French anxieties shaped a feminine disease, redemptive despite its associations with prostitution, syphilis, and the cabaret. While Sontag (1978) argues for a conception of illness based in reality instead of metaphor, Barnes (1995) counters, "it is difficult to imagine … how a culture could understand or conceive of illness *without* metaphor," that is, "outside of history and culture." (70)

Indeed, TB in particular has loomed large in the British cultural imagination. Among others, both Clark Lawlor (2006) and Katherine Byrne (2011) elaborate on the literary metaphors to which TB has been put. Lawlor (2006) emphasizes the disease's role in the romantic aesthetic and its use as a signifier of sensibility, making TB desirable and its sufferers poetic. Byrne (2011), through a series of case studies of consumption in Victorian culture, examines the nineteenth-century British medical establishment's insistence in medical literature on TB as a disease essential to the sufferer through heredity or immoral behavior and the interplay of this concept of the disease with cultural representations of TB. Her examples illustrate TB as the wages of industrial capitalism, an expression of pathological femininity, and — as contagious etiologies took hold at the close of the nineteenth century — a dangerous colonizing force.

TB was preoccupying for medical writers, too, and much historiography has traced the development of scientific and medical theories about the disease. These include the germinal account of Dubos and Dubos (1952), which traces changes in the understanding of the disease in Western medical culture; Dubos and Dubos (1952) emphasize alterations in medical constructs of and approaches to TB during the nineteenth century such as those I have discussed above, in addition to referencing cultural assumptions surrounding the disease similar to those later addressed by Sontag (1978), Lawlor (2006), and Byrne (2011). Dubos and Dubos (1952) most notably characterized the control of TB as a social rather than medical problem, rooted as much in inequality as infection.

More recent research examines in fine detail the British histories of the development of modern medicine. Thus, for example, Dorothy Porter and Roy Porter (1989) explain the increasing social importance of trained practitioners and orthodox medical treatment in the eighteenth-century UK, which led to an increasing willingness for the sick to become

patients. F. B. Smith (1979) discusses the increasing British concern with public health during the nineteenth century, situating medical responses to TB within a detailed framework in which medical professionalization and the health of the metaphorical social body figured prominently.

A number of works further describe medical-historical understandings of TB and of particular etiologies that have figured prominently in both medical and cultural constructs of the disease. Smith (1988) went on to address TB specifically, in a history of the disease in the UK highlighting the perception of environmental factors as critical to the decline in British TB mortality. Linda Bryder's (1988) history of British TB control in the twentieth century builds upon the developments of the nineteenth century to address patients' experiences under the widespread acceptance of tubercular contagion and enactment of public health measures for its control. As an example of works more limited in scope, Worboys' (2000; 2001) work, as I discussed above, argues by examination of published medical discourse that infectious etiologies complimented rather than replaced older hereditary and behavioral explanations for disease in the wake of Koch's (1882a) discovery of the tubercle bacillus.

OVERVIEW OF THE DISSERTATION

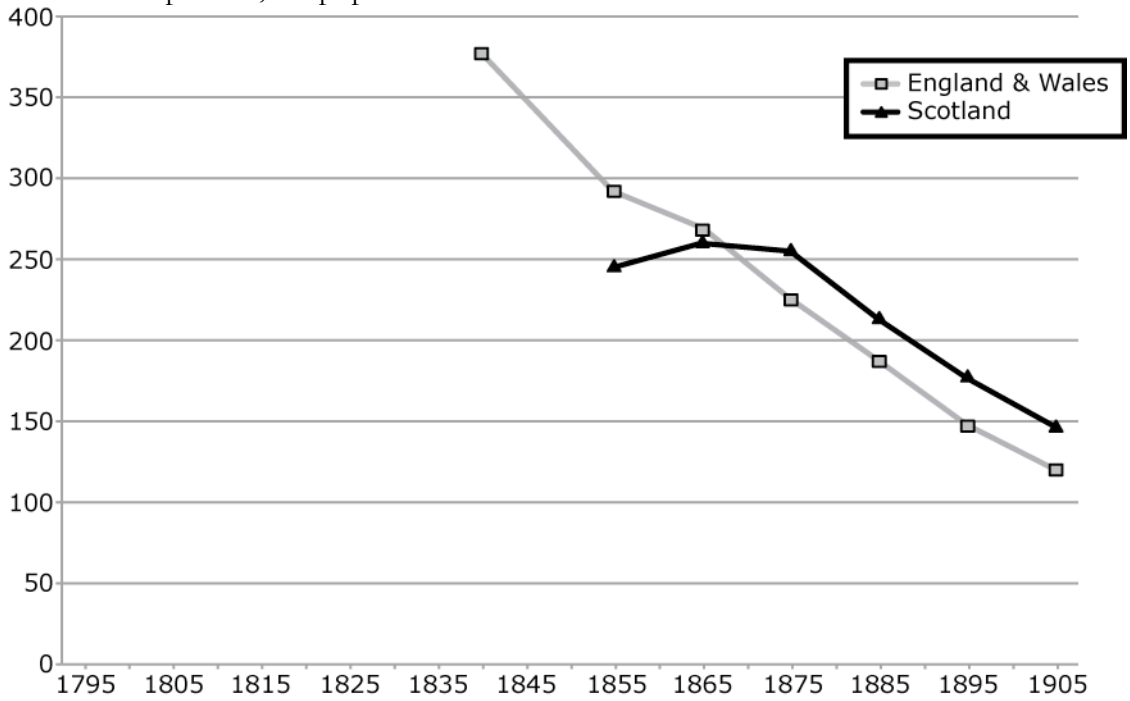
My work herein builds upon existing research to address the enactment in medical practice of scientific and cultural concepts of TB and analogous conditions such as scrofula and tabes mesenterica, and how such concepts shaped patients' lived experience of these disorders. While — as I discussed above — a wealth of research explores TB's significance in Western culture and the development of scientific and medical theories about the disease, these have tended to examine medical literature rather than practice and specialist rather than general institutions. By emphasizing medical practice, my argument centers on the

effects of medical theory on patient experience; this foregrounds practitioners' interactions *with* patients rather than their interactions with each other *about* patients in an effort to access the effects of theoretical developments on those suffering from tubercular disease. By placing the diagnosis and treatment of TB within the scope of diseases encountered by practitioners at large general institutions, I was able to explore the ways in which TB was unique as well as its commonalities with other disorders in a manner prohibited by focusing on specialist hospitals.

Existing research has furthermore examined Britain as a monolithic whole, or explicitly excluded Scotland from analysis. However, Scotland was critical to the nineteenth-century development of modern Western medical understanding of TB. Changing ideas about disease and diagnosis occurred throughout the Western world during the nineteenth century. Scotland was at this time a preeminent center for medical training, however. In contrast to England and France, where university education and clinical apprenticeship were distinct, and Germany, where the training clinics accessible to students had fewer beds (Bonner 1995), Scottish medical schools offered cohesive and broad-based academic and practical training (Jenkinson 2012; Lawrence 2006). By the early nineteenth century, about one-fourth of Edinburgh medical graduates were Scottish; an additional one-fourth were English or Welsh, and fully half originated elsewhere (Turner 1937). For doctors the world over, cases observed in Scotland provided the models for diagnosis and treatment. In addition, Scotland tended to suffer a greater burden from TB: despite the declining mortality from pulmonary TB in the *Reports of the Registrar-General* for England and Scotland (1907; 1841; 1856; 1857; 1867; 1868; 1877; 1880; 1886; 1888; 1896; 1897; 1906), throughout much of the latter half of the nineteenth century — for which mortality rates are available for

Scotland as well as England and Wales — Scotland sustained approximately 10 percent more deaths per 100,000 population than England and Wales (figure 1.1).

Figure 1.1. Mortality in Great Britain from consumption, phthisis, and pulmonary tuberculosis per 100,000 population.



In this work, I trace the development of medical constructs of TB and analogous conditions in nineteenth-century Scottish medical practice, a context critical to the development of modern biomedical theory and practice. In chapter two, I provide background on the institutions included in this study, the Glasgow Royal Infirmary (GRI) and the Royal Infirmary of Edinburgh (RIE). I also discuss the study's methodology, including sampling procedures, an overview of the sample, and my methods for archival data collection and for qualitative and quantitative analysis.

In chapters three through seven, I examine the chronological evolution of medical practice surrounding TB and related conditions. In chapter three, I focus on the period prior to the introduction of the stethoscope, from my earliest data of 1794 through 1820. During this period, essentialist explanations for TB-related disorders persisted; I discuss these etiologies and their enactment in practice at GRI, including their application to diagnosis and treatment of these diseases prior to the dramatic changes precipitated in the 1820s by the widespread Scottish acceptance of the stethoscope and access to corpses for dissection.

Chapters four, five, and six examine the effects in medical practice of the stethoscope's introduction and the increased access to cadavers facilitated by the 1832 Anatomy Act. Together, these chapters span the period from 1821 through 1880, just before Robert Koch announced his identification of the tubercle bacillus (1882a). Chapter four emphasizes the years from 1821 through 1840 at GRI, and introduces medical concepts of TB and analogous conditions centered around bodily pathology and pathological processes. I discuss the rise to predominance of pathological and anatomical thinking in diagnosis and treatment of these disorders, and the ways in which diagnosis was increasingly specific, reflecting the accumulated observations from the physical examination of live patients and dissected subjects. Specifically and perhaps most importantly, such observations led medical practitioners increasingly to question the received wisdom that TB and analogous conditions were incurable.

This question formed a raison d'être for the subject of chapter five, John Hughes Bennett. An Edinburgh physician who served as pathologist at RIE and subsequently as a professor of medicine and RIE ward doctor, Bennett wrote prolifically on TB-related diseases specifically (Bennett 1841; 1853; 1856; 1859) and medicine and physiology more generally (Bennett 1851; 1855; 1858; 1860; 1868). He included for publication a number of

case studies from among my RIE clinical sample, allowing a case study of the concordance between published medical theory and practice with respect to TB and its analogues. Bennett was a passionate advocate for the curability of the diseases he called tuberculosis and phthisis pulmonalis, and is credited with introducing to Britain the cod liver oil treatment that would remain popular throughout the nineteenth century (Bennett 1841). He emphasized the need for medical students to become adept with the stethoscope in order to correctly diagnose TB-like diseases in the early, more curable stages, and held somewhat iconoclastic opinions on the diseases' etiology. This chapter will emphasize treatment in Bennett's RIE wards, with comparative data from other RIE wards and GRI, for the period 1845 to 1874.

Chapter six examines practice at GRI and RIE more broadly for the period 1841 through 1880. Despite the emphasis on anatomy and pathology introduced in chapter four and furthered by Bennett's advocacy discussed in chapter five, behavioral and constitutional etiologies persisted in practice. This chapter will address the circumstances surrounding the persistence of these explanations and their relationship with the newer concepts of pathology and the curability of TB-like diseases. In addition, I explore the ways in which class and gender ideologies served to further behavioral and constitutional explanations for TB and its analogues.

In chapter seven, I address the introduction of bacteriological diagnosis over the period 1881 through the close of my sample in 1905. I discuss the impact of Koch's 1882 discovery of the tubercle bacillus on the diagnosis and treatment of TB and its analogues at GRI, and the incorporation of bacteriological methods into practice alongside techniques for physical examination established by the latter nineteenth century. These new methods ostensibly offered objective diagnosis — the presence of bacteria — at the same time

subjective cultural constructs of degeneration and detrimental heredity were used increasingly to explain differences among individuals and groups. I further examine the ways in which this cultural preoccupation with degeneration and the relationship of class constructs thereto were approached in medical practice.

In my concluding remarks, I will discuss the pertinence to modern health disparities of historical medical and social constructs of sickness. This work is, overall, a study of the social practice of medicine and the ways in which social and medical perceptions combined to shape nineteenth-century TB patients' experience of medical treatment. In the influential context of nineteenth-century Scotland, as I will show, the construction of TB as a biological entity developed within and relied upon a social framework and boundaries among social classes and genders, reinforcing the notion that biological disease cannot be segregated from the social.

CHAPTER 2

METHODS

I selected two sizable teaching institutions for data collection: the Glasgow Royal Infirmary (GRI) and Royal Infirmary of Edinburgh (RIE). Both institutions were established in the eighteenth century — RIE in 1729 and GRI in 1794 — as voluntary charitable institutions to provide for the medical needs of the poor; medical students and their instructors provided treatment in the context of medical training. The size and scope of these institutions allowed me to obtain a large, nineteenth-century Scottish sample of patient records including cases of TB and its historical analogues as well as cases of both pulmonary and non-pulmonary non-tuberculous disorders (see table 2.1 for the diagnoses included in each category). However, the dual aims of charity and education guiding the selection of patients admitted to RIE and GRI necessarily shaped the composition of my research sample.

Hospital policies constrained the socioeconomic range of inpatients. A distinction between the worthy and unworthy recipients of voluntary charity was pervasive in Victorian Britain, reflecting the insistence of middle-class donors upon middle-class behavioral standards among beneficiaries. During the nineteenth century, the management of both RIE and GRI explicitly sought to limit admissions to the "worthy poor," those working and morally upright. Treatment of the so-called idle poor they considered the responsibility of individuals' home parishes, which relied increasingly upon tax-funded poorhouses following the 1845 Poor Law (Scotland) Act (8 and 9 Victoria c. 83) encouraging the development of such institutions. In practice at these two institutions, however, the worthy/unworthy distinction was somewhat porous. Medical practitioners acknowledged poorhouse facilities

to be unequal to the demand for treatment: RIE's medical managers complained, "when it is said, that patients fitted only for an Alms House ought not to be admitted to the Hospital, it ought to be remembered, that there are no Alms Houses in Scotland to which the great majority of the incurable patients applying there can … claim admission and that those which exist are on so limited a scale and generally so full, as to be unable to admit many such applicants." (LHB1/1/13 1844, 508) As a result, technically ineligible patients were regularly admitted, diagnosed, and treated at GRI or RIE, although these institutions might later transfer such patients to the care of a poorhouse, or seek reimbursement for the cost of treatment from their parishes.

Table 2.1. Classification of diagnoses

TB-Related	Non-TB Pulmonary	Non-TB, Non-Pulmonary
Cachexia	Apoplexy of lung	All other diagnoses
Consumption	Asthma	
Haemoptysis	Bronchitis	
Hydrocephalus	Cancer *or* neoplasm of lung	
Lupus	Catarrh	
Phthisis	Cough	
Pott's disease	Dyspnoea	
Psoas abscess	Emphysema	
Scrofula	Empyema	
Strumous disease	Fibroid degeneration of lung	
Sub-tubercular nephritis	Hooping cough	
Tabes mesenterica	Hydropneumothorax	
Tuberculosis	Hydrothorax	
Tubercular disease	Pleural effusion	
	Pleurisy	
	Pleuritis	
	Pleurodynia	
	Pneumonia	
	Pulmonary complaints	
	Pulmonary gangrene	
	Strictura thoracis	

Note: See appendix B for a more detailed list of TB-related disorders and their frequencies.

For the most part, the inpatients at GRI and RIE were seriously ill. For less severe disorders, the working classes might self-treat or seek treatment from charitable outpatient dispensaries, apothecaries, folk healers, or poorhouses; those particularly well-off might, like the middle classes and the aristocracy, engage house calls from a physician or surgeon. Hospitals were popularly believed to experiment on patients — a fear shared throughout the UK and on the continent — and in the wake of the 1832 Anatomy Act (2 and 3 William IV c. 75) poor patients and their families also feared the possibility of postmortem dissection following death in a hospital or poorhouse (Richardson 1988, 275). As a result, for much of the nineteenth century the working classes sought hospital treatment only in extremis. Even so, the institutions from which I draw my observations admitted thousands of patients annually in the latter three-quarters of the nineteenth century (GRI: series HH67/56; RIE: series LHB1/126). Patients were thus limited in kind, but less so in number.

As a result of the medical training they conducted, GRI and RIE furthermore included categories of patients other British institutions refused. These categories included terminal patients and those suffering from diseases commonly considered incurable, including TB and its analogues. Indeed, in 1844 RIE medical staff explicitly resisted efforts by hospital management to exclude incurable and terminal patients, arguing that such patients benefitted more from hospital care than those with more easily treatable conditions, and that students' medical training required observing the full spectrum of disease (LHB1/1/13, 506–9). That part of the stated educational value in treating terminal patients lay in the possibility that their bodies would become available for dissection after dying in hospital tended to detract somewhat from the humanitarian tone of the argument as a whole. Nonetheless, medical staff at both institutions commonly petitioned on behalf of

their patients for exemptions to the stated sixty-day limit on hospital stays. Management generally approved such requests, with the result that patients regularly exceeded this limit: in an extreme example, John F., a phthisis patient, was admitted at RIE December 21, 1850 and not discharged until August 13, 1852, for a total stay of 601 days (LHB1/129/2/7; LHB1/129/2/9).

Although a sample drawn from these two institutions restricts my observations to hospital treatment by medical instructors and students of seriously ill, working-class patients, these shortcomings are mitigated by the size and aims of the hospitals themselves. These teaching institutions were influential; for doctors the world over, cases observed at GRI or RIE provided formative models for diagnosis and treatment, rendering them potentially more broadly informative. These hospitals also treated a large number of inpatients with an extensive range of disorders, allowing me to draw a sizable and far-ranging sample comparing treatment of TB and analogous conditions with other disorders.

My data collection protocol included three types of records: case notes, admission registers, and minutes of administrative meetings. These documents pertain to patient-practitioner interactions and medical practice for the full spectrum of disorders treated at the institution, as well as the attitudes of hospital management toward both patients and practitioners. I further consulted pertinent published works by nineteenth-century medical authors, particularly the influential Edinburgh physician John Hughes Bennett, in order to compare medical theory and published cases of upper-, middle-, and working-class patients with the approaches in medical practice to working-class patients as documented in case notes at GRI and RIE.

DATA COLLECTION

Because United Kingdom (UK) data protection law allows public access to personal health records more than 75 years old (100 years old if relating to minors), Arizona State University's Institutional Review Board granted my data collection protocol exempt status (appendix A).

CASE NOTES

At both RIE and GRI, case notes consisted of bound notebooks containing handwritten details of each admitted case. Each ward maintained its own casebook chronologically under the direction of the supervising physician or surgeon, resulting in a series of volumes for each ward. Each volume was generally fronted with some combination of the ward number, description (e.g., "Female Medical"), inclusive dates, and medical staff, most often the ward's supervising physician or surgeon and the principal clerk. A list of the volume's cases often followed this information, comprising some combination of patient name, admission and discharge dates, diagnosis, the result of treatment, and the page on which the clerk first recorded each patient's case. Medical staff generally described the outcome of treatment by one of eight standardized discharge notes, listed and defined in table 2.2: advice, by [patient's] desire, cured, died, improper, irregular, no change/in statu quo, and relieved.

Following this front matter, medical staff detailed patients' cases on the remaining pages, generally beginning with biographical details such as name, age, occupation, and religion, followed by the patient's own history of his or her case, a report of medical examination of the patient, and the initial course of treatment prescribed. Subsequent updates recorded changes to treatment and status reports by the patient and medical staff.

The chronological organization of these volumes caused patients' records to cover discontiguous pages. For example, in an 1835 casebook for GRI Male Medical Ward 1, John C.'s case is recorded on pages 32–33, 57, 92, 102, 112, 119 and 150 (HB 14/5/4). However, as each sequence of pages for a patient's case generally opened with the patient's name and concluded with the page on which his or her record continued, I was very rarely required to infer the identity of the patient described on a particular page contextually (matching dates, treatments, or signs and symptoms with prior entries for a patient). In instances in which I was unable to conclusively infer patient identity from text or context, I omitted subsequent updates.

Table 2.2. Standard case results recorded by GRI and RIE medical staff

Result	Explanation
Advice	Patient examined and offered advice for self-treatment of condition
By desire	Patient granted permission to leave hospital against medical advice
Cured	Patient considered cured of at least one diagnosed complaint
Died	Patient died in hospital
Improper	Patient ejected from hospital in consequence of behavioral violations
Irregular	Patient left hospital against medical advice and without permission
No change	Patient discharged in same state as admitted
Relieved	Patient improved in health

Case notes are preserved for GRI from 1794 through 1839 and from 1860 into the twentieth century; for RIE, these records are preserved from 1845 through 1875. I sampled cases according to an initial scheme that I later amended in the interest of time, designing both the initial and amended protocols to capture all TB-related cases and a comparison sample of other pulmonary disorders and of neither tuberculous nor pulmonary disorders. I utilized as illustrative examples select cases from the complete sample, consisting of data collected under both the initial and amended schemes. For quantitative analysis, I resampled

the data collected under the initial scheme to adhere the amended selection protocol as described below.

I systematically sampled every available casebook for the years 1794 through 1812 at GRI and 1845 through 1849 at RIE under the initial scheme as follows. At GRI, for which fewer casebooks were available covering each year during this earliest time period and fewer cases included in each volume, I sampled every case for which intake notes were available, as the initial consultation provided the richest source of data on patient history, the practitioner's and patient's perceptions of the disease and treatment, and the practitioner's diagnostic procedures. At RIE these earliest casebooks are more numerous and bear more cases per volume than at GRI; because of this, I used the list of cases at the beginning of each volume to select every third case for inclusion. I added to this sample all omitted cases of TB or conditions considered analogous by nineteenth-century medical authorities. Any selected cases for which intake notes were not available I omitted.

Under the amended sampling scheme, I targeted every fifth year for sampling beginning in 1795 and ending in 1905. When records for a selected year were absent, contained no TB-related cases, or contained no intake notes, I substituted the nearest year or years for which records were preserved (table 2.3). For the periods from 1813 through 1905 at GRI and from 1850 through 1875 at RIE, I sampled every casebook from sampled years as follows. Using the list of cases at the beginning of each volume, I selected for inclusion every TB-related case; I then systematically sampled half that number each of non-tuberculous pulmonary cases and cases that were diagnosed as neither tuberculous nor pulmonary, rounding up where half the number of TB-related cases was not an integer. For example, if a casebook listed seven cases of TB and analogous conditions, I would select for inclusion four cases each of non-TB pulmonary disorders and non-TB, non-pulmonary

conditions distributed as evenly as possible throughout the chronological list. Thus, if eight cases of non-tuberculous pulmonary disorders were listed, I would sample every other non-tuberculous pulmonary disorder listed. I omitted casebooks with no TB-related cases.

Table 2.3. Sampled years for case notes, amended protocol

	GRI	RIE
Year Targeted	Year(s) Sampled	Year(s) Sampled
1795	1794 – 95	*No records extant*
1800	1798, 1801	*No records extant*
1805	1807	*No records extant*
1810	1811 – 12	*No records extant*
1815	1814	*No records extant*
1820	1819	*No records extant*
1825	1826	*No records extant*
1830	1829 – 30	*No records extant*
1835	1835 – 37	*No records extant*
1840	1838 – 39	*No records extant*
1845	*No records extant*	1845 – 47
1850	*No records extant*	1850
1855	*No records extant*	1855
1860	1860	1860
1865	*No sufficiently detailed cases*	1865
1870	1870	1869
1875	1875	1874 – 75
1880	1879	*No records extant*
1885	1885	*No records extant*
1890	1893	*No records extant*
1895	1895	*No records extant*
1900	1900	*No records extant*
1905	1905	*No records extant*

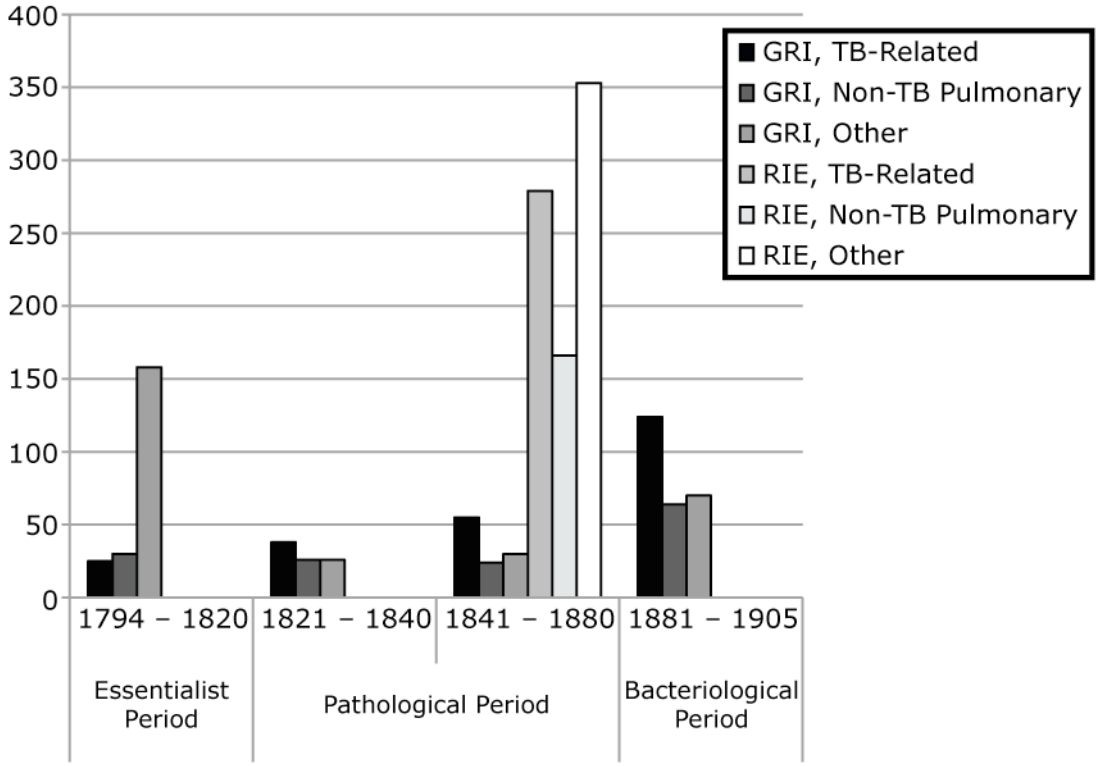
For quantitative analysis, I subsequently re-sampled the data collected under the initial sampling protocol. For the years 1794 through 1812 at GRI and 1845 through 1849 at RIE, I omitted data from casebooks that listed no TB-related diagnoses. For the casebooks with TB-related cases, I retained in the amended sample all such cases, as well as half that

number each of non-TB pulmonary cases and non-TB, non-pulmonary cases, distributed evenly throughout each volume. Under both sampling schemes, I assigned each sampled case an index number consisting of the infirmary designation, year, and an identifying number: for example, RIE1865.089 designates the 89th record sampled from RIE for the year 1865. For each sampled casebook, I recorded any front matter data, including the case list data pertaining to sampled cases. I then transcribed all case notes for each sampled case. I made every effort to trace sampled cases across casebooks from admission to discharge and to locate and transcribe any earlier or later admissions indicated for these patients, even where such readmissions included years otherwise not sampled.

Thus sampled, the complete dataset of case notes consists of a total of 1468 patient records, 670 at GRI spanning the years 1794 through 1839 and 1860 through 1905 and 798 at RIE spanning the years 1845 through 1875 (table 2.4). Of these, 213 — all from GRI — fall within a period dominated by essentialist disease concepts (1794 – 1820, late Georgian/Regency era), 998 (GRI, n = 199; RIE, n = 798) fall within a period in which medical practitioners emphasized pathological disease concepts (1821 – 1880, late Georgian/Victorian era); and 258 (all from GRI) fall within a period in which bacteriological disease concepts began to influence British medical practice (1881 – 1905, late Victorian era). I further subdivided the pathological period sample (1821 – 1880) into a period distinguished by the introduction of the stethoscope and other physical examination techniques (1821 – 1840, late Georgian/early Victorian era: GRI, n = 90) and a period marked by social influences on disease diagnosis and treatment (1841 – 1880, mid-Victorian era: GRI, n = 109; RIE, n = 798). Figure 2.1 shows the distribution of TB-related, non-TB pulmonary, and other diagnoses in the complete sample for each infirmary during each of these periods. As I

designed the sampling scheme to maximize representation of TB-related cases, figure 2.1 shows a preponderance of these diagnoses compared to the two non-TB diagnosis categories.

Figure 2.1. Distribution of diagnosis categories, complete sample



The amended case notes dataset comprises a total of 923 patient records, 506 at GRI and 417 at RIE, spanning the same years as the complete sample (table 2.4). Of these, 51 (all from GRI) fall within the essentialist period, 88 (all from GRI) fall within the earlier pathological period, 526 (GRI, n = 109; RIE, n = 417) fall within the later pathological period, and 258 (all from GRI) fall within the bacteriological period. Figure 2.2 shows the distribution of TB-related, non-TB pulmonary, and other diagnoses in the amended sample during each of these periods, again illustrating my deliberately greater inclusion of TB-related cases. For GRI, my resampling procedure to create the amended dataset for quantitative

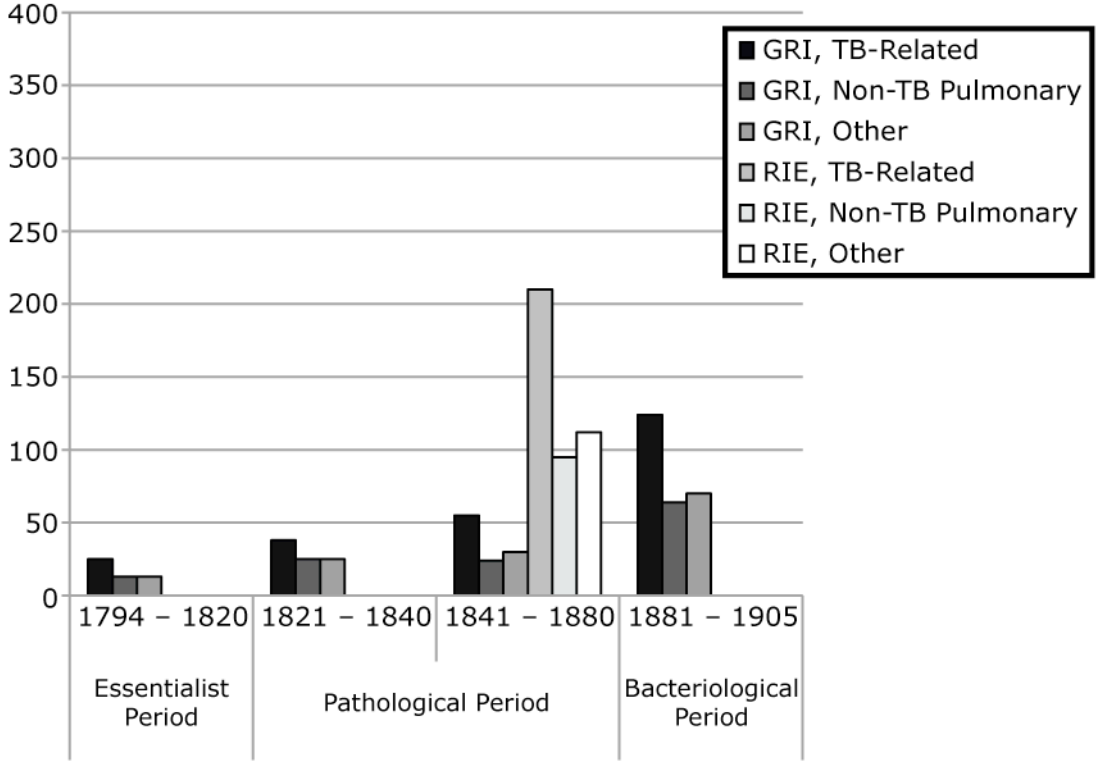
analysis reduced only the number of non-TB-related cases. For RIE, because my resampling removed entire years as well as excess non-TB-related cases, the amended dataset contains reduced numbers in all three categories of diagnoses, but especially for non-TB-related disorders.

Table 2.4. Cases comprising the complete and amended samples

	GRI			RIE		
Year Targeted	Year(s) Sampled	n_{Complete}	n_{Amended}	Year(s) Sampled	n_{Complete}	n_{Amended}
1795	1794 – 95	21	8	—		
1800	1798, 1801	69	6	—		
1805	1807	38	3	—		
1810	1811 – 12	58	7	—		
1815	1814	4	4	—		
1820	1819	23	23	—		
1825	1826	11	11	—		
1830	1829 – 30	5	5	—		
1835	1835 – 37	41	39	—		
1840	1838 – 39	33	33	—		
1845	—			1845 – 47[a]	313	24
1850	—			1850[b]	162	70
1855	—			1855[c]	102	102
1860	1860	65	65	1860	68	68
1865	—			1865	103	103
1870	1870	11	11	1869	41	41
1875	1875	28	28	1874 – 75	9	9
1880	1879	5	5	—		
1885	1885	6	6	—		
1890	1893	75	75	—		
1895	1895	11	11	—		
1900	1900	61	61	—		
1905	1905	105	105	—		
TOTAL		670	506		798	417

[a] The complete sample for this period included the years 1845 – 1849.
[b] The complete sample for this period included the years 1850 – 1854.
[c] The complete sample for this period included the years 1855 – 1857.

Figure 2.2. Distribution of diagnosis categories, amended sample



ADMISSION REGISTERS

As the extant casebooks were incomplete for many sampled years, and because I designed sampling of these volumes to capture TB-related data and comparison cases rather than a representative sample of hospital admissions as a whole, I also collected data from the more-complete admission registers to determine the proportion of admissions attributable to TB and its analogues, non-tuberculous pulmonary disorders, and other diagnoses. At RIE, the admission registers were organized by patient, comprising largely uniform volumes of tables in which clerks recorded some combination of the following data: a patient number assigned sequentially upon admission, the dates admitted and dismissed, the ward to which medical staff admitted the patient, duration of the disease prior to admission, disease(s)

diagnosed (principal, preceding, and subsequent), outcome of treatment (table 2.2), and total length of stay, as well as patient demographic data including name, age, sex, occupation, residence, native place, and whether a female patient was married.

At GRI, the admission registers were organized similarly to those at RIE for 1795 through 1800, but were subsequently organized by date in tables in which clerks recorded data pertaining to patients admitted and dismissed for a given period. For patients admitted, these data included the date admitted, by whom the patient had been recommended for admission, and demographic data including name, age, occupation, marital status, country of origin, and religion. For patients dismissed these data comprised the date dismissed, the patient's name, the ward to which medical staff admitted the patient, the disease(s) diagnosed, outcome, and total length of stay.

For GRI, due to time constraints, I sampled admission registers for every tenth year from 1795 through 1905; for RIE, I sampled every fifth year for the period for which case notes were also available (1845 – 1875). Where admissions registers were not preserved for a targeted year, I substituted the nearest year available (table 2.5), as described for casebooks above. For patients included in my case notes sample, I recorded any demographic data from the admission register that had not been included in the patients' case histories. I then recorded the total number of cases admitted under each of my three categories of diagnoses (TB-related, non-TB pulmonary, and non-TB, non-pulmonary) and the distribution of outcomes for each category. I recorded these data in a spreadsheet by register page and admission month.

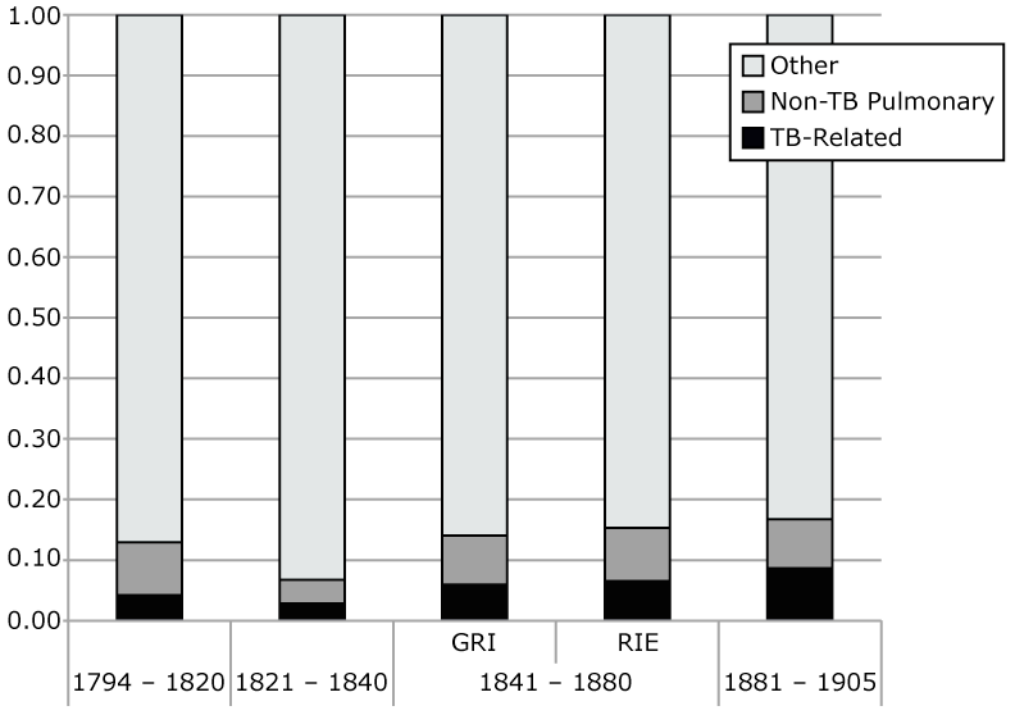
The admissions registers sampled thusly yielded a total of 68,358 patient records, 39,090 (57.2 percent) at GRI and 29,268 (42.8 percent) at RIE. Table 2.5 summarizes the

years targeted and sampled and the number of admissions by year for which I collected data. Of these, 1029 (1.5 percent, all from GRI) fall within the essentialist period (1794 – 1820), 2,323 (3.4 percent, all from GRI) fall within the early pathological period (1821 – 1840), 46,294 (67.7 percent: 17,026 (24.9 percent) from GRI, 29,268 (42.8 percent) from RIE) fall within the late pathological period (1841 – 1880), and 13,765 (20.1 percent, all from GRI) fall within the bacteriological period (1881 – 1905). Figure 2.3 shows the overall admission proportions of TB-related, non-TB pulmonary, and other diagnoses during each of these periods. These proportions differ markedly from those in my case notes sample owing to the non-representative sampling scheme I adopted in order to emphasize TB and related disorders; figure 2.3 reflects more accurately the wide range of diseases medical staff treated at these institutions, of which TB and its analogues comprised a relatively small fraction.

Table 2.5. Sampled years for admissions registers

	GRI		RIE	
Year Targeted	Year Sampled	*n*	Year Sampled	*n*
1795	1795	254	—	
1805	1803	775	—	
1815	*No records extant*		—	
1825	1826	2323	—	
1835	*No records extant*		—	
1845	1845	2987	1845	3307
1850	—		1850	4121
1855	1855	2604	1855	4029
1860	—		1860	3931
1865	1865	8644	1865	4816
1870	—		1869	4393
1875	1875	2791	1875	4671
1885	1885	4947	—	
1895	1895	5822	—	
1905	1905	7943	—	
TOTAL		39090		29268

Figure 2.3. Diagnosis category proportions, admissions sample



ADMINISTRATIVE RECORDS

In order to obtain an overview of each institution's governing principles as they related to the admission, diagnosis, and treatment of TB and analogous conditions, I also examined administrative records pertaining to hospital management and operation. For GRI, the available administrative records comprised primarily the minutes of monthly meetings of the board of managers, which I sampled every tenth year from 1795 through 1905. I also examined annual reports of the works and finances of the hospital — published to solicit charitable contributions — for every tenth year, as well as hospital correspondence of interest. For RIE, I consulted the minutes of monthly meetings of the board of managers as well as notes from these managers' site visits for every tenth year from 1845 to 1875. In sampling these records for both GRI and RIE, I recorded synopses and/or transcriptions of discussions of interest, including those related to the admission and treatment of patients

with TB-related disorders, the admission and treatment of chronic and terminal disorders more generally, the characteristics of patients considered appropriate to admit, and relations among middle- and upper-class management, working- and middle-class staff, working-class patients, and male and female staff and patients.

ANALYSIS

QUALITATIVE METHODS

I approached the analysis of practitioner concepts of diagnostic categories and of patients qualitatively across the complete sample, based upon published models for class, gender, and medical and lay constructs of illness during the study period. This aspect of my analysis included changes associated with social developments and developments in medical science, as well as any differences in clinician constructs and treatment of TB and analogous conditions among subgroups of patients by gender, class, and other medical conditions.

I applied the models that structured my analysis — described in the chapters to which they pertain — after parsing the notes from each case into statements attributable to the patient or to medical staff, as well as inferring therefrom the existence of doubt or conflict between the patient and staff members. Patient and staff accounts of the patient's case were not clearly segregated in the records for the essentialist period (1794 – 1820) as a whole, nor throughout for surgical cases, indicating the relative unimportance of this distinction in these types of records. In the pathological (1821 – 1880) and bacteriological (1881 – 1905) periods, patient history and medical examination were recorded under separate headings, offering an initial, coarse distinction between patient and medical voices.

While the explicitly medical portions of case reports could be clearly attributed to the voices of the medical practitioners and staff, I could not say the same for the patients' accounts recorded in casebooks. These originated with patients' narratives of their illnesses upon admission, but such narratives were guided by questions presented by medical staff, as in cases in which clerks recorded further explanation on a point discontiguously at the close of the patient's account. Medical staff further distilled the version recorded into those aspects considered pertinent to the patient's case from a practitioner's perspective. Thus, while I regarded the patient's recorded narrative as representative of the patient's construction of certain past events as pertinent to his or her current understanding of his or her illness (cf. Garro 1992), I was also obliged to recognize that, filtered by medical staff, it was in essence a double narrative. That is, the recorded patient history represents the construction placed by the practitioner on the patient's own construction of his or her past experience with respect to the illness. Thus distilled by the practitioner, the patient history portion of a case reinforces those events agreed by the practitioner to be pertinent and potentially elides those in conflict with the practitioner's initial interpretation of the patient's disease.

Despite this reconstruction of patients' narratives by practitioners, however, the patient's voice is not wholly inaccessible. Practitioners at times represented statements as direct quotations from the patient in case notes. In other instances, practitioners distanced themselves from patient statements using rhetorical strategies such as prefacing a statement with "as he says." W. H. Allchin (1887) explicitly advocated this approach to disavow patient statements in his instructions for taking case histories: "Since much of the information … obtained [from the patient] is liable to be uncertain, it is well to qualify it in the record with some such saving clause as 'the patient says.'" (quoted in Buscemi 2009, 1) As a result, I was

able to infer patient voice — albeit filtered by the practitioner — from such evidence of discordance between patient and practitioner accounts. I was further able to estimate from contextual cues such as the use of pejorative language or subsequent termination of treatment whether such discordance suggested conflict between the patient and practitioner.

Reading patient case notes thusly — as practitioners' representations of their own and their patients' perceptions of the patients' illnesses — my qualitative analysis evaluated a number of propositions that follow from published literature on nineteenth-century Britons and British medicine. Broadly, the propositions were as follows:

P_1: The structure of social interactions between middle-class men and working-class men and women is reflected in the medical interactions of practitioners and patients.

P_2: Published medical and social constructs of illness are reflected in practitioner and patient accounts of disease, respectively.

P_3: The intersection in medical practice of medical and social constructs of class, gender, and illness is reflected in gendered and class-based etiologies for illness in the sampled case notes.

The models and theory informing the formulation and examination of these propositions, discussed briefly in the introduction, I shall detail more fully in the pertinent chapters that follow. In addition, I undertook time-period-specific qualitative analyses, also explained in the pertinent chapters.

QUANTITATIVE METHODS

I utilized a quantitative approach to analyze the specificity of diagnosis and treatment and salient signs and symptoms used in the diagnosis of TB and analogous conditions. In order to test for statistical differences in frequency between clinical indicators associated

with TB, its analogues, and other diagnoses, I first generated contingency tables of diagnoses by signs and symptoms for each time period and institution (appendices C – G). I pooled rare clinical indicators under coarser labels (e.g., immorality), indicated in appendices C – G by notes listing the indicators comprising a pooled label. I collected clinical indicators by category or anatomical system (e.g., respiratory), then implemented Fisher's exact test in R 2.15.0 (R Development Core Team 2012) to discern between the following hypotheses:

H_{0a}: The clinical indicators recorded in cases diagnosed as TB or analogous conditions do not differ from those recorded for cases diagnosed as non-tuberculous, non-pulmonary disorders.

H_{1a}: A set of clinical indicators exists that is more commonly associated with diagnoses of TB or its analogues than with diagnoses of non-tuberculous, non-pulmonary disorders.

Owing to the large number of clinical indicators for each period, I applied Fisher's exact test to each category of signs and symptoms separately. When a category was particularly heavily populated, I was not able to dedicate sufficient computational space for R to complete the test; in such instances, I specified R use Monte Carlo simulation with 2,000 replicates to calculate the *p*-value. I have indicated *p*-values obtained through Monte Carlo simulation with a footnote in the pertinent results tables.

I performed subsequent analyses testing whether the signs and symptoms associated with TB-related diagnoses differed significantly from those associated with other pulmonary disorders, a class of conditions that might be expected to show greater overlap with the clinical indicators of TB-related diseases:

H_{0b}: The clinical indicators recorded in cases diagnosed as TB or analogous conditions do not differ from those recorded for cases diagnosed as non-tuberculous pulmonary disorders.

H_{1b}: A set of clinical indicators exists that is more commonly associated with diagnoses of TB or its analogues than with diagnoses of non-tuberculous pulmonary disorders.

I then performed a corresponding analysis — also using Fisher's exact test in R 2.15.0 — upon contingency tables for each time period of diagnoses by treatments (appendices H – L) to discern between the hypotheses:

H_{0c}: The treatments recorded in cases diagnosed as TB or analogous conditions do not differ from those recorded for cases diagnosed as non-tuberculous, non-pulmonary disorders.

H_{1c}: A set of treatments exists that is more commonly associated with diagnoses of TB or its analogues than with diagnoses of non-tuberculous, non-pulmonary disorders.

Similarly, I performed additional analyses testing whether the treatments associated with TB-related diagnoses differed significantly from those associated with other pulmonary diseases only:

H_{0d}: The treatments recorded in cases diagnosed as TB or analogous conditions do not differ from those recorded for cases diagnosed as non-tuberculous pulmonary disorders.

H_{1d}: A set of treatments exists that is more commonly associated with diagnoses of TB or its analogues than with diagnoses of non-tuberculous pulmonary disorders.

Finally, I used Fisher's exact test as described above to evaluate the following hypotheses as to outcomes:

H_{0e}: The outcomes recorded in cases diagnosed as TB or analogous conditions do not differ from those recorded for cases diagnosed as non-tuberculous, non-pulmonary disorders.

H_{1e}: The distribution of outcomes differs between TB-related and non-TB, non-pulmonary cases.

H_{0f}: The outcomes recorded in cases diagnosed as TB or analogous conditions do not differ from those recorded for cases diagnosed as non-tuberculous pulmonary disorders.

H_{1f}: The distribution of outcomes differs between TB-related and non-TB pulmonary cases.

I applied a significance threshold of $p < 0.05$ throughout each of these tests. In instances in which null hypotheses were not rejected, I included in my qualitative analysis the ways in which disease categories were constructed and applied in the absence of specific clinical indicators or treatments associated with such categories. I discuss the results of all the analyses described here in chapters three through seven.

CHAPTER 3

ESSENTIALISM, 1794 – 1820

It is no coincidence that the Glasgow Royal Infirmary (GRI) began operation in 1794. The eighteenth-century emphases on individual benevolence and virtue by Enlightenment thinkers and the Church of England combined with the popular cult of sensibility to drive an increase in private charity. One expression of this was the foundation of new voluntary hospitals throughout Britain, the earliest established in the capitol and in centers of learning like Cambridge and Edinburgh. Early industrial centers Manchester and Birmingham garnered their own voluntary hospitals midcentury, while the relatively late establishment of GRI reflects the timing of the city's rise to prominence in shipping and industry. At the turn of the nineteenth century, Glasgow was poised for the dramatic alterations that would come with continued industrialization and the Victorian era.

On the cusp of the major social, political and scientific developments of the Victorian era, the late Georgian and Regency period spanning 1794 through 1820 I examine here in many ways marks a transition. British colonialism and the coincident ascendency of British trade during the eighteenth century drove the development of a wealthy mercantile class, the nascent middle class. By the turn of the nineteenth century, the working classes were also coalescing into a self-conscious, organized entity capable of industrial action and, Tories feared, developing revolutionary tendencies — or at least a worrying level of support for the Radicals' calls to broaden political enfranchisement (Thompson 1963, 77–78). At the same time, Thomas Malthus' (1798) *Essay on the Principle of Population* upended both traditional and Scottish Enlightenment parallels between the healthy body and societal health

by recasting — as Catherine Gallagher put it — "the healthy, and consequently *reproducing* body … [as] the harbinger of the disordered society full of starving bodies" (1986, 85; emphasis retained). Together, these latter two developments would allow the middle class to consolidate political power through their ostensible representation of working-class interests (Joshi 2004, 358), driving the Victorian obsession with working-class "improvement."

Prior to Malthus, however, the health of the individual was a barometer for the health of the social body (Gallagher 1986), a precept that during the eighteenth century prompted the voluntary hospital movement to address the health of the poor. The new voluntary hospitals in Britain, like similar institutions on the continent, dramatically altered the medical landscape by treating poor patients in aggregate — expanding their access to orthodox medical care and allowing the accumulation of clinical observations critical to developing medical science — and often in association with medical training, improving the medical care available to the middle and upper classes through improved practical training (Foucault 1963, 84). The seventeenth- and eighteenth-century expansion of trade in exotic botanicals used as medical drugs added to the changing face of medicine during this period, making a wide range of compounds more available to medical consumers (Wallis 2012). At the same time, the reformation of medicine from the "poor philosophy and worse practice" of the seventeenth century (Pender 2006, 13) to an increasingly demanded service in the eighteenth century hinged upon recasting the profession as competent, respectable and necessary to a health-conscious population (Porter and Porter 1989, 208–209).

In this chapter, I address the culmination of such social and medical developments in hospital practice, exemplified in the diagnosis and treatment of tuberculosis and its historical analogues at GRI during the period 1794 – 1820. Using data from 782 admissions and 214

individuals' case notes (table 3.1), I first discuss the disease concepts in evidence at GRI, comparing these to expectations generated from published eighteenth-century medical works and contemporary medical historiography. I similarly address the treatment of tuberculosis-like disorders with respect to the era's competing Galenic and Paracelsian models of medicine. Finally, I examine diagnostic process from both the patient's and physician's points of view, relating hospital examinations to the development of medical knowledge and class conceptions during this period. My analysis only weakly supports the existing conception that constructs of disease as non-specific, holistic and externally visible, with strong moral etiologies, dominated medical practice at the turn of the nineteenth century; instead, I argue for a practice in dynamic transition toward the pathological disease concepts that would come to dominate nineteenth-century medicine.

Table 3.1. GRI admission registers and case notes samples, 1794 – 1820

	Admission Registers[a]	Casenotes[b]	
Diagnosis Groups		Complete Sample	Amended Sample
TB-Related	33	25	25
Non-TB Pulmonary	68	30	13
Other	681	159	13
TOTAL	782	214	51

[a] Sampled years include 1795 and 1803 only.
[b] Sampled years include 1794, 1795, 1798, 1801, 1807, 1811, 1812, 1814 and 1819.

DISEASE CONCEPTS

In the germinal account of Dorothy and Roy Porter (1989), eighteenth-century disease concepts were not specific, but rather "mark[ed] a general imbalance of the humours" (163). Attributed to the second-century work of Galen, this humoral model explains disease through the disruption of the natural balance among blood, phlegm, and

yellow and black bile within the body, or between the body and hot, cold, wet or dry aspects of the external environment. In contrast, however, more recent research has dated to the seventeenth century or earlier a crisis in the explanatory power of Galenism (Pender 2006) and the beginning of the transition to Paracelsian models (Mortimer 2009; Wallis 2012). These later models are based in Paracelsus' sixteenth-century conception of disease resulting from malign substances introduced to the body from without. Thus, while Porter and Porter (1989) characterize eighteenth-century disease concepts as holistic and non-specific, the more recent work of Pender (2006), Mortimer (2009), and Wallis (2012) suggests that by the eighteenth century, medical practitioners were rejecting such concepts in favor of a construct of disease resulting from malign and infinitely variable external influences.

It follows from Galenic precepts that adherence to humoral disease concepts by either patients or physicians implies the enactment in practice of illness and/or disease constructs that are both *holistic* and *non-specific*. Of interest to such a discussion are two influential late eighteenth-century medical systems originating in Scotland. While not strictly Galenic, the systems of both William Cullen (1778, 1789, 1797) and John Brown (1795), his one-time protégé at Edinburgh, drew upon the core idea that disease originates with systemic imbalance.

Cullen's conception, which his students at Edinburgh received enthusiastically, hewed more closely than Brown's to older Galenic models. In his system, disease could be traced to bodily disorder — "usually excess, usually of the passions," notes Stott (1987, 126), with the weary tone of one accustomed to reading early modern medical treatises. However, Cullen replaced the classical humors with three chemically inspired systems of the animal economy — the simple solids, the animal fluids, and the nervous system (Cullen 1797; Stott 1987). Each of these systems could become deranged individually or in combination,

resulting in disease. In Cullen's conception, such derangements were systemic, although they could produce localized effects. As a result, Cullen's medical system, like Galenic models, remained at heart essentialist, with disease an expression of the state of the body as a whole. In practice, I expect both Galenic medical models and Cullen's system to result in constructs of disease as systemic.

Brunonian medicine, while differing from Cullen's system in the ultimate sources of disease, similarly viewed disease as systemic. Brown traced all morbid conditions to the relationship between an individual's state of excitement (i.e., the degree of action in various bodily systems, driven by the nerves) and excitability (i.e., the degree to which bodily systems are capable of becoming excited) (Brown 1795), thus formulating disease as the outgrowth of holistic influences. While local influences were capable of causing local disorders, these could lead to generalized disease; disorders remaining localized Brown did not consider the province of physicians (Brown 1795, 1–2). The Scottish medical establishment generally rejected Brown's conception; indeed, when Brown sought the position of theoretical chair of medicine at Edinburgh, his former mentor Cullen reportedly commented in Scotland's "vulgar dialect" upon his application, "Why, sure, this can never be our Jock!" (Beddoes 1795a, lvii). As a result, I expect the influence of Brunonianism on practice at GRI to be minimal, despite the popularity this system attained among both practitioners and laypeople in England, the United States, and continental Europe, particularly Germany (Lawrence 1988). Nonetheless, under Brown's system, as in Cullen's, constructs of disease I expect to be largely holistic.

Despite the differences in ultimate causation between the medical systems developed by Cullen and Brown, their shared systemic concepts of disease caused both systems to rely

heavily upon behavioral influences on health. Both Brown and Cullen emphasize personal responsibility in the maintenance of health, in the case of Cullen through active maintenance of healthy qualities in the solids, fluids and nervous system (Stott 1987, 131), and in the case of Brown through careful management of excitability and one's exposure to exciting or sedating influences (Beddoes 1795b, cxxix). The result of this emphasis on personal responsibility is endorsement of the virtue believed inherent in the "Stoic self-command" embraced by both Cullen and his compatriot Adam Smith (Stott 1987, 126), and by reflection the immorality of the sorts of behavioral excesses leading to disease. As a result, I expect that moral causes for disease were retained in the medical models of both Cullen and Brown.

Essentialist concepts of disease and the importance of personal responsibility in resisting bodily disruption intersected with the essentialist concepts of gender arguably cemented during the eighteenth century (Porter and Porter 1989, 176) to suggest an additional role for gender in patient and physician concepts of disease. Humoral theory associated women with phlegm and black bile, men with blood and yellow bile (Fletcher 1995, figure 12). Women, as the "weaker vessel", were furthermore "governed by their reproductive system … weaker, supersensitive, highly nervous by nature" (Porter and Porter 1989, 172, 176). These suggest that under Galenic models, medical practitioners would have perceived women as more susceptible to excesses of phlegm and black bile. Additionally, essentialist concepts of disease — such as Brunonianism — combined with essentialist concepts of gender indicate that practitioners would have perceived women as more susceptible to bodily disorder than men.

Gender was also a powerful influence on disease in Cullen's view, which held that one's sensibility, or susceptibility to negative influences, dictated the degree to which the

nervous system might be disrupted by such influences. He argued that timidity and fear resulted from debility, or a reduction in bodily vigor, and thus were harbingers of bodily disorder. Refined women, sheltered by men, developed greater debility and sensibility than lower-class women, whose exposure to hardship and lack of male indulgence was strengthening, though perhaps to a harmfully extreme degree (Stott 1987, 137–38). This indicates that, in contrast to humoral and other essentialist models of health and disease, Cullen's medical system specifically suggested that any gender differences in health would be reduced among the working class.

With respect to diagnosis, existing research suggests that throughout the late Georgian/Regency period examined here, medical practitioners retained concepts of disease that were at least somewhat non-specific. For Rosenberg (1997), the perception of disease as a "discrete entity — or even a well-defined physiological process with a peculiar natural history — was not generally accepted" until the mid-nineteenth century (29). To determine whether this was the case, one must first consider that there are two dimensions along which diseases might be considered discrete. The first is specific diagnosis, that is, a discrete set of signs and symptoms that defines the clinical presentation of disease. The second is specific etiology, that is, disease that is understood to result from a unique cause or process. As essentialist theories of medicine traced all disorders to a universal cause — bodily imbalance — diseases in this time period were indeed unlikely to be discrete with respect to etiology. However, that diseases were uniformly ill- defined with respect to diagnosis seems unlikely given, for example, William Buchan's description of symptoms defining consumptions in his popular lay medical guide, *Domestic Medicine*, in its 1790 eleventh edition:

> This disease generally begins with a dry cough, which often continues for some months. If a disposition to vomit after eating be excited by it, there is still greater

> reason to fear an approaching consumption. The patient complains of a more than usual degree of heat, a pain and oppression of the breast, especially after motion; his spittle is of a saltish taste, and sometimes mixed with blood. … There is generally a quick, soft, small pulse; though sometimes the pulse is pretty full, and rather hard. … [In more advanced disease,] the patient begins to spit a greenish, white, or bloody matter. His body is extenuated by the hectic fever, and colliquative sweats, which mutually succeed one another, viz. The one towards night, and the other in the morning. A looseness [i.e., of the bowels] and an excessive discharge of urine, are often troublesome symptoms at this time, and greatly weaken the patient. There is a burning heat in the palms of the hands, and the face generally flushes after eating[.] (176–77)

While some of these symptoms are more specific than others — one is left with the impression that any abnormality of the pulse could be considered indicative of consumption — taken together, they represent an impressively cogent construct of the disease. An expectation of ill-specified diagnoses also contrasts sharply with the ideas of Cullen, who in his medical lectures argued explicitly for the specific classification of diseases along the lines of Linnaeus (Cullen 1797, 4–6).

Conversely, for Brown relatively large subsets of diagnoses could result from similar levels of excitement and excitability: apoplexy, palsy, plague, confluent smallpox, contagious dysentery, and phthisis all resulted from the combination of low excitability and high excitement (Lynch 1795). Because treatment the levels of excitement and excitability rather than the specific diagnosis dictated treatment (Lawrence 1988, 6), however, it follows that the Brunonians considered the exact identification of disease unimportant. As a result, I expect adherents to Cullen's medical system to favor specific diagnoses; those of Galenism and Brunonianism, non-specific diagnoses.

Table 3.2 summarizes the expectations for enactment of medical constructs variously influenced by Galen, Paracelsus, Cullen and Brown. While Paracelsian models are highly distinctive from the other three owing to their individual, externalized construct of disease,

the models of Galen, Cullen and Brown are more subtly differentiated along axes of disease etiology, specificity of diagnosis, and expected gender differences. These distinctions among the medical models I expected to be in evidence at GRI during the late Georgian/Regency period informed my qualitative and quantitative analyses comparing TB-related disorders to non-TB pulmonary diseases and non-TB, non-pulmonary disorders. I shall discuss the results of these analyses with respect to each axis in turn: etiology, essentialism, moral influences, gender influence, and finally specificity of diagnoses.

Table 3.2. Expectations under the models of Galen, Paracelsus, Cullen, and Brown

Aspects of Disease	Medical System			
	Galen	Paracelsus	Cullen	Brown
Etiology	Humoral imbalance	External source	Systemic imbalance	Excitement, excitability
Essentialism	Yes	No	Yes	Yes
Moral Influence	Yes	No	Yes	Yes
Gender Influence	Yes	Not necessarily	Reduced for working class	Yes
Diagnosis	Nonspecific	Individual	Specific	Nonspecific
Treatment	Balancing, Botanical, Bleeding	Like, Mineral, No bleeding	Individual	Nonspecific

ESSENTIALISM

In this late Georgian/Regency sample, there are few explicit references to the systemic or localized nature of disease, although the expression of one patient is significant. Alexander K., an 1819 patient diagnosed with chronic peritonitis and pleuritis, described his health history as follows: "The cough [is] to a slight degree of long continuance. Says that

two months ago, was bled for inflammation of the chest and that the abdomen swelled about that time. His health, however, he says had been broken before, first in consequence of a fever four months ago, shortly after which [he] had erysipelas first of one leg and then of the other." (HB14/5/3: 105–6) Alexander K. thus defined as sequelae to the initial fever multiple episodes of ill health occurring over the four months previous to his admission. His conception of health — and we can be confident that it is his conception rather than the clinician's owing to the designation "he says" — was one of a monolithic entity which could be systemically destroyed, initiating a months-long series of localized disorders in disparate regions of the body, all attributable to the initial insult of the fever. While this single instance is insufficient to draw definitive conclusions, for Alexander K. at least, health and illness constituted holistic entities. This indicates the presence, if not the frequency, of essentialist concepts of health and disease during this period at GRI.

MORAL INFLUENCES

While both GRI patients and clinicians exhibited an understanding that behavior contributed to disease in the period 1794 – 1820, Glasgow practitioners rarely attributed disease to immorality. Behavioral links in cases of TB-related disorders related only to improvidence, as in the case of Jonathan B.'s fatal phthisis, which in August 1819 medical staff traced to his having six weeks previously fallen "asleep on the Green, and a heavy shower coming on, he found himself wet through when he awoke." (HB14/5/3, 47) Indeed, only in two cases in the amended sample did immorality play even a minor role. In the first, the clerk initially recorded 1798 syphilis patient Agnes S. as having "denie[d] the primary symptoms of syphilis," although the clerk later struck out her denial, altering the entry to

"own[ed] the primary symptoms of syphilis." (HB14/5/13, 172) This change evidently explains the alteration in her diagnosis from the non-venereal disease sibbens in the admission register to syphilis in the case notes journal, but appears to have affected her treatment and conceptions of her disease no further.

In the second case of recorded immorality, the clerk noted just before the dismissal of Catherine M., admitted in 1814 suffering amenorrheic symptoms, "Though she would not acknowledge her pregnancy, the certainty of it has now been discovered; and she is dismissed, as an improper patient." (HH67/2/2, 112) While it is unclear whether Catherine M. deliberately misled hospital staff as to the nature of her complaint, the clinician certainly believed her to be doing so; however, medical staff related her perceived dishonesty only to the question of her continuation at the hospital, and not to her broader health status. Thus, moral influences on health status were not evident at this early period, suggesting affinity of clinician constructs of disease with Paracelsian models. Interestingly, the moral or immoral behavior of patients would become an evident concern of clinicians only later in time — an observation I will discuss in chapter six — suggesting that moral influences on health became more rather than less salient coincident with Victorian preoccupations with the behavior of the working class.

GENDER INFLUENCES

Gender differences in the etiology, progression or diagnosis of TB-related disorders — or indeed any disorders — were similarly not evident for this earliest period at GRI. This supports the models of Paracelsus or Cullen, and may thus be informative regarding concepts of gender held by clinicians with respect to their working-class patients, and/or those held by the patients themselves. Cullen's view was that working-class women differed

less from men with respect to risk of disease than their higher-class counterparts; however, it is poorly understood how widely held this view was at the turn of the nineteenth century. There is reason to believe that later gender constructs with respect to class also saw less distinction between working-class men and women than between men and women of the higher classes. Boddice (2011) reports that mid- to late nineteenth-century social thought perceived decreased sexual differences among "uncivilized" groups, including the lower classes (334), an idea that might have extended to reduced differences in perceived risk of disease. For this late Georgian/Regency period, though, there is insufficient evidence to attribute the apparent absence of perceived influences of gender on health to a broader social construct that differentiated less among genders for the working class than for the upper classes.

ETIOLOGY

In this period's sample, patients rather than medical staff generally contributed the cause of their current disease across diagnoses. In the amended sample, patients traced their current disorders to a wide range of causes (appendix C). These included previous injuries and disorders: an 1819 diarrhea patient, Jonathan K., reported having previously contracted dysentery while serving with the British army outside New Orleans, presumably in the War of 1812 (HB14/5/3, 195). A number of phthisis patients linked their disorder to prior episodes of pulmonary disease, such as the 1819 incipient phthisis patient Ebenezer A., who began the history of his disorder by stating that he "three months ago was affected w't [i.e., with] cough, which lasted six weeks" (HB14/5/3, 14).

The largest number of TB-related patients attributing cause to their disorder invoked elemental exposure. For example, William D., who died of phthisis after two months'

hospitalization in 1795, "attributed [his complaints] to fatigue and exposure to cold" (HB14/5/1, n.p.). Similarly, James H., a phthisis patient in the same year, "attributed [his complaints] to exposure to cold after a fever" (HB14/5/1, 141). In quantitative analysis, patient history significantly distinguished TB-related cases from neither non-TB pulmonary diagnoses nor other disorders (table 3.3, line 7); however, at $p = 0.0538$ for TB-related vs. non-TB pulmonary and $p = 0.0829$ for TB-related vs. other, both of these tests approached significance with $p < 0.10$.

Table 3.3. P-values for Fisher's exact tests of differences in clinical indicators, 1794 – 1820

Signs and Symptoms	TB-Related vs. Non-TB Pulmonary	TB-Related vs. Other
1. Appearance and Comportment	0.1176	0.0425*
2. Circulatory	0.4034	0.0233*
3. Dermatological	0.3432	0.0018**
4. Digestive	0.1781	0.8376
5. General	0.2627	0.5442
6. Genitourinary	0.7143	1
7. History	0.0538†	0.0829†
8. Mouth and Throat	0.9422	0.702
9. Pain	0.835	0.0042**
10. Respiratory	0.0053**	0.0550†
11. Miscellaneous	1	0.0025**

Note: See appendix C for a complete list of signs and symptoms comprising each category.
† $p < 0.10$
* $p < 0.05$
** $p < 0.01$

There is little evidence for concepts of disease resulting from external contamination, with the notable exception of syphilis and the related disorder sibbens. Both patients and medical staff knew syphilis to be sexually transmitted, with the result that neither seemed to feel it necessary to attribute cause to such disorders except to report any previous experience with the disease. The lesions of sibbens, a closely-related non-venereal disease (Morton

1967), strongly resembled those of syphilis, and medical staff generally assumed such lesions to result from venereal syphilis until demonstrated otherwise. As a result, any attribution of sibbens was generally limited to statements by the patient indicating an absence of syphilitic history (e.g., William M.: HB14/5/1 1795, 87). As a result, Paracelsian models were not indicated for diagnoses other than these closely associated diseases.

The more popular notion patients exhibited in this period is that disease, perhaps TB- related disease in particular, results from adverse environmental exposures. This does link patient concepts of disease to systemic perturbation. In Galenic models, exposure to excessively wet, dry, cold or hot conditions could affect humoral balance positively or negatively, causing disease where such conditions exacerbated an existing tendency of the patient toward excess of the promoted humor. In Cullen's system of medicine, extreme environmental conditions could similarly perturb systemic balance, with excessive heat or cold affecting the density of the fluids and solids as well as the body's "generating power" (Stott 1987, 126). Similarly, for Brown heat and cold were important sources of stimulus or sedation (Brown 1795). Thus, the environmental etiology to which patients most commonly ascribed TB-related disease during this period supports an essentialist, non-Paracelsian model of disease, albeit not conclusively.

SPECIFICITY OF DIAGNOSIS

As expected from the emphasis on specific diagnosis in such works as Buchan (1790) and Cullen (1797), quantitative analysis demonstrates that TB-related diagnoses are distinguished from both non-TB pulmonary and non-TB, non-pulmonary disorders in a number of symptom categories (table 3.3). The clinical indicators recorded for cases of TB-related disorders differed significantly ($p < 0.05$) from those of non-TB, non-pulmonary

disorders in the categories of patient appearance and comportment, circulatory symptoms, dermatological symptoms, and reported pain (see appendix C for a complete listing of symptoms and their frequencies); differences approached significance ($p < 0.10$) for two additional categories, patient history (discussed above) and respiratory symptoms. The insignificant difference in respiratory symptoms between TB-related and other disorders may appear surprising, and suggestive of an indistinct clinical construct of TB-related disorders; however, this was likely the result of the paucity of respiratory symptoms recorded for non-TB, non-pulmonary diagnoses. Only seven respiratory signs and symptoms were recorded across 13 non-TB, non-pulmonary diagnoses, or an average of 0.54 respiratory indicators per case, while 128 respiratory symptoms were recorded across 25 TB-related diagnoses, or 5.12 respiratory indicators per case (appendix C) — a nearly tenfold difference.

The significant differences between TB-related and non-TB, non-pulmonary disorders in the clinical indicator categories of patient appearance and comportment, dermatological symptoms, and reported pain are likely attributable to a suite of clinical indicators long associated with consumption. Salient differences in patient appearance and comportment included debility and emaciation; among dermatological symptoms, perspiration, particularly at night; and reported pain localized in the chest and thorax and exacerbated by cough. Also contributing to the significant difference in recorded dermatological symptoms was likely the more common occurrence of syphilis and sibbens nodes and ulcers in cases of non-TB, non-pulmonary disorders, which of course include these diseases. With respect to the circulatory symptoms differing between these two categories of disease, significance was likely driven by pulse characteristics, with pulses below ninety-four beats per minute more commonly recorded among non-TB, non-pulmonary

disorders and those greater than ninety-five beats per minute and unusual in quality — feeble, weak, hard or sharp — more commonly represented in cases of TB-related diseases.

The clinical indicators significantly distinguishing between TB-related diagnoses and other pulmonary disorders occurred only among respiratory symptoms, with differences also approaching significance for patient history (discussed above). Among recorded respiratory symptoms, the difference between TB-related and non-TB pulmonary disorders appears to be driven by characteristics of expectorated matter, with TB-related disorders defined by abundant expectoration of yellow matter, pus, and/or blood and non-TB pulmonary disorders characterized by white expectoration. This contrasts with twenty-first-century interpretation of sputum color, for which research indicates yellow, purulent matter is associated with bacterial infection (Miravitlles et al. 2012; Stockley et al. 2000), but not TB specifically. In my sample, there were no cases in which yellow and/or purulent expectoration were recorded that did not bear a TB-related diagnosis, and only one of fourteen recorded instances of bloody expectoration and/or hemoptysis occurred in a case not diagnosed as TB-related. The frequency and timing of coughing may have also distinguished between TB-related and other pulmonary disorders, with TB-related cases more commonly associated with a cough that disturbs sleep or a cough of unspecified character, although this last is somewhat unhelpful to developing our understanding of the clinical construct of TB's historical analogues during this period.

Overall, several classic symptoms of TB-related disorders — debility, emaciation, night sweats, chest pain, and expectoration of pus or blood — appear to have driven significant differences between TB-related and non-TB disorders, while others — particularly coughing — were not particularly salient in defining TB-related disorders in this sample. While non-TB, non-pulmonary disorders were distinguished from TB's historical

analogues for more categories of clinical indicator than were other pulmonary disorders, sharp distinctions were nonetheless apparent between TB-related and other pulmonary disorders in the frequency with which purulent, yellow and/or bloody expectoration were recorded. In this sample, TB-related disorders were diagnostically distinct from both non-TB pulmonary and non-TB, non-pulmonary diseases even at this early stage, suggesting the influence of Cullen's emphasis on pathology and specificity of diagnosis and/or Paracelsian concepts of individualized disorders. It is furthermore clear that at GRI, clinical constructs of consumption and phthisis had coalesced around a number of characteristic clinical indicators by the close of the eighteenth century.

CARE AND TREATMENT

The various disease concepts I have suggested to have been in active use at GRI during the period 1794 – 1820 — the models attributed to Galen, Paracelsus, Cullen and Brown — had implications for treatment as well. In Galenic medicine, treatment was for the most part complementary, intended to rebalance the humors by eliminating excesses and/or bolstering deficiencies (Porter and Porter 1989, 163). For Paracelsus, like was countered with like: the most effective treatments shared elemental principles with the disease. Under Cullen's medical system, as might be expected from his emphasis on the specific diagnosis of disease, treatment was highly individualized. It was necessary to Cullen (1789) to consider the state of the patient, as the action of drugs may differ idiosyncratically: "the general operation of medicines may be variously modified, according to the different states and circumstances in which the human body may be upon different occasions." (56) Cullen also reminded readers of his *Treatise of the Materia Medica* that drugs had holistic effects: "with

respect to the operation of medicines, this in general is to be observed, that as there seems to be a possible communication of motion from every part of the nervous system to every other part of it; so medicines, though applied to one small part of the body only, do often, in consequence of the communication mentioned, show their effects in many other parts of the body." (59) In stark contrast to the individualized treatment advocated by Cullen, Brunonian medicine found the idea of idiosyncratic treatment almost ludicrous; if all disease resulted from excitement or sedation, so all treatment should be simply the reverse (Lawrence 1988, 6).

Further differences in treatment regimens segregated Galenic and Paracelsian models. Galenic and Paracelsian models differed sharply from one another on the matter of therapeutic bloodletting. Humoral medicine had for centuries embraced bloodletting to counteract a plethora or to purge stale blood (Porter and Porter 1989, 170). In contrast, the Paracelsian worldview held that blood was the body's life force, and as a result, discouraged bloodletting as dangerously depleting. Galenic treatments emphasized not only bloodletting, but also botanical drugs, whereas Paracelsian treatments emphasized metal and mineral compounds, particularly mercury and antimony (Porter and Porter 1989, 158–59). Neither Cullen's nor Brown's medical models were so circumscribed as Galenism and Paracelsianism respecting treatment, with both utilizing the full range of drugs available in the late eighteenth century. Brunonianism, Porter and Porter (1989) report, was particularly optimistic about the applications of opium compounds (150). In sum, I expect adherents of Galenic models to have utilized complementary therapies including bloodletting and botanical compounds, in contrast to Paracelsian practitioners, whom I expect to have rejected bloodletting and instead emphasized metal and mineral treatments. Practitioners

under either Cullen's or Brown's medical models are expected to make use of the range of treatments available, but while Cullen's model prioritized treatment tailored to the individual, Brunonianism applied the same treatments across disparate diseases sharing a common cause.

Table 3.2 summarizes the expectations for treatment under medical models variously influenced by Galen, Paracelsus, Cullen and Brown, while appendix H details the treatments applied by GRI medical staff in cases of TB-related disorders, non-TB pulmonary disorders, and non-TB, non-pulmonary disorders during this period. Immediately apparent was the frequency with which bloodletting techniques were applied; cupping and/or venesection were recorded in 21 of 51 cases across disorders. In two 1819 pulmonary cases, the bloodletting can only be described as enthusiastic: catarrh patient Jonathan C. had 81 ounces (2.4L) of blood removed over the course of five days (HB14/5/3, 185, 190), while 89 ounces (2.6L) were removed from pneumonia patient James B. in the same time span (HB14/5/3, 159, 165, 203). It is clear that the Galenic fervor for bloodletting was maintained at GRI into the early nineteenth century.

In contrast to expectations for either Galenic or Paracelsian models, GRI practitioners made use of the full range of drugs available at the turn of the nineteenth century (appendix H). Galenic botanicals such as compounds of digitalis and squill — a flowering plant sharing a taxonomic subfamily with hyacinths — were used alongside newer imports such as ipecac. Practitioners also prescribed metal and mineral compounds such as mercury and antimony preparations. Opiates were in common use, as were blisters and other applications. Laxatives were virtually inescapable — each patient in the amended sample received an average of 1.2 laxatives during his or her hospital stay, reflecting an emphasis on maintaining animal functions (cf. Cullen 1797) and/or purging waste or excess (cf. humoral

theory). The treatments in use thus did not adhere to a single medical model, but rather borrowed from both Galenic and Paracelsian treatments in the manner expected for Cullen's model or Brunonianism.

Quantitative analysis, the results of which are summarized in table 3.4, reveals that the treatments in use at GRI for the period 1794 – 1820 were largely nonspecific. Medical staff prescribed no category of treatment with significantly different ($p < 0.05$) frequency in cases of TB-related disorders compared to non-TB, non-pulmonary cases. Only one category for this comparison approached significance ($p < 0.10$), that of the commonly prescribed botanicals assafoetida — a flowering plant in the carrot and parsley family — digitalis and squill. This is most likely the result of prescribing patterns for the lattermost remedy, as practitioners recognized squill to have expectorant properties (e.g., Cullen 1789, 211) and indeed is still available in cough preparations today (EMC+ Medicine Guides 2011).

Table 3.4. P-values for Fisher's exact tests of differences in treatments, 1794 – 1820

Treatments	TB-Related vs. Non-TB Pulmonary	TB-Related vs. Other
1. Applications	0.885	0.357
2. Bleeding	0.6305	1
3. Diets	1	0.7333
4. Drugs, Other	0.7899	0.0857†
5. Inhalations	0.0357*	1
6. Laxatives	0.2168	0.681
7. Mercurials	0.4444	0.1009
8. Mixtures	0.8524	0.288
9. Opiates	1	0.5539
10. Vomits	1	1
11. Washes	1	1
12. Other	0.8797	0.9223

Note: See appendix H for a complete list of signs and symptoms comprising each category.
† $p < 0.10$
* $p < 0.05$

In comparison with treatments clinicians prescribed for patients with non-TB pulmonary disorders, the treatments applied in cases of TB-related diseases differed significantly in only one category, inhalations. The significance of this difference was driven by the inclusion of substances other than water in inhalations prescribed to patients with TB-related diagnoses. These inclusions ran the gamut of drugs in use during the late eighteenth and early nineteenth century, including antimony, the botanicals gentian and senna, vinegar, and chemicals such as ethers (aether nitrosis spiritus, aether sulphericus), sodae carbonis and hydrocarbon. These cover categories as disparate as tonics (gentian), antispasmodics (ethers, hydrocarbon), and cathartics (senna) (Cullen 1789, 193–215). No additional categories approached significance for TB-related disorders vs. non-TB pulmonary disorders.

The dramatic range of inclusions in inhalations prescribed for individuals diagnosed with TB-related disorders suggests two alternate explanations. In the first, medical staff employed the individualized treatment advocated by Cullen, prescribing differing inhalations in response to differences in patient constitution, symptoms, and/or disease stage that are not clearly indicated by the recorded case notes. In the second, the variety of inhalations represented perhaps somewhat desperate experimental attempts by medical staff to identify an effective treatment for TB-related disorders. This period predates by decades the dramatic increase in medical books about consumption that Byrne (2011) dates to the 1840s (190), perhaps indicating — if this second explanation is correct — an early expansion of medical interest in the curability of TB-related diseases.

The impetus for such experimentation is clear from table 3.5, showing the outcomes recorded for TB-related, non-TB pulmonary, and other disorders in the admission register

sample for this period. The outcomes recorded by medical staff in cases of TB-related disease differ highly significantly ($p < 0.001$) from the outcomes of both other pulmonary and non-pulmonary cases. The most common outcome in TB-related cases was dismissal by the patient's desire, indicating that medical staff acquiesced to the patient's request to leave the hospital although little change was effected in the patient's condition. In contrast, the most common outcome in cases of both non-TB pulmonary disorders and non-TB, non-pulmonary diseases was cure, illustrating the stark ineffectiveness of medical treatments against TB-related disorders during the late Georgian/Regency period.

Table 3.5. Outcomes, 1794 – 1820

Outcomes	TB-Related	Non-TB Pulmonary	Other
Advice	3	0	20
By Desire	15	2	76
Cured	5	56	463
Died	8	4	38
Improper	0	0	3
Irregular	0	1	22
Relieved	1	5	54
[Blank]	1	0	5
P-Values[a]		1.653×10^{-12}***	2.399×10^{-10}***

[a] P-values resulted from Fisher's exact tests of difference in TB-related vs. non-TB pulmonary and TB-related vs. other outcomes.
*** $p < 0.001$

The perception of hospital treatment at the turn of the nineteenth century afforded by this GRI sample is overwhelmingly resistant to categorization. The drugs and treatments used were neither uniformly Galenic nor Paracelsian, while the overall lack of patterning in their prescription across disorders could be indicative of either individualized treatment following the Cullen system or generalized treatment along Brunonian disease categories,

which differ from the diagnostic categories I employ here. As I discussed above, the general rejection of Brunonianism in Scotland renders the latter unlikely; additionally, Brunonianism would predict some uniformity in prescriptions within TB-related disorders. Instead, however, a broad array of drugs and treatments were brought to bear, including the prescription of multiple classes of drugs in the form of inhalations. The emergent picture from the care and treatment of TB's historical analogues during this period is one of transition, in which trusted older remedies were retained and newer schemes of treatment incorporated into medical practice, indicative of both the inadequacy of older medical models to effectively treat these diseases and optimism that a remedy might yet be identified.

THE ROLE OF PHYSICIANS

The guiding principle of medical consultations in the eighteenth century was that disease was externalized, or identifiable through externally detectable characteristics alone (Porter and Porter 1989). In Britain and beyond, diagnosis took as its substance the patient's narrative regarding his or her disease as well as outward signs and symptoms. The physician's unique skill was in keen questioning of the patient for adequate detail and perspicacious assimilation of the available data into a cogent hypothesis regarding the patient's disorder and consequently the course of treatment most likely to be effective. As a result, Porter and Porter (1989: 74–75) argue, during the eighteenth century patient history was primary during medical consultations, with examination of the patient secondary and dominated by indirect observations rather than later hands-on techniques such as percussion and auscultation.

Both of these expectations were borne out with respect to medical consultations at GRI in the late Georgian/Regency period considered here. Across disorders, patient history

took pride of place in records of the intake consultation, with little distinction between the patients' and physicians' observations on the disorder in question. However, while Porter and Porter (1989) suggest that the eighteenth century was marked by medical practitioners' acquiescence to patients' feelings and their conceptions of their own disorders (137), this assessment is also contradicted by Porter and Porter's inclusion of Buchan's 1796 admonishment demanding full disclosure from patients (140) and the contentious, bullying approach to patients successfully adopted by John Radcliffe at the turn of the eighteenth century (142). Indeed, it was clear from rhetorical cues that in the late eighteenth/early nineteenth-century setting observed here, practitioners did not uniformly accept patient reports without question. However, evidence of out-and-out contention is largely absent, save for the two cases discussed as examples of reactions to immoral behavior above.

Medical examinations furthermore were largely superficial in this setting. Physical examination was limited to taking the pulse and visual observation of the tongue and external appearance, as well as closer examination of any areas of concern indicated by the patient. Porter and Porter (1989) attribute this to resistance by physicians of the physicality associated with surgery, as well as confidence in their special ability to read the correct diagnosis from outward signs and patient narrative (74–75). A similar fetishization of visual acuity is indicated in Porter's (1985) discussion of eighteenth-century physiognomy, in that both medicine and physiognomy are argued to privilege unaugmented vision, with interpretation relying on the observer's perspicuity. In both physiognomy and medicine, the truth is plain on the body's surface for those trained to see it: those able to see the externalized truth have no need to look beyond the surface. The emphasis on patient

observation rather than examination is thus a marker of discernment for physicians, setting them apart from the untrained and the surgeons.

Indeed, Porter (1993) points out that the emphasis of physicians upon patient history and superficial diagnosis was rooted in the idea that the physician's proper instrument was the head, in contrast to the surgeon's hand. Porter attributes this in part to an increasing squeamishness surrounding matters of the flesh in early modern Britain, causing touch to become morally charged and leading to fears of impropriety in practitioners' intimate contact with patients (186–87). At the same time, as Christopher Lawrence (1998) describes, physicians drew upon concepts of gentility established in the late seventeenth century that emphasized learning, personal bearing, and freedom from manual labor (164) to cast their work as respectable and distinct from the taint of butchery attached to surgery (183). The éclat garnered by physicians was mental rather than manual skill — "judgment," an acute perceptivity regarding others' bodies providing "the ability to reason out the complexities of a case and tailor therapy to individual needs, rather than applying … a [universal] remedy" (166). The physician at the turn of the nineteenth century thus distinguished himself from the surgeon and apothecary by his ability to diagnose and treat patients through "verbal and visual, not physical examination." (Porter 1993, 184)

This parallels Ludmilla Jordanova's (1996) argument, in which the turn of the nineteenth century witnessed the forging of physician identity "around a middle-class ideology" (104) privileging mental over physical labor. The avoidance of imagery invoking the physicality of medicine — tools, anatomical specimens — that Jordanova (1996) traces in medical portraiture for the period 1780 – 1820 closely parallels the avoidance of physicality in medical practice described by Porter and Porter (1989), Porter (1993), and

Lawrence (1998) as well as that documented in the GRI sample addressed here. Together, these observations create a strong sense of late Georgian/Regency physicians aligning themselves with the nascent middle class through rejection of physical interactions with patients.

Although convincing, Porter and Porter's (1989) discussion of physicians' class-based resistance to physical examination fails to take into account the state of pathological anatomy at the turn of the nineteenth century. As anatomical specimens were generally wanting in the years prior to the 1832 Anatomy Act, it is likely that physical examination would not have added significantly to physicians' external examinations. Without the link between externally observable signs and symptoms and internal pathology forged by the increased access to cadavers following the Anatomy Act, techniques such as percussion and auscultation could not be used to "see" within the body in the same way they would be later in time. The professional and class concerns of physicians during this period may have coincided with — and indeed possibly arisen in response to — a plateau in the acquisition of medical knowledge that left practitioners bereft of the diagnostic and treatment acumen necessary to effectively address diseases such as phthisis and consumption.

In light of this, the postmortem dissections performed and advocated in Scotland during the late eighteenth and early nineteenth century are indicative of the growing medical focus on pathological anatomy. Cullen was an early advocate for the importance of postmortem dissection in understanding disease, stating in his clinical lectures that after diagnosis, "we shall next endeavor to find what is the particular state of the body under [the diagnosed disease]" and arguing that "the dissection of morbid bodies … is the surest method of judging of internal diseases" (Cullen 1797, 6). The physicians at GRI clearly agreed, as six of the eleven individuals in my amended sample who died in hospital were

examined postmortem. That five of the six dissected individuals had been diagnosed with TB-related disorders evokes a sense of the urgency with which medical staff sought greater understanding of these disorders in particular.

CONCLUSIONS

In sum, medical practice at GRI during the period 1794 – 1820 was in flux, wholly committed to neither the new medical models of Cullen and Brown nor the older models attributed to Galen and Paracelsus. The transition characterizing this period was apparent not only in the diagnosis of TB-related disorders, but also in their treatment and in practitioner-patient interactions. In practice, GRI physicians adhered to class-based proscriptions against physical examination supported by their self-conception as uniquely learned and uniquely perspicuous. At the same time, however, they seem to have actively sought new approaches to the treatment of TB-related disorders as well as pursued new knowledge about these diseases in the least external manner possible: the postmortem dissection of phthisis and consumption victims. This new interest in and reliance upon pathological anatomy would develop more fully in the years addressed in the following three chapters.

CHAPTER 4

THE RISE OF PATHOLOGY, 1821 – 1840

"In the History of Medicine," wrote an anonymous author in the July 1822 issue of the *Edinburgh Medical and Surgical Journal* (*EMSJ*), "the present period will be characterized by the progress made in the study of Morbid Anatomy, and its beneficial applications to Nosography and Practice." (447) This prescient statement heralded the British popularization of R. T. H. Laënnec's treatise on the stethoscope (1819) via John Forbes's English translation (Laënnec 1821). Laënnec's (1819) treatise on auscultation, along with earlier work on thoracic percussion by Auenbrugger (1761), linked heart and lung sounds observed using his new method to internal pathology identified during postmortem dissection, arguing for the utility of pathology to diagnosis and introducing an effective means of assessing internal pathology from without.

The growing importance of pathological anatomy to diagnosis and treatment acknowledged by the 1822 *EMSJ* reviewer signaled a transition to concepts of disease in which disorders were increasingly generalizable and internalized. This arose from medical men's observation of anatomical lesions in cadavers, more broadly available for dissection in France beginning in the late eighteenth century and in the UK from the passage of the 1832 Anatomy Act (2 and 3 William IV c. 75), which I discuss in detail below. Such observations favored a redefinition of diseases, including tubercular disease, rooted in pathology generalizable across cases rather than that arising from perturbations to health acting upon individual constitutions. This emphasis on anatomical lesions furthermore turned the focus of practitioners internally in detecting disease, making the practice of diagnosis one of

seeking external signs of internal pathology rather than divining the patient's history and pattern of constitutional imbalance (e.g., Porter 1993). The former required close physical — rather than discursive — examination of patients using hands-on techniques and instruments like the stethoscope.

In the pages that follow, I discuss the adoption of physical examination — specifically, percussion and auscultation — at GRI through my analysis of 2312 admissions and the case notes of 88 individuals (table 4.1). I address the contribution of the rapidly expanding knowledge of pathological anatomy afforded by the 1832 Anatomy Act to the standardized use of percussion and auscultation at GRI, and the increasingly internalized medical conception of TB and its analogues that resulted. This new construct was characterized not by replacement of older disease models, but by the addition of internal signs to the classic, external signs and symptoms of these diseases familiar from earlier practice. At the same time, I describe the practitioner and patient attitudes that led in the period 1821 through 1840 to a deterioration in practitioner-patient relationships, with earlier, more collaborative interactions becoming strained. In the setting of charitable institutions like GRI, physical examination corresponding to pathological disease concepts revealed the living bodies of the working classes in ways analogous to the broad medical access to the corpses of the poor for dissection, contributing to a new practitioner-patient relationship marked by inequality, exploitation, and resistance.

Table 4.1. GRI admission registers and case notes samples, 1821 – 1840

Diagnosis Groups	Admission Registers[a]	Casenotes[b]
TB-Related	66	38
Non-TB Pulmonary	90	25
Other	2156	25
Total	2312	88

[a] Sampled year is 1826.
[b] Sampled years include 1826, 1829 – 30, and 1835 – 39.

DISSECTION AND THE ANATOMY ACT

Frequent human dissection was necessary to understanding the pathological anatomy underlying disease, and thus to developing the physical examination and pathological disease concepts I discuss here. From the time of Henry VIII to the passage of the Anatomy Act, the gallows was the only legal source of bodies for dissection (Richardson 1988, xv). As medical education became increasingly reliant on anatomical knowledge approaching the Victorian era, demand far outstripped the legal supply. As is well known from depictions of bodysnatchers like Dickens' (1859) Jerry Cruncher, anatomists filled the void by purchasing bodies and body parts acquired by bodysnatchers from pit burials, individual graves, vaults, and even private homes (Richardson 1988, c. 3), often reselling the corpses in parts to students at a markup (Richardson 1988, xv). While the trade was certainly offensive to prevailing morals and attracted a great deal of outcry (Richardson 1988, c. 4), it operated within the outskirts of legality: as the body itself was not considered legal property, bodysnatching could not be prosecuted as theft unless clothing and accoutrements were taken as well (Richardson 1988, 58).

In the decades prior to the 1832 Anatomy Act, bodysnatching became a preoccupation of the British public. The great demand for anatomical specimens in the early

decades of the nineteenth century — in 1826, twelve London anatomy schools reported the dissection of 592 individuals by 701 students (Richardson 1988, 54) — increased the stakes for bodysnatchers and body consumers alike. Bodysnatching became big business, particularly in high-demand metropolitan areas, with paupers' pit burials and poor-quality coffins known to be popular targets (61–63). Perpetrators banded together in small gangs (59), worked in concert with or bribed officials for access to burial grounds (62), and were said to recruit female accomplices to falsely claim bodies from workhouses (64). At times, they achieved startling hauls: on a single occasion, one enterprising individual extracted enough teeth from corpses interred in a burial vault to earn £60 (67), the equivalent of 40 weeks' pay for a London artisan.[1] The famous English surgeon Sir Astley Cooper is known to have worked closely with his preferred corpse purveyors, even commissioning them on one occasion to collect a body of interest; while it is unknown whether the body was freely consigned to Cooper, it seems unlikely that he would send bodysnatchers to acquire a donation (Richardson 1988, 64).

Public outcry regarding the market in corpses reached a crisis point when the trade escalated to Burking. The eponymous William Burke, along with his partner William Hare, were accused of murdering sixteen individuals in 1828 Edinburgh and selling their bodies to the anatomist Robert Knox (for an excellent discussion of the Burke and Hare murders, see Rosner 2010). After Hare provided evidence in exchange for immunity, Burke was convicted and hanged in a highly publicized trial (Richardson 1988, 133). This case, along with the similar one of Bishop and Williams in 1831 London (Burrell and Gill 2005, 488), unsurprisingly caused a moral panic surrounding not only the Burkers and bodysnatchers, but also the anatomists receiving bodies. "Burke's the butcher, Hare's the thief/And Knox

[1] Bowley (1900) estimated the average weekly wage for a London artisan to have been 30 shillings in 1824 (70).

the boy who buys the beef," went a popular song in the wake of the Burke and Hare murders (Rosner 2010, 246); two weeks after Burke's conviction, an angry mob paraded an effigy bearing what was said to be a "tolerable resemblance" to Knox through the streets of Edinburgh, hanging it in view of Knox's house (Richardson 1988, 138). Associated protests broke out throughout the city, with one group exhibiting resentment of the surgeons as a body by breaking windows at the College of Surgeons (139).

Scottish cities, particularly Edinburgh, figured prominently in the bodysnatching scandals of the early nineteenth century. Medical instruction in Scotland customarily included surgical training (Jenkinson 2012), which increased the demand for bodies beyond that expected from the popularity of medical instruction there alone. Scottish anatomists had long worked to develop aboveboard sources of corpses for dissection — Scottish Barber-Surgeons were granted unclaimed bodies in the late seventeenth century (Fido 1988, 14–15). However, David Hamilton (1981) reports that in early nineteenth-century Glasgow, anatomy-school officials waived tuition for students providing corpses, indicating that medical education authorities at best turned a blind eye toward student involvement in bodysnatching (152). In 1826, two years prior to the Burke and Hare murders, authorities found a large-scale corpse-dealing operation in Liverpool to be in possession of thirty-three bodies in various stages of pickling; they discovered the cache following the interception of eleven bodies in barrels marked "bitter salts" at the Liverpool docks, packaged for shipment to Edinburgh (Burrell and Gill 2005, 489–90). Thus, by the nineteenth century, the Scots were well-known consumers of the bodysnatchers' wares.

Seeking not only to assuage public anxiety regarding bodysnatching, but also to improve medical training and medical science in the UK (Richardson 1988, 101–102), in

1828 the House of Commons first appointed a Select Committee to examine the sources and legal issues surrounding anatomical subjects (101). The need for anatomical specimens was a foregone conclusion; the question was only how they might be best supplied. After not particularly seriously mooting a range of alternative solutions, including criminals, freely donated bodies and imported corpses from the continent (c. 7), those framing the Anatomy Act in the UK decided on a course similar to France, which granted the bodies of the very poor to medical men for dissection from the close of the eighteenth century (102). Enactment of the 1832 Anatomy Act ensured a plentiful, aboveboard supply of anatomical specimens for medical training by granting unclaimed bodies from public institutions for dissection. As Richardson (1988) discusses in depth, this ended bodysnatching and Burking by eliminating the market for corpses, sparing the middle and upper classes by offering up the bodies of the poor in their stead. Dissection was popularly dreaded owing not only to the long history of dissection as criminal punishment (Fido 1988, 2–3), but also to cultural belief in physical resurrection (Richardson 1988, c. 4). Following the Anatomy Act, the poor were at risk for such a fate not just by virtue of their insecure burials, but also by legal statute.

The Anatomy Act was consistent with a more generalized hardening of attitudes toward the poor in the early nineteenth century UK. In essence, the Anatomy Act, like the 1834 Poor Law Amendment Act (4 and 5 William IV c. 76), constructed the recipients of public charity as debtors: those unable or unwilling to engage in productive work placed a burden upon society that must be repaid. In emphasizing the workhouse, the Poor Law Amendment Act sought to recoup inmates' "debt to society" not only through the exchange of labor for charitable relief, but also in the punitive sense of that phrase. In rendering dissection a likely consequence of entering the workhouse, the Anatomy Act reinforced a punitive attitude toward recipients of public charity through the longstanding association of

dissection with crime. However, due to the obligation incurred by paupers' acceptance of public charity, the Anatomy Act also construed the taxpaying public as entitled to the medical knowledge gained through paupers' dissection. Under the Anatomy Act, paupers would discharge the burden they placed on society by furthering medical science. The medical progress represented by physical examination and pathological concepts of disease was thus by design built upon the dissected bodies of the poor, an exploitative relationship reflected in subsequent interactions between practitioners and working-class patients, as I will show.

THE BRITISH INTRODUCTION OF PERCUSSION AND AUSCULTATION

The early establishment in France of postmortem dissection of the poor allowed the development there of two techniques that would dramatically alter British medical practice. One of these was thoracic percussion; John Forbes translated Auenbrugger's treatise on the topic (1761) into English with commentary in 1824 (Auenbrugger et al. 1824). Practitioners throughout Europe largely ignored Auenbrugger's percussion prior to Corvisart's (1808) French translation, which popularized and greatly extended the technique (O'Neal 1998). Percussion called for the practitioner to tap the patient's thorax with grouped, extended fingertips, mapping pathology within by the degree of resonance produced: an area of dullness corresponded to disease (Auenbrugger et al. 1824, 13). Corvisart claimed that an experienced practitioner could detect an area of dullness as small as a walnut; even where percussion was least useful, the central chest, he could identify an obstruction no larger than an egg (O'Neal 1998, 482). Auenbrugger likened the practice to ascertaining the level of liquid in a cask by striking it: for both the cask and the lungs, the resonance of the sound produced increases with the volume of air in the vessel (Auenbrugger et al. 1824, 15).

The other technique was mediate auscultation, introduced by Laënnec's 1819 treatise, which John Forbes translated into English with commentary in 1821 (Laënnec 1821). Laënnec, a student of Corvisart in Paris, drew upon the existing techniques of percussion and direct auscultation, or listening to chest sounds by holding the ear flush against the patient's thorax, to develop mediate auscultation — listening to chest sounds by means of an instrument, the stethoscope. He identified and described a wide range of sounds produced by the heart and lungs, tracing the various rattles, crackles, whistles, gurgles, and murmurs to the pathological changes associated with various thoracic diseases. As a result of this specificity, mediate auscultation was a notable improvement on percussion and generated a great deal of interest: by 1827, Forbes noted in his preface to the second edition of his translation of Laënnec's treatise, mediate auscultation had made "great progress … in every country of Europe. … There is, indeed, hardly any one of the civilized nations of the world, which cannot now afford examples of its acknowledged utility …. Certain it is … how extensively and accurately [auscultation is] practised by many physicians and surgeons, of distinguished talents, in England, Scotland, and Ireland." (reprinted in Laënnec 1834, ix–x)

Scotland adopted the stethoscope more rapidly than the rest of the UK; Malcolm Nicolson (1993) attributes this to Scotland's closer intellectual connection to the continent, particularly France, whereas English physicians rejected the Parisian innovation on nationalistic grounds (150–51). Forbes singled out Scotland — or at least one Scottish physician — as noteworthy for early acceptance. In his 1824 work on percussion and auscultation, written with the express purpose of popularizing these practices, Forbes lamented:

> A few years since, when the work of M. Laennec was first introduced … it appeared from the reports of the Medical Journals, and the unusually rapid sale of the

> Translation, that a very considerable impression had been made on the public mind, in favor of the diagnostic measures therein recommended. Since that time, however, if we may judge from the same Journals, the impression then made seems to have been productive of few practical results; as I am not aware that even a single case, illustrative of the use of Auscultation, has appeared in any one of them. From this circumstance, and from not having heard of its employment in any hospital, or indeed by any individual practitioner in this country (with the exception of my friend Dr. Duncan, Jun. of Edinburgh), I am led to fear that the impression made was more lively than profound[.] (viii)

However, as Nicolson (1993) has shown, as late as 1818, the physicians of Edinburgh — including Andrew Duncan, Jr., at that time Professor of Medical Jurisprudence at the University of Edinburgh and editor of the *EMSJ* — were less receptive to percussion. William Cullen mentioned Auenbrugger's work, albeit equivocally ("How far the method of Auenbrugger will apply to ascertain the presence of water and the quantity of it in the chest [in cases of hydrothorax], I have not had occasion or opportunity to observe." (Cullen 1784, v. 4, 314)) in his *First Lines of the Practice of Physic* as early as the 1784 edition (Nicolson 1993, 136). This comment continued unchanged through subsequent editions of 1788 to 1822 (Jarcho 1961), the lattermost having been edited by Cullen's successors following his death in 1790. Despite outwardly positive reception of the technique — such as the favorable 1811 review of Corvisart's *Essay sur les maladies et les lésions organiques du coeur et des gros vaisseaux* in the *EMSJ* likely written by Andrew Duncan, Jr. himself (Nicolson 1993, 136) — references to the use of percussion are rare in detailed notes of Duncan's practice through at least 1818 (Nicolson 1993, 137–38).

In contrast, interest in and use of the stethoscope at Edinburgh propagated relatively rapidly. Medical staff recorded the earliest published use of the stethoscope in clinical examination at Edinburgh in late 1820 (Nicolson 1993, 142), just a year after Laënnec's original publication (1819) and prior to Forbes's popular translation (Laënnec 1821). Not

long thereafter, an MD thesis (Locock 1821) indicated additional theoretical interest in, if not the practical application of, stethoscope examination in Edinburgh (Nicolson 1993, 140–41). By 1825, however, Edinburgh practitioners were advanced enough in use of the instrument to improve upon Laënnec's observations: James Hope's MD thesis in that year demonstrated, contra Laënnec, that the instrument could be used to identify aortic aneurisms (Nicolson 1993, 145).

Adoption of the stethoscope in Edinburgh was aided by expert training. In the early 1820s, most likely late 1822, the younger William Cullen — grand-nephew of the William Cullen discussed in chapter three and above — undertook training in the use of the stethoscope under Laënnec himself in Paris; by 1824, the younger Cullen was a house surgeon at RIE and lectured there on the use of the instrument (Nicolson 1993, 144). Similarly, James Crauford Gregory travelled to Paris for training by Laënnec in 1824, returning to practice as a physician at RIE in 1826 (ibid.). Both Gregory and the younger Cullen participated in editing and updating the posthumous 1829 edition of the elder Cullen's *First Lines in the Practice of Physic*, which argued that the older editions' union of pneumonia and pleurisy could no longer be supported in light of stethoscope data (148).

Acceptance of the stethoscope was not universal in Great Britain. Queen Victoria, despite having been treated by indefatigable advocate of percussion and auscultation John Forbes (Thomson 1912, 62), resisted physical examination to the end of her life. According to Porter (1993), her last personal physician, Sir James Reid, "noted her 'great aversion' to the stethoscope" — "it was only after her death that [Reid] discovered that she had a 'ventral hernia, and a prolapse of the uterus' — surely proof that he had never once given her a full physical examination." (180)

Queen Victoria's opposition was not unique; resistance to the instrument in England remained entrenched to the mid-nineteenth century. Although the early reception of Forbes' translation of Laënnec (1821) was overall positive outside of Scotland as well as within, Nicolson (1993) points out that much praise of the volume centered around Laënnec's discussion of pathological anatomy, not the diagnostic potential of the stethoscope itself (139). Indeed, an 1825 *Lancet* review of William Stokes's (1825) Edinburgh volume on use of the instrument mocked the superlative hype surrounding the stethoscope's introduction and the instrument's potential to supplant the experienced physician's uniquely perspicuous interpretation of patient history. "It came into the world, from the hands of Laennec, like Minerva from the hands of Jupiter, perfect in all its parts," the reviewer sniffed:

> Through its agency, the morbid mysteries of nature, which had hitherto baffled human experience, were now to be unveiled to the crude perceptions of the merest tyro in the profession of medicine. … The antiquated system of question and answer, … by which our goodly ancestors groped out the seat and nature of thoracic complaints, were to be exchanged for the more certain and compendious divination of the stethoscope. A physician had only to put his hand in his pocket, apply this mahogany oracle to his ear, and, *presto*, all was to have been as plain to his understanding as if he had actually made a dissection of the parts themselves. ("Review from the West" 1825, 471)

The reviewer went on to reluctantly acknowledge that although the instrument may be of some use — "We are far from … assert[ing] that it will not facilitate diagnosis in practised hands, though we have frequently seen its vatic nations falsified by dissection" (472) — its effective application required a fine discernment of the great variety of chest sounds that was beyond the grasp of all but the most dedicated practitioners.

This attitude was neither isolated nor of short duration. Nicolson (1993) reports that in 1827, in response to Duncan's "satisfaction to see that [percussion and stethoscopy] are duly appreciated by the whole profession," a London correspondent to the *EMSJ* countered, "These means are very far from being duly appreciated by the whole profession, or by one

half of the profession … on the day we received [Duncan's] journal, a professor of physic, and a public lecturer in the metropolis … publicly denounced the stethoscope as a … piece of quackery, which he never could countenance!" (both Duncan and anon. quoted in Nicolson 1993, 150) A biographer of the mid-century professors of London's St. George's Hospital described them as "despising the stethoscope and microscope … [as] impractical toys." (quoted in Lawrence 1985, 514) By 1863, Sydney Jones, Assistant Surgeon at St. Thomas' Hospital — also in London — encouraged incoming students to apply themselves to the study of instruments like the stethoscope, despite their medical ancestors' characterization of such technology as "lumber," such terms as which he had "not unfrequently heard … applied." (Jones 1863, 400) At the same time, however, Jones cautioned that instruments "may afford you very valuable aid; but mind, you are to use them as aids, and not as substitutes for other knowledge." (ibid.) Such attitudes contrast sharply with those Nicolson (1993) describes for Edinburgh. There, Nicolson identified only one critical reference to the stethoscope: in an 1825 letter, William Stokes found unlikely the possibility that the stethoscope could detect a fetal heartbeat (149), a relatively mild criticism related more to the instrument's scope than its overall utility in diagnostic practice.

As I stated above, Nicolson (1993) attributes the early adoption of the stethoscope in Edinburgh to Scotland's intellectual affinity with France; I would argue that the early emphasis on anatomical and pathological knowledge in Scottish medical training compared to that in England further supported acceptance of the stethoscope in the former. The association of diseases with distinct internal pathology moved diagnosis inside the body; the utility of the stethoscope in diagnosis rested upon linking externally detectable signs — the sounds observed through mediate auscultation — to distinct internal pathology. Mediate auscultation could only add appreciably to the clinician's understanding of a patient's case

through a framework to connect sound to specific lesion to specific disease. Without first ascertaining that caverns in the lungs form most commonly from softening and evacuation of tubercular matter and secondarily from pulmonary gangrene or abscess, and subsequently linking pectoriloquy — the transmission of the patient's voice into the lung — to such excavations (Laënnec and Laënnec 1831, v. 1, 67; Laënnec 1834, 38), the identification of pectoriloquy in a patient through auscultation could contribute little to differential diagnosis. Anatomy and pathology had long been uniquely emphasized in Scotland, likely rendering Scottish-trained physicians and surgeons more prepared than their English colleagues to grasp the practice of auscultation and turn it to productive use and placing Scottish medical practice at the leading edge of early nineteenth-century advances.

THE STETHOSCOPE IN GLASGOW

Although Nicolson (1993) provides a thorough understanding of the adoption of the stethoscope by Edinburgh professors and practitioners of medicine, my work for the first time examines its use and acceptance at GRI. My earliest sampled references to percussion and auscultation at GRI occur in the case of Mary Anne D., a 27-year-old single woman admitted to GRI on July 5, 1826 (HB14/5/15: 187). Diagnosed with phthisis and liver disease, she received treatment in hospital for thirty days before dying on August 4 (HB14/5/15: 206). In the notes of Mary Anne D.'s postmortem dissection, the clerk noted:

> The right lung was about the size of a fist firm and covered by flaky purulent matter. [T]he surface retained no vestige of pleural covering but appeared ragged as if a portion had been forcibly torn away. [B]eneath the lung, lying on the pleura costalis, were five ounces of thick pus. The pipe of a bellows was placed in the trachea and air blown into the lungs but from right lung it passed into the cavity of the chest by innumerable bronchial apertures which had been opened by the progress of the disease. Left lung contained a few immature tubercles at its superior part, otherwise it was it was [*sic*] quite healthy. …

> Stethoscope
> The stethoscope was applied to this person at different periods during three weeks previous to death [i.e., from approximately July 14]. The metallic tinkling which M. Lannec [*sic*] describes as a test of the existence (generally) of empyema and fistulous communication with the bronchia giving rise to pneumo-thorax, was most clearly and extensively heard beneath and above right clavicle. [O]ther diagnostics as percussion and want of respiration on this side corroborated the conclusion[.] (HB14/5/15, 206, 242; emphasis retained)

The absence of auscultation and percussion findings from the case notes recorded during Mary Anne D.'s lifetime speaks to the experimental nature of this first instance of these techniques. Instead, the inclusion of physical examination findings only after their corroboration by postmortem suggests an early, tentative and unorthodox trial, yet one considered of sufficient import to include with the postmortem results, set off and emphasized by a separate heading.

While this case is unique among records prior to 1835 in my sample, I cannot say whether it truly represents the first trial of percussion and auscultation by GRI practitioners, nor whether Mary Anne D.'s phthisis made her particularly attractive for a trial of the newly popular techniques. Following the case notes from December 1819 that I discuss in chapter three, no ward journals are preserved before those of late March 1826. The cases I sampled for the period 1826 – 1830 are furthermore sparse; owing to a paucity of ward journals available, only 11 patients were sampled for 1826 (HB14/5/15), three for 1829 (HH67/3/1), and two for 1830 (HH67/3/1). It may be that percussion and auscultation were undertaken on every patient during the period 1826 – 1830, but the results were never recorded in the ward journals because no opportunity presented itself for postmortem confirmation of the findings. However, one individual other than Mary Anne D. did die in hospital: Christian C., diagnosed with "pneumonia &c." (HB14/5/15, front matter), was also dissected after her death, but medical staff made no mention of auscultation and percussion in the postmortem

findings (HB14/5/14, 123). This suggests that clinicians did not universally undertake physical examination during this period. As a result, the extent to which Mary Anne D.'s physical examination was unique remains unknown, but its use in her case indicates the hesitancy with which such examinations were undertaken prior to 1835, likely due to these techniques' novelty.

By 1835, for which ward journals were more frequently preserved, percussion and auscultation were established at GRI as common techniques for patient examination. Although the lack of case notes for the period 1826 – 1830 prevents any informed assessment of the reasons behind the transition, the difference is stark: for the period 1835 – 1840, clinicians recorded auscultation alone or both auscultation and percussion for virtually all patients admitted under TB-related or other pulmonary diagnoses. Practitioners also used auscultation and/or percussion in examining one-third of patients admitted under other diagnoses — those for whom thoracic pathology was indicated (table 4.2). The only pulmonary case for whom no physical examination was specified, John S. (HH67/9/2, 191), likely did receive auscultation at least: the clerk noted only that "the functions of respiration appear to be in their normal condition," indicating an examination in which no abnormal findings were present. The reference to respiratory function rather than resonance suggests auscultation rather than, or in addition to, percussion. The absence of auscultation and percussion findings for most non-TB, non-pulmonary cases may indicate that at this early stage in the adoption of these techniques, medical staff applied them only where indicated by thoracic symptoms reported by the patient; alternatively, they may have used auscultation and percussion in every case, but only recorded abnormal findings — or those counter to expectations, as for John S.

Table 4.2. Auscultation and percussion examinations, 1835 – 1840

	Diagnosis Groups			
Examination(s) Noted	TB-Related	Non-TB Pulmonary	Other	TOTAL
Percussion Only	0	0	0	0
Auscultation Only	3	5	1	9
Both	30	13	4	47
Neither	0	1[a]	15	16
TOTAL	33	19	20	72

[a] It is likely that this individual, John S., actually received an unspecified physical examination; his case is discussed in the text.

Despite the common usage of auscultation and percussion at GRI during the period 1835 – 1840, it is clear that the former technique was still relatively unfamiliar to practitioners. That medical staff also considered auscultation the primary of the two is indicated by its notation either alone or with percussion; never are percussion findings recorded in the absence of auscultation results. The results of stethoscopy are sometimes recorded doubtfully, however, in contrast to the straightforward presentation of percussion findings. For example, in the case of Michael O'H., diagnosed in 1835 with pleuro-pneumonia, the clerk noted, "From inferior angle of right scapula downwards a superficial crepitus is heard resembling the pleuritic … more than the moist crepitous rattle, but this latter râle becomes very distinct on making a full inspiration" (HB14/5/4, 50). Notation of the observer's equivocation as to the initial character of the rale indicates not only a measure of doubt at first observation, but also a nod to the possibility that this initial detail will contribute to diagnosis. This suggests a degree of uncertainty in both the observation of sounds — supporting the claim of the 1825 *Lancet* reviewer that appropriately distinguishing sounds proves difficult for many (472) — as well as their interpretation.

Indeed, the reliance of Glasgow practitioners on Laënnec's teachings is clear from the retention of French terms to describe auscultatory signs. Thus, "frottement" is at times substituted for friction (HB14/5/4 1835, 89), "gargouillement" (HH67/2/6 1838, 50) or the misspelled "gurgouillement" (HH67/2/6 1838, 108) for gurgling, and "soufflet" for bellows murmur (HH67/9/1 1838, 125). This retention of French terms suggests that, as in Edinburgh (Nicolson 1993, 144), Glasgow practitioners undertook training for the technique in France, then returned to teach the French method at GRI. At the least, it indicates a number of GRI practitioners studied the technique from the original French texts rather than English translations and reformulations, supporting an affinity among Glasgow students of the new technique for primary, continental sources.

Taken together, the observations I have discussed indicate that while the use of auscultation and percussion became commonplace at GRI over the period 1821 – 1840, practitioners were not wholly fluent in these new methods; nonetheless, physical examination had clearly already altered diagnostic practice and constructs of TB-related diseases. In quantitative analysis, TB-related diagnoses continued to be distinguished from both non-TB pulmonary and non-TB, non-pulmonary disorders in multiple symptom categories (table 4.3). TB-related disorders differed significantly ($p < 0.05$) from those of non-TB, non-pulmonary disorders in the categories of appearance and comportment, dermatological, digestive, history, pain, and miscellaneous clinical indicators (see appendix D for a complete listing of signs and symptoms and their frequencies); differences approached significance ($p < 0.10$) for an additional category, mouth and throat. As for the period 1794 – 1820, the lack of statistical significance for respiratory symptoms most likely results from the paucity of observations in these categories for non-TB, non-pulmonary disorders. Relative to

the significant differences observed for the period 1794 – 1820, four symptom categories gained significance or near significance: digestive, history, mouth and throat, and pain. General respiratory symptoms lost near-significance.

TB-related diagnoses were distinguished significantly ($p < 0.05$) from other pulmonary disorders in the five categories of appearance and comportment, circulatory, digestive, general respiratory, and respiratory auscultation/percussion (table 4.3). Differences also approach significance ($p < 0.10$) in an additional two categories, dermatological and history. Compared to the earlier cases discussed in chapter three, four categories — appearance and comportment, circulatory, dermatological, and digestive symptoms — gained significance or near significance, while none lost significance.

Table 4.3. P-values for Fisher's exact tests of differences in clinical indicators, 1821 – 1840

Signs and Symptoms	TB-Related vs. Non-TB Pulmonary	TB-Related vs. Other
1. Appearance and Comportment	0.0011**	0.0429*
2. Circulatory	0.0221*	0.1627
3. Dermatological	0.0569†	1.24×10^{-9}***
4. Digestive	0.0048**	0.0011**
5. General	0.7377	0.4691
6. Genitourinary	0.4312	0.24
7. History	0.0820†	4.54×10^{-5}***
8. Mouth and Throat	0.8235	0.0874†
9. Pain	0.9277	4.43×10^{-7}***
10. Neurological	1	0.5675
11. Respiratory, General	0.0017**	0.4737
12. Respiratory, Auscultation/Percussion	1.88×10^{-5}***	0.6153
13. Respiratory, Chest Form/Sensation	0.7604	1
14. Miscellaneous	0.1533	0.0477*

Note: See appendix D for a complete list of signs and symptoms comprising each category.
† $p < 0.10$
* $p < 0.05$
** $p < 0.01$
*** $p < 0.001$

These findings indicate that the signs of disease clinicians observed using new physical examination techniques expanded rather than replaced older concepts of TB and its historical analogues. TB-related disorders were more strongly distinguished from both non-TB pulmonary disorders and non-TB, non-pulmonary diseases in the period 1821 – 1840 compared to 1794 – 1820: multiple established categories of signs and symptoms gained significance for the later period addressed here in addition to the significant difference between TB-related and other pulmonary disorders in the new category of respiratory auscultation and percussion. It is possible to make too much of these newly significant differences, as the increased statistical distinction between diagnosis groups may be an artifact of sample size — I performed quantitative analysis on a total amended sample of 51 patients (table 3.1) and 839 signs and symptoms (appendix C) for the period 1794 – 1820, compared with 88 patients (table 4.1) and 1750 signs and symptoms (appendix D) for the period 1821 – 1840. Nonetheless, the significant distinctions between TB and each other diagnosis group across multiple categories of clinical indicators demonstrates that the diagnosis of TB-related disorders continued to rely upon established symptoms categories. Despite the utility and promised specificity of the new techniques, percussion and auscultation findings formed only one aspect of diagnosis.

Indeed, TB-related disorders for this period continued to be distinguished from both non-TB pulmonary and other diseases by classical symptoms, despite contemporaneous disease concepts emphasizing tubercular pathology. Laënnec equated phthisis pulmonalis with pulmonary tubercles (Laënnec and Laënnec 1831, v. 2, 5; Laënnec 1834, 252),[2] and as a result he argued that accurate diagnosis depended upon their detection. He accordingly

[2] Where I have referenced specific passages by Laënnec, I include page numbers for the cited content in both the original French works and Forbes's English translations; all quotations are Forbes's translations, for which I independently verified the accuracy.

emphasized the percussive and auscultatory signs associated with the accumulation, softening, and excavation of tubercular matter in the lungs, for him the critical stages in development of the disease (1831, v. 2, 127–51; 1834, 308–17). Laënnec furthermore discounted classic symptoms such as emaciation, hectic fever — a daily-recurring fever accompanied by facial flushing — and the color and consistency of expectoration as tangential to the essential progress of the disease and insufficiently specific (1831, v. 2, 151–72; 1834, 317–29): for example, "Notwithstanding the efforts which have been made, in all ages, to deduce pathognomonic signs from the appearance of the expectoration in phthisis," Laënnec stated, "it must be confessed that this affords no peculiar characters which are not met with in the sputa of chronic catarrh. … Neither chemical analysis nor the physical characters of these matters, enable us certainly to discriminate them from each other." (1831, v. 2, 157; 1834, 321) Accordingly, he cautioned, "we ought never to assert positively the existence of phthisis pulmonalis, in cases where none of the physical signs afforded by percussion and auscultation, are found to exist." (1831, v. 2, 164; 1834, 325)

It is evident that GRI practitioners in the late Georgian – early Victorian period examined here did not share Laënnec's insistence upon the detection of pulmonary tubercles via physical examination as the only reliable means of diagnosing phthisis pulmonalis. The symptom categories significantly distinguishing TB-related disorders from non-TB pulmonary and other diseases overwhelmingly reflect classic symptoms of the former diagnoses: the differences driving significance in the category of appearance and comportment, for example, include possessing a delicate constitution, which I discuss below. Additional classic symptoms appear to have contributed strongly to significant differences, such as debility, emaciation, perspiration (particularly profuse perspiration and night sweats),

hemoptysis and bloody expectoration, as did common sequelae of advanced tubercular disease such as diarrhea. Indeed, GRI practitioners thus appear to have hewn more closely to Forbes's (1834) advice: "I would lay it down as a general rule, that the physician ought, in the first place, to endeavour to ascertain the nature and state of the disease by the common symptoms alone, and that [the stethoscope] should be only had recourse to afterwards, as a sort of *experimentum crucis*, to fortify his convictions in obvious cases, or remove his doubts in difficult ones." (ix)

Notable in this period is the continued, albeit limited, presence of constitutional risk for disease. A total of 10 (11 percent) patient records, all from 1838 and 1839, bear notes on constitution or humoral temperament, including three of good or tolerably good constitution (one from each diagnosis group), six of delicate or weak constitution (four TB-related, one non-TB pulmonary), and one of bilious temperament (TB-related) (table 4.4). All of these were recorded in volumes listing the same clerk, William Johnson, and neither sampled volume bearing his name lacked references to patient constitution (HH67/2/6; HH67/9/2). While references to good constitution were distributed across diagnosis groups, individuals said to be delicate, weak, or bilious were predominately those bearing TB-related diagnoses, indicating a conceptual link for William Johnson between these latter constitutional descriptors and TB-related disorders. It cannot be said that GRI staff more broadly held a similar link — indeed, the contrary appears to be the case. However, this observation speaks to the persistence, albeit limited, of constitutional constructs of TB and its historical analogues into the Victorian era. That these continued to hold explanatory power for early-Victorian practitioners demonstrates how deeply entrenched the paradigm of tubercular disorders as diseases essential to the sufferer remained in the face of pathological anatomy;

indeed, essentialist explanations for these disorders would persist into the twentieth century, as I discuss in chapters six and seven.

Table 4.4. Constitutional and humoral remarks

	Diagnosis Groups			
Constitution Noted	TB-Related	Non-TB Pulmonary	Other	TOTAL
Good/Tolerably Good	1	1	1	3
Delicate/Weak	5	1	0	6
Bilious Temperament	1	0	0	1
TOTAL	7	2	1	10

At the same time, GRI practitioners evidently were in this period incorporating Laënnec's internalized, tubercular construct of phthisis pulmonalis into classical concepts of the disease. Indicating support for the new construct was the highly significant difference between TB-related and other pulmonary disorders in the new category of respiratory auscultation and percussion ($p = 1.88 \times 10^{-5}$). This is driven by the signs endorsed by Laënnec (1831; 1834) as indicative of tubercle softening or pulmonary excavations resulting from the evacuation of softened tubercular material — bronchophony (1831, v. 1, 65–67; 1834, 37–38), cavernous respiration (1831, v. 1, 57; 1834, 34), gurgling (mucus rhonchus: 1831, v. 1, 100–105; 1834, 50–53), and pectoriloquy (1831, v. 1, 67–69; 1834, 38–39).

A number of the respiratory auscultation and percussion signs differentiating TB-related from other pulmonary disorders in my sample departed somewhat from Laënnec, however. Also over-represented in the TB-related sample were increased vocal resonance, an equivocal description of pectoriloquy likely reflecting practitioners' lack of confidence in identifying the latter sign, and tubercular crepitus, a sound defined by neither Laënnec (1831;

1834) nor the GRI practitioners but evidently (and circularly) bearing the confidence of the latter in its significance in diagnosing tubercular disease. The similarly named signs emphyematous and pleuritic crepitus, as well as friction, are under-represented in the TB-related sample, as is expected of signs clearly considered related to other pulmonary disorders. Counterintuitively, the undefined yet seemingly circularly-termed pneumonic crepitus was represented only in TB-related cases and never in cases of other pulmonary disorders, which naturally included pneumonia, demonstrating incomplete clinical segregation between pneumonia and tubercular disorders during this period. Taken together, these eponymous signs may suggest the development of a regional or institution-specific vocabulary of auscultation and percussion, departing from and building upon Laënnec's work through shorthand descriptions of particular signs as characteristic of particular disorders. The relationship of these non-Laënnec descriptors to those observed at RIE will be addressed in chapter six.

In contrast to the increasingly specific construct of TB-related disorders reflected in analysis of diagnostic signs and symptoms, the treatments prescribed at GRI remain largely nonspecific for the period 1821 – 1840. As in the period 1794 – 1820, practitioners employed a broad range of drugs in the late Georgian – early Victorian period (appendix I). Quantitative analysis, summarized in table 4.5, demonstrates an overall lack of specificity. Clinicians prescribed no category of treatment with significantly different ($p < 0.05$) frequency in cases of TB-related versus non-TB, non-pulmonary disorders. Mercurial treatments did approach significance ($p < 0.10$), however. Mercury was something of an all-purpose drug, considered by Cullen (1789) to have cathartic, corrosive, diaphoretic, emetic, errhinic (nasal decongestant), demagogic (menstruation inducing), and sialogic (salivating)

properties, but was most commonly prescribed in this context as an anti-syphilitic; it is this association that drives the near-significant difference observed.

Table 4.5. P-values for Fisher's exact tests of differences in treatments, 1821 – 1840

Treatments	TB-Related vs. Non-TB Pulmonary	TB-Related vs. Other
1. Anodyne	1	1
2. Antacid	1	1
3. Antispasmodic	0.8142	0.5413
4. Astringent	1	0.4286
5. Bleeding	0.2892	0.807
6. Cathartic	0.6543	0.2644
7. Corrosive	0.4444	0.2857
8. Demulcent	0.4706	1
9. Diaphoretic	0.7063	0.65
10. Diuretic	0.3971	0.7173
11. Emetic	0.0166*	0.8265
12. Emollient	—	—
13. Expectorant	1	1
14. Mercury	1	0.0657†
15. Opium	1	1
16. Sedative	0.25	1
17. Stimulant	0.8036	0.8253
18. Tonic	0.6182	0.849
19. Indeterminate Function	1	0.1013
20. Unclassified Drugs	0.3383	0.6682

Note: See appendix I for a complete list of treatments comprising each category.
† $p < 0.10$
* $p < 0.05$

The treatments applied in cases of non-TB pulmonary disorders were similarly not distinguished from those of TB-related diseases. These diagnosis groups differed significantly in only one category, emetics ($p = 0.0166$). This distinction was driven by the prescription of emetics other than antimony, primarily ipecac, in cases of TB-related disorders. As practitioners prescribed this class of drug with comparable frequency in both diagnosis groups (0.45 prescriptions per TB-related case; 0.4 prescriptions per non-TB

pulmonary case), I am left with the possibilities that antimony compounds were considered either somewhat contraindicated in treating TB and related disorders, or particularly beneficial in cases of other pulmonary disorders. As antimony compounds could be prescribed as a cathartic or diaphoretic in addition to an emetic, it may also be that clinicians prescribed this drug with different intents for various TB-related and other pulmonary disorders. With little textual support to distinguish among these alternatives, the question remains unresolved. However, this single significant difference disturbs the overwhelming perception of nonspecific treatment but little.

As in the period 1794 – 1820, these treatments furthermore seem to have been largely ineffective in cases of TB-related disorders. Table 4.6 shows the outcomes recorded for each of the three diagnosis groups in the admission register sample for the period 1821 – 1840, consisting of the 1826 register only, owing to poor preservation of records for this time span. Whereas the two most common outcomes in TB-related cases are relief and death, those for both non-TB pulmonary and other disorders are cure and death. Despite the commonality of death, the distribution of recorded outcomes differs highly significantly ($p < 0.01$) between TB-related diagnoses and both non-TB pulmonary cases ($p = 1.707 \times 10^{-11}$) and non-TB, non-pulmonary disorders ($p = 2.2 \times 10^{-16}$). Owing to the commonality of death, however, one gains an impression of the continued ineffectiveness of medical treatment in cases of TB and analogous conditions, despite the dramatic advances gained in the study of pathological anatomy during this period.

Table 4.6. Outcomes, 1821 – 1840

Outcomes	TB-Related	Non-TB Pulmonary	Other
Advice	3	1	39
By Desire	5	1	55
Cured	10	60	1705
Died	13	16	175
Improper	2	0	7
Irregular	1	1	19
Relieved	31	9	118
Remitted/Other[a]	0	2	29
[Blank]	1	0	9
P-Values		1.707×10^{-11}***	2.2×10^{-16}***

[a] Remitted patients were transferred to another ward or hospital, while "other" discharges were specified only as such in the register.
*** $p < 0.001$

DISSECTION, PHYSICAL EXAMINATION, AND CHARITABLE MEDICAL PRACTICE

The hands-on nature of physical examination techniques introduced in charitable medical practice at GRI during the period 1821 – 1840 demanded increased access to poor individuals' bodies similar to that demanded and achieved by medical men advocating for the Anatomy Act. While Queen Victoria resisted physical examination, even with the stethoscope interposed between herself and her physician, until the end of her life (Porter 1993, 180), the pulmonary patients at GRI had no such prerogative: as early as 1835, all were examined with the stethoscope and often percussion as a matter of course. Indeed, uniform access to physically examine patients — and the training benefits practitioners thus accrued — was assumed by the *EMSJ*'s anonymous 1822 reviewer of Laënnec (1819, 1821). He urged that "Any one who will take the trouble to apply the cylinder [i.e., the stethoscope] with tolerable attention and adroitness, to the chests of half a dozen of patients in an [*sic*]

hospital, will easily satisfy himself of the great diversity to be observed in the action and rhythm of the heart, and in the sounds caused by respiration" (457). That the reviewer suggests this experiment specifically on *hospital* patients speaks to the freedom with which hospital practitioners regarded their patients' bodies.

Together with this assumption of access to poor patients came a lack of consideration for their prognosis and feelings. The 1822 *EMSJ* reviewer countered a common argument against the stethoscope, writing:

> In an [*sic*] hospital, where the thorough examination of every patient, whatever the malady be, is a matter of course, nothing is thought of [stethoscope examination], and no bad consequence can follow. But in private practice, we have found the greatest objection to its use arise from its leaving no doubt in many cases of tuberculous consumption, as to the incurable nature of the complaint, so that we could no longer flatter ourselves, or honestly support the spirits of our patients by holding out any hopes of recovery; in such cases, it is not to be employed without mature deliberation. (457)

While diagnosing terminal consumption in a private patient dispirits both the practitioner and the patient seriously enough to require "mature deliberation" before deploying the diagnostic power of the stethoscope, "no bad consequence can follow" from its indiscriminate use on every patient in a hospital. Whether this was because hospital patients were not granted the same right as private patients to know their diagnosis and prognosis, or because the practitioner would be unconcerned with the effect of such information on the spirits of a poor patient is unclear. In either case, however, clinicians sharply distinguished their treatment of poor and wealthier patients in this respect.

The treatment of charity hospital patients with respect to both consideration and bodily access reveals a distinct gulf between the poor and their wealthier contemporaries in the medical setting specifically. That is, class distinction evident within the medical context examined here reflects the increasingly well-defined class distinctions evident in such broader

developments as the Poor Law Amendment Act and the Anatomy Act discussed above. During this period of increasing class distinction and hardened attitudes toward the poor, a new, less collaborative practitioner-patient relationship was evident. Increasing distinction between patient and practitioner accounts of disease, absent from GRI case notes from the period 1794 – 1820, was evident in 1821 – 1840. The earlier Georgian – Regency intake notes at GRI took the form of a single case narrative blending patient and practitioner observations regarding the patient's health — a procedure consistent with the externalized, discursive diagnostic procedures common at the time. By the late Georgian – early Victorian period, however, practitioners took care to distance themselves from certain statements by more often attributing them to the patient with such prefaces as "he says:" "says he only coughs when his feet are cold," (John W., phthisis: HB14/5/4 1835, 81) for example, or "Took ardent spirits but she says not to excess" (Mary Anne D., discussed above: HB14/5/15 1826, 187).

As this latter example indicates, disparaging comments on patient behavior also began to appear in the period 1821 – 1840. Medical staff noted patients' "intemperate habits" (e.g., William E., phthisis: HH67/9/2 1838, 79), particularly in relation to disreputable disorders like syphilis: "Of intemperate habits but denies most strenuously ever having had primary syphilis," the clerk wrote of secondary syphilis patient Catherine G., who also "denie[d] ever having had hemoptysis." (HH67/2/6 1838, 50) The use of "denial," with its connotation of willful dishonesty, is significant here. Medical staff occasionally brought their complaints about patient behavior to the GRI Board of Managers: the managers' minutes for February 21, 1826 noted, "D'r [*sic*] Millar stated that the public makes this [the outpatient clinic] a perfect lounge; that they come in with trifling ailments; that they comply

with, or neglect the advice they receive as whim strikes them; and that they sometimes even tear the written prescriptions before they leave the premises" (HB14/1/5, 64).

At the same time, GRI practitioners did not receive criticism of themselves gracefully. In 1828, Thomas Carter, a surgical dresser at GRI, wrote two inflammatory letters to the *Lancet*. In the first, appearing October 11, he described a case in which the GRI surgeons erroneously diagnosed a nurse at the infirmary, having fallen and injured her hip, with a dislocation of the femoral head (Carter 1828b). They accordingly subjected her to two attempted reductions of the nonexistent dislocation:

> suppose the patient … again dragged into the operating theatre, to have the reduction of this imaginary dislocation effected. … Imagine [the surgeon] sending her once more to bed, but not till after he has the second time made a great and heroic attempt (with the assistance of his pulleys, and some four-and-twenty men of strength,) to give the poor unfortunate, relief. … [T]ell me what must be your surprise and astonishment, when … you are coolly informed that … [it is] uncertain whether there '*now be*, or *ever have been, any dislocation!!!*' What, let me ask, must the public, as well as the pupils, think of such a surgeon? (60; emphasis retained)

In his second letter, appearing November 15, Carter reported having been summoned to a disciplinary hearing and threatened with expulsion. On his being retained on the basis that "*nothing* could be done to me, as I had only stated those things which were in reality true," visiting surgeon John Couper reportedly commented, "we must take care not to have any literary dressers again." (Carter 1828b, 219; emphasis retained). In response to this, Carter wrote, "Now, what the man really meant by this wise speech of his, is, perhaps, somewhat doubtful. There appears to me, however, but one clear and obvious construction of which it admits; viz., that conscious of his own ignorance and inability to discharge the duties of a hospital surgeon as he ought to do, he is anxious not to have any dressers under him, who being better informed than himself, will be able to expose his errors, and hold him up to deserved ridicule." (ibid.) Subsequent to his authoring this second letter to the *Lancet*,

the GRI managers evidently decided they could do something after all: the minutes of the meeting of the Board of Managers for December 11, 1828 record that they called Carter for a second disciplinary hearing at which they expelled him from GRI for improper conduct (HB14/1/5, 241–42).

Indeed, Carter could have criticized GRI practitioners more broadly for their exploitative approach to patients. The outpatient clinic was discussed again in 1835: on multiple visits, patients "find a new [practitioner] in the lobby who knows nothing of the complaint…. This change of medical attendants is disheartening to the patient, because he has to tell the same tale every time he appears and probably to a different person each time; he cannot help feeling that there is no interest taken in his case the consequence is that after making his appearance once or twice he never returns." (HB14/1/7, 117) This circumstance was primarily addressed with reference to its impact on the infirmary rather than the patient, however, the record continuing, "Many cases interesting in themselves and useful to the students are lost in this way." (ibid.) The students were themselves not above taking advantage of patients for their own benefit. In a letter of May 2, 1821, the medical staff reported that students had preyed upon patients' fears of the hospital to convince patients to instead engage the students for private practice: "In several instances some (we sincerely hope a very few) of the pupils have secretly tampered with the patients by telling them that they are merely the subjects of experiment in the hands of the surgeon; that they have been improperly treated; and that they will be maimed for life. By having their apprehension thus excited, some patients have been induced to quit the house, and to take lodgings in town; willing to submit themselves to the treatment of these their base advisers, in the expectation that they will expedite their cure." (W. Couper et al. to GRI Managers: HB14/6/2, 1–2)

Through such incidents, it is clear that an exploitative attitude toward the medical value of the poor went beyond the corpse trade and the Anatomy Act.

The patients and the working classes did not leave such exploitation unchallenged, however. The Edinburgh protests against Knox's participation in the Burke and Hare murders have already been discussed. Burrell and Gill (2005) describe spirited public demonstrations directed at hospitals during the Liverpool cholera epidemic of 1832. Convinced that cholera patients were being Burked and dissected in the hospitals established to combat the epidemic, angry crowds hurled epithets ("a Burker!"), mud, brickbats, and stones at hospital staff (485). In a subtler vein, patients also found ways to contravene hospital arrangements. In 1821, a GRI junior surgeon suggested the addition of ceiling ducts for ventilation as an improvement upon opening windows, which were "subject to the control or caprice of the patients, who shut them up at pleasure." (W. B. Gibbons to GRI Managers: HB14/2/6, 1) Gibbons reported "that in every hospital wherein such [ducts have] been adopted, [their benefits] have been made fully apparent," although he acknowledged that "In the Hilsea Depot Hospital, … the tubes had been artfully stopped by the patients," who it would seem found a means to control this system of ventilation to their liking as well (4)

Despite such contention between patients and practitioners, however, during this period echoes of older, more collaborative relationships do persist. Medical staff prescribed Charles S., treated for incipient phthisis in 1835, a combination of Dover's powder — a commonly prescribed diaphoretic of opium and ipecac — and squill, an expectorant (HB14/5/4, 31). They replaced this with a gentian tincture two weeks later, on November 13; two days later, the clerk noted, "Thinks he is not so well in respect to cough and expectoration since he gave up the powder." (35) On this — solely the patient's evaluation,

based on the preface "Thinks he" — clinicians promptly restarted the powder (ibid.). This type of interaction is uncharacteristic of the present period, however, and practitioner-patient relationships are more usually distant and even strained during this period.

CONCLUSIONS

The introduction of pathological disease concepts was made possible by broad medical access to the corpses of the poor for dissection; in hospital treatment at GRI, physical examination corresponding to the new disease concepts similarly displayed the living bodies of the working classes, such that the practitioner-patient relationship became marked by inequality, exploitation, and resistance. The Anatomy Act, following a decade behind the introduction of physical examination using the stethoscope and the newly popularized method of percussion, built upon a growing interest of medical men in pathological anatomy and its identification in the living and the dead. At the same time, these new methods of examination required greater access to patient bodies, both living — owing to the physical nature of such examinations — and dead, due to the necessity of connecting living observations to postmortem analysis. Both of these burdens were overwhelmingly borne by the poor, and practitioners justified doing so by new constructs of the recipients of charity as indebted to the public. It is likely that this exploitative relationship — of which patients were aware, and which patients resisted — contributed to the less collaborative relationship between patient and practitioner observed during this period and exaggerated in the periods that follow.

CHAPTER 5

JOHN HUGHES BENNETT, PHYSICAL DIAGNOSIS, AND THE POSSIBILITY OF CURE

Scottish medical practice during the mid-nineteenth century was marked by transitions. Older, essentialist concepts of disease were giving way to constructs linking externally observed signs and symptoms with internal pathology. At the same time, medical practice and training began to require greater anatomical knowledge, as well as expertise with new technologies like the stethoscope. Practitioners' relationships with the recipients of charity treatment were becoming more distant and strained, consistent with general attitudes toward the poor developing in Britain. The ways in which individual practitioners responded to these tensions was critical in shaping the direction such changes would take into the latter half of the nineteenth century.

One such practitioner was John Hughes Bennett, a physician and professor at RIE whose approach to medicine — traditional yet technologically advanced, anatomical while retaining essentialist ideas, simultaneously sympathetic to and distancing from charity patients — reflects the transitional nature of the mid-nineteenth century medical profession. Born in England in 1812, his medical education began in 1829 with an apprenticeship to a surgeon in Kent and continued with his matriculation to study medicine at Edinburgh in 1833. Graduating from Edinburgh with honors in 1837, he undertook two years of clinical study in Paris and two in "the principal University cities" of Germany ("Obituary: John Hughes Bennett" 1875, 473), likely some combination of Heidelberg, Baden Baden, Stuttgart, Halle, Berlin, and/or Vienna, where Bennett listed medical colleagues (1841, x).

Upon his return to Edinburgh in 1841, while working as an independent instructor in histology and the use of the microscope, he published his *Treatise on the Oleum Jecoris Aselli, or Cod Liver Oil* (Bennett 1841) introducing to the UK the treatment he had seen used with success in German cases of gout, rheumatism, rickets, and TB-related disorders including scrofula, "scrofulous caries of the bones" (i.e., bone and joint TB: xi), and phthisis pulmonalis. He served as pathologist of RIE from 1843 to 1848, in which capacity he performed by his own account more than 2,000 postmortem dissections (Bennett 1853, 5). He also began lecturing in medicine at this time, and in 1848 became a clinical professor of medicine at the University of Edinburgh and RIE, a position he held until resigning due to ill health in 1874. He died in 1875 shortly after a surgery to remove a urinary tract stone (Cadge 1875).

As a physician, Bennett was both ordinary and extraordinary. As I will discuss in this chapter, his published works and practice at RIE exemplified many of the tensions in early-Victorian charitable endeavors and medical practice, particularly with respect to TB-related disorders. The close examination I present here of his approaches to patients and medical practice, which were in many ways typical of Scottish medicine at the time, allows a more fine-grained analysis of these aspects of mid-nineteenth-century TB than is possible in my broader analyses of chapters four and six.

Bennett also atypically influenced both Victorian medicine in general and clinical constructs of TB and its analogues specifically. Lauded as a charismatic instructor by the author of his obituary, Bennett trained three decades of practitioners in clinical medicine and established at Edinburgh a methodical system of clinical training influenced by that he experienced in Paris ("Obituary: John Hughes Bennett" 1875, 473). Bennett's collected *Clinical Lectures on the Principles and Practice of Medicine* (e.g., Bennett 1858) — *Lectures on Clinical*

Medicine in its first edition (1850) — proved very popular, and had by the time of his death reached five UK editions and six United States editions, as well as having been translated into French, Russian, and Hindi ("Obituary: John Hughes Bennett" 1875, 476).

With respect to tubercular disorders, the cod liver oil treatment Bennett championed (1841) was revolutionary in that it targeted what many thought to be the underlying etiology of TB specifically rather than constitutional imbalance more generally. Cod liver oil and its successors, nutritional tonics such as Angier's Emulsion (Council on Pharmacy and Chemistry 1914) and Parrish's and Fellow's syrups (Reed 1998; Fellows 1882) remained popular in the treatment of TB and its analogues into the twentieth century, as I will discuss in chapter seven. Bennett's introduction of cod liver oil treatment and his works on the etiology, pathology, and diagnosis of tubercular disease made him a recognized authority on the topic: J. Russell Reynolds singled out Bennett to contribute the article on phthisis pulmonalis to Reynolds' sweeping *System of Medicine* (Bennett 1871).

As I will discuss in this chapter, Bennett respected traditional modes of medical practice, but he also expressed great optimism regarding the potential for new modes of treatment and the future contributions of scientific investigation to understanding, diagnosing, and treating disease. I begin with a discussion of Bennett's approaches to medicine and interactions with patients. I then explore Bennett's conception of TB and related disorders, the ways in which his views accorded with and diverged from those of his contemporaries, and his confidence in the curability of these diseases, particularly phthisis pulmonalis. Finally, I address his introduction of cod liver oil as a treatment for TB-related disorders, and his use of the treatment at RIE. Overall, the practice and writings of John Hughes Bennett illustrate the ways in which one practitioner responded to tensions in the Scottish medical profession — between external and internal modes of diagnosis, between

technology and tradition, and between patient and practitioner — at the mid-nineteenth century transition from the essentialism of the eighteenth century to the new disease constructs prominent during the nineteenth.

MEDICAL THEORY, TRAINING, AND PRACTICE

Bennett respected traditional, discursive modes of medical practice; at the same time, he embraced the new technology and pathological knowledge of the nineteenth century and eagerly anticipated medical developments to come. In addressing medical students, he endorsed the balance between theory and practice advocated by Cullen in the latter half of the eighteenth century (Bennett 1855, 10; 1858, 1). It was to this balance that Bennett credited Edinburgh's great success in medical training: "by a union of theoretical knowledge and practical skill, we may advance both to their farthest limits," he wrote in the introduction to the 1858 edition of his *Clinical Lectures on the Principles and Practice of Medicine*. "It is by cultivating medicine in this spirit that the clinical school of Edinburgh has rendered itself so famous …. Indeed, it is impossible to estimate too highly the advantages … [of] such a system, as it has been carried on … for one hundred and ten years." (Bennett 1858, 6)

At the same time, however, Bennett was aware of the mid-nineteenth-century shortcomings of existing theory and practice. He characterized medicine as an "inexact science," lacking a "primitive fact" comparable to gravity for the physical sciences. Because of this absence, he observed, "The same means, apparently, which operate at one time fail to do so at another." (Bennett 1858, 2) Despite his evident frustration, though, Bennett cultivated optimism that medical science would discover a unifying theory to revolutionize the field, telling his students:

> It is true that the contradictory character of medical doctrine has, in all times, excited the ridicule of the weak-minded, and still constitutes the ground on which Medicine is attacked by the ignorant and superficial. Yet … [a]ll these contradictions depend upon imperfect attempts at correct theory; and this latter once rendered perfect, it will be seen that both health and disease are governed by laws as determinate as the motion of the planets and the currents of the ocean. … Everything promises, that before long, a law of true harmony will be formed out of the discordant materials which surround us; and if *we*, your predecessors, have failed, to *you*, I trust, will belong the honour of building up a system of Medicine which, from its consistency, simplicity, and truth, may at the same time attract the confidence of the public, and command the respect of the scientific world. (1855, 22–23; emphasis retained)

For Bennett, in common with many of his contemporaries, knowledge of the body was the way forward for medicine. "Amidst the wreck of ancient [medical] systems," he cautioned students, "you will, I trust, adhere to that medicine which is based on Anatomy and Physiology." (1855, 23) These disciplines created a scientific basis for medical practice, to which Bennett attributed the major advances in medicine since Cullen's time (Bennett 1858, 1–2), and through which he expected practice to become more effective (4–5).

The basis of medical practice in scientifically observed fact required that students learn new ways of observing the patient. Medical practice comprised "the correct appreciation of actual facts" regarding the patient's condition joined with correct deduction from these of a diagnosis and course of treatment (Bennett 1858, 6). Proper medical observation required sense-training, however: defining as medical fact "anything which is obvious to the well-cultivated senses of the observer" (ibid.), Bennett cautioned, "Remark, I say *well-cultivated*, because the senses require to be educated before they can receive proper impressions." (7; emphasis retained) Only the trained observer could truly "see" the facts critical to medical practice.

Sensory education was not novel to mid-nineteenth-century medical training. In Susan Lawrence's account (1993), medical training in late eighteenth- and early nineteenth-

century London already emphasized perceptual education and sensory information, although in the late eighteenth century practitioners drew little distinction between the sensory knowledge of the practitioner and the patient (155). Rather, at that time the unique ability of the physician lay in the interpretation of the patient's narrative and external aspect (Porter and Porter 1989, 74–75).

Bennett emphasized the use of new technology alongside traditional modes of medical perspicuity to expand the practitioner's unique view of the patient. Following eighteenth-century traditions, he instructed students that interpreting the patient's narrative was a privilege of the practitioner: "Remember that the importance of particular symptoms is not known to the patient, and that, consequently, it is not in his power voluntarily to inform you of the necessary particulars. It is always your duty to discover them." (Bennett 1858, 20) To this he added the deeper knowledge of the patient made possible by new technology, extolling the virtues of "chemical tests, microscopes, stethoscopes, plexiметers, specula, etc." in facilitating the "detection of facts" and "carry[ing] observation … to its utmost extent." (7)

Carrying observation even further, and in keeping with the mid-nineteenth-century setting within which he practiced, Bennett also strongly advocated the postmortem dissection of patients. "When a patient dies, the examination is not completed," he informed students: "You should consider [inspection of the dead body] as a most important part of the clinical course. It is invariably regarded with the greatest interest by those who practise their profession with skill. It is only in this manner that any errors they have committed can be corrected; that the value of physical diagnosis can be demonstrated and properly appreciated, and the true nature of pathology of diseases, and the mode of treating them

rationally, can ever be discovered."[1] (Bennett 1858, 22) For Bennett, the value of postmortem dissection was threefold: to identify the cause of death, to evaluate the pathology associated with antemortem signs and symptoms, and to understand disease more broadly (23).

With respect to diagnosis, Bennett argued that postmortem dissection was critical to developing acumen in patient examination. With antemortem patient examination, the practitioner hypothesized internal pathology from externally detectable signs and symptoms; postmortem examination was the only method to test such hypotheses. Properly cultivating the senses required confirmation of physical examination findings: "an inspection of the dead body confirms or nullifies the diagnosis of the observer," (Bennett 1858, 22) allowing errors to be corrected in future cases. Bennett advocated going beyond mere confirmation, however, instructing students to avoid confirmation bias by dissecting the entire body rather than a limited area such as the thoracic cavity (23). Referencing his experience as RIE pathologist, Bennett offered as cautionary examples a number of ostensibly hypothetical cases in which a physician, having confirmed the presence of the disease to which he attributed the patient's death, ceased dissection — leaving Bennett to discover through his own, more complete dissection protocol "important lesions that were never suspected." (22).

Complete dissection was, for Bennett, necessary to the development of a scientific medicine. A thorough postmortem avoided confirmation bias; repeating Bennett's refrain that the profession was at the threshold of change, complete dissection also had the

[1] Following on my discussion of the stethoscope and physical examination in chapter four, it is interesting to note that Bennett includes justification of physical diagnosis among the benefits of dissection. It is evident that Bennett, at least, remained concerned that physical examination methods were not appreciated nearly forty years after Laënnec's introduction of the stethoscope.

potential to move beyond the individual case at hand to contribute to broader medical truths. Bennett argued that to do so was a duty, informing students:

> Nothing is more injurious to the scientific progress of medicine than the habit of examining only one [region of the body], to satisfy the curiosity of the practitioner, or to determine his doubts on this or that point. … [I]t must be obvious that this throws no light upon the nature of the disease, or its mode of cure. … [O]bserve the circumstances which produced [the lesion], as well as the symptoms and physical signs to which it gives rise, the secondary disorders, and the order of their sequence; their duration and mode of termination. This is the kind of extended investigation which can alone be serviceable to the advancement of medicine, and such, I trust, will be the object all of you will have in view in examining dead bodies. (Bennett 1858, 22–23)

Acknowledging that his students might be less interested in revolutionizing than practicing medicine, Bennett added, "At all events, such are the views that I shall constantly endeavour to place before you during this course of clinical instruction." (ibid.)

The mid-nineteenth-century practitioner in Bennett's view possessed unique understanding not only of the patient's experience — through the verbal examination inherited from the eighteenth century — but also of his or her body through physical and ultimately postmortem examination. Through examination, the practitioner thus gains knowledge of the patient beyond what the patient can know of him- or herself. In 1853's *Pathology and Treatment of Pulmonary Tuberculosis*, Bennett lamented that in treating this disease, the practitioner "struggle[d] not only with the deadly nature of the disorder" (80) but also with the patient's misunderstanding of his or her own health. The physician had "great difficulty … to convince the patient that, notwithstanding the removal of his urgent symptoms, the disease is not cured," (80) with RIE patients commonly leaving "in their own opinion perfectly cured, though assured to the contrary, and urged to remain." (81) The trained practitioner alone could uncover the occult truth of the patient's condition.

The skill to expose such truths through medical examination was critical for not only effective practice, but also professional status. "No doubt," Bennett informed students in 1858:

> questioning a patient, to arrive at a knowledge of his condition, requires as much skill in the medical practitioner, as examining a witness does in council at the bar. They make it an especial study, and you must do so likewise. You should remember that, in proportion as this duty is performed well or ill, is the probability that your opinion of the case may be correct or incorrect; and that, not only will the reputation you hold among your colleagues greatly depend on your ability in this matter, but that the public itself will promptly give its confidence to him whose interrogations reveal sagacity and talent. (17)

It is important to note that Bennett's use of the terms "questioning" and "interrogation" do not comprise only traditional, discursive modes of medical examination, and this too offers an indication of the transitional nature of mid-nineteenth-century practice. In the pages following the above passage, he refers to "interrogat[ing]" various bodily systems (18) and organs (20). Bennett thus classes both physical and verbal "questioning" together, likening both to legal examination. In so doing, he may have sought to align the increasingly hands-on practice of nineteenth-century physicians with the more genteel profession of law: mental, rather than manual, labor was salient in middle-class identity, and earlier generations of physicians had accordingly rejected physical examination, as I discussed in chapter three.

In order for medical examination to accomplish an accurate judgment of the patient's condition, Bennett taught that it, like postmortem dissection, must be complete and systematic. Following the plan of examination in which he was trained in Paris (Bennett 1858, 17), Bennett advised first identifying the seat and duration of disease by asking, "Where do you feel pain?" and "How long have you been ill?" (18). The practitioner should then examine the part indicated as being primarily affected, along with closely associated

regions of the body and followed by all other bodily systems — altogether comprising the circulatory, pulmonary, nervous, digestive, genitourinary, and integumentary systems — whether affected or not. After also interviewing the patient as to the past history of his or her case, the practitioner, Bennett declared with a touch of dramatic flourish, "shall have arrived at all the information necessary for the formation of a diagnosis." (ibid.)

Bennett's curriculum addressed social as well as mechanical aspects of patient examination, training students to be sensitive to their patients' emotions and perspectives. "It should never be forgotten," he instructed, that the patient "possesses the same sensitiveness to pain, and the same feelings as you do, and that everything that can increase the one or wound the other should be most carefully avoided." (Bennett 1858, 20) He recommended that practitioners tailor their language to their patients' understanding: "When an individual has a limited intelligence, or is accustomed to a particular dialect, you will not arrive at your object by becoming impatient, or talking in a loud voice, but by putting your interrogations in a clear manner, and in language proportioned to the intelligence of an individual." (ibid.) Indeed, Bennett further advised students to take into account cultural and gender differences in patient examination. "It is often necessary, after asking … 'Where do you feel pain?' to tell the patient to put his or her hand on the part. An Irish peasant applies the term 'heart' to an indefinite region, extending over great part of the chest and abdomen; and a woman, in speaking of pain in the stomach, often means the lower part of the abdomen." (ibid.)

Bennett taught that such sensitivity to the patient should be maintained even after death. In postmortem dissection:

> Great care should be taken never to disfigure the body. Incisions through the skin, therefore, should be made in such directions that when the edges are afterwards sewn together, the necessary dissections below may not be visible. Neither should

> the body be exposed more than is needful, and delicacy demands that the genitals should always be kept covered. The wishes and feelings of friends and relations should invariably be held in consideration. (1858, 24)

This contrasts sharply with the reported attitudes of Anatomy Act-era anatomists. In a confession to the Edinburgh *Evening Courant*, Burke, the anatomy murderer, described Edinburgh anatomist Knox and his students admiring the "handsome … well shaped" and nude body of a female victim, an incident also related in a published pamphlet attributed to a former employee of Knox (Richardson 1988, 135–6).

While Bennett's sensitivity to the concerns of patients and their survivors during postmortem dissection may be simply a nod to public sensibility, it is also possible that this represents a distinct change in attitude toward at least some anatomical subjects. The public perception of anatomists suffered a blow with the highly public Burke and Hare murders and the debate surrounding passage of the 1832 Anatomy Act. It would be natural for medical practitioners and anatomists, concerned with the political and social standing of their professions (e.g., Bennett 1858, 9–16) to respond with attempts to improve public opinion with respect to dissection such as assurances that subjects would be handled respectfully.

Indeed, medical staff frequently conducted postmortems at RIE and GRI both before and after passage of the Anatomy Act. Bennett's writing suggests that, at RIE, these were cases in which clinicians obtained permission to dissect from the patient's relatives (1858, 23), and that practitioners conducted such examinations on private patients (25) as well as those receiving charitable care. It may be that the circumstances of permitted postmortem examination inspired — or demanded — greater respect for the subject than anatomical dissection of unclaimed individuals.

Through passages urging sensitivity to patients' emotions and understanding in both life and death, Bennett reveals a continued affinity to older, collaborative patient – practitioner relationships. In light of these injunctions to maintain awareness of the patient's feelings, knowledge, and dialect, the physician's unique knowledge of the patient's body and history takes on the character of a responsibility rather than a right. Bennett's references to both limited intelligence and Irish peasantry connote working class patients, indicating that the "prudence, kindness, and delicacy" (1858, 20) he advocated were not recommended solely with respect to wealthier clients. Bennett's was not the only mid-century voice reflecting empathy between working-class patient and practitioner. Sydney Jones, a surgeon of London's St. Thomas' Hospital, shared with students the emotional reward of seeing a charity patient cured (1863), while an unnamed clerk transcribed on the title page GRI's March 1852 – December 1855 admissions register lines from Milton seeming to express his sympathy for patients' suffering:

> Dropsies and Asthmas and Joint racking rheums
> Dire was the tossing, deep the Groans: Despair
> Tended the sick, busiest from Couch to Couch;
> And over them triumphant, Death his dart
> Shook; but delayed to strike, tho' oft invoaked [*sic*]
> With vows as their chief good and final hope (HH67/56/19 1852, n.p.; Milton 1674, lines 488–93)

Paradise Lost goes on to ask, "Sight so deform what heart of Rock could long/Drie-ey'd behold? *Adam* could not, but wept." (Milton 1674, lines 494–5)[2] At the same time, however, the unique knowledge of the patient afforded the practitioner speaks to the division between patient and practitioner becoming characteristic of practitioner – patient relationships nearing the mid-nineteenth century.

[2] The empathy voiced here is somewhat eroded by these lines' context: only 20 lines later, Michael explains to Adam that the ill are being punished for their sins (Milton 1674, lines 515–25).

Further supporting a division between the practitioner and working-class patient in particular are subtle differences in Bennett's published accounts regarding private and charity hospital patients. As regards postmortem dissection, respect for the subject may have varied according to patient class: Bennett assured his students that while organs could be examined in situ — and generally would be so in private homes — at RIE he would remove pathological tissues and pass them around the lecture room for the students' edification (Bennett 1858, 25). While this distinction may owe more to the circumstances of death (in a teaching hospital as opposed to a private home) than the subject's social class per se, it nonetheless resulted in the public exposure of working-class bodies for the benefit of others, as the 1832 Anatomy Act had encouraged by law.

Similarly disparate exposure is evident in the case reports Bennett included in his works on TB-related diseases, *The Pathology and Treatment of Pulmonary Tuberculosis* (1853) and *The Pathology and Treatment of Pulmonary Consumption* (1859). In these two volumes, Bennett related the examination and treatment of 51 individuals, including cases observed by himself in private consultations and charity practice at RIE and the Edinburgh Dispensary as well as cases related to him from other practitioners' private practice. The cases included for publication, both public and private, were in some instances followed long term, and practitioner and patient evidently developed congenial relationships.

RIE medical staff treated Patrick B. for scrofulous disease of the femur, phthisis pulmonalis, and heart disease over several admissions from 1849 to 1852 (LHB1/129/2/5, – 7, – 9; Bennett 1853, 101–6; 1859, 160–67). Patrick B. returned to RIE twice while largely well, evidently to obtain advice as to whether his health was sufficiently sound to permit his traveling to join his sister in Philadelphia (Bennett 1853, 38; 1859, 165–66; LHB1/129/2/9

1852, 126). On the second occasion, he was examined by Bennett as well as "Dr Christison, the various clerks, and students" (Bennett 1859, 166); "He leaves for Philadelphia tomorrow," his RIE record concluded (LHB1/129/2/9 1853, 138). Bennett added in his 1859 volume that he provided Patrick B. with a letter of introduction to Dr. Wood, a Philadelphia medical professor (166). Dr. Wood reported, "I gave the boy [Patrick B.] … a small sum of money, telling him … to call on me again before long. He promised to do so. I have not seen him since." (ibid.) Upon receiving no further updates, Bennett made the extraordinary effort to follow up with a mutual friend three years later: "In a subsequent letter (1856) from Dr Dunglison, who at my request asked Dr Wood concerning him, it appeared that he had not since been heard of." (ibid.)

Bennett exhibited similarly continued interest in Robert K., whom he treated at RIE from August 1844 to January 1845 (Bennett 1853, 110–11; 1859, 171–73). This patient required two weeks of treatment before he could stand unassisted, but by January 1845, Bennett was dismayed to find that Robert K. had appeared so well to the committee of hospital managers touring the facility that he had been discharged in order to free his bed for more pressing cases (Bennett 1853, 111). However, Bennett was able to take advantage of a fortuitous opportunity to follow up with him:

> I lost sight of this man for eighteen months; but one day, in June 1846, I met him on the South Bridge, looking remarkably well. He told me that he had continued taking the oil [i.e., cod liver oil] for several months after leaving the Infirmary, and had obtained employment as a labourer on the North British Railway, where he was then employed. I took him into a common stair and examined his chest. … He stated that on going up a hill or a flight of stairs great breathlessness was excited, but that in every other respect he was in good health. (ibid.)

In the cases of both Patrick B. and Robert K., Bennett exhibited extended interest in the physical facts of their diseases — the spontaneous public-stairwell auscultation is

particularly delightful to imagine — but also appears to have been genuinely pleased at their continued good health. For their parts, each patient appeared to trust Bennett and voluntarily underwent examination outside the context of treatment in order that he and Bennett — and in Patrick B.'s case, Christison and a number of clerks and students — might be informed as to his condition. Patrick B., in particular, seems to agree with Bennett that the medical practitioner could know facts about the patient of which the patient himself was ignorant: rather than consult himself alone to determine whether he was well enough to emigrate, he consulted physicians.

Despite these notably congenial relationships, Bennett maintained a distinction in his publications between private and charity patients: while obscuring the identities of the former, he published the names of the latter in full. Of the 51 cases Bennett included for publication, 33 were treated in private consultation, 15 at RIE, and three at the dispensary (table 5.1). Whereas Bennett identified all 33 private patients and all three dispensary patients with truncated names (e.g., "Mr W.": Bennett 1853, 113) or descriptors (e.g., "The daughter of a medical man": Bennett 1859, 67), only three of the 15 RIE patients have their identities similarly concealed; the other twelve are identified by full name.

Table 5.1. Private and charity cases published by J.H. Bennett under full or obscured names

	Identification Type	
Treatment Location	Full Name	Obscured Name
Private	0	33
RIE	12	3
Dispensary	0	3
P-Value		1.169×10^{-07}***

Source: Bennett (1853; 1859)
*** $p < 0.001$

This distribution is very unlikely to have occurred by chance; although the sample is small, a Fisher test comparing the frequencies of identified and unidentified patients across private and charitable (RIE and dispensary patients combined) cases returned a *p*-value of less than 0.001 (table 5.1). The exceptional circumstances in which Bennett did obscure charitable patients' identities underscores his more standard practice of neglecting to do so. Of the six charitable patients (three RIE, three dispensary) whose identities Bennett obscured, five were somewhat unusual: four were included among a series of very brief descriptions of cases illustrating the mistaken diagnosis of TB-related disorders (Bennett 1859, 65–69) and one was a prostitute with syphilis (Bennett 1853, 128–30). The identities of this latter patient ("Margaret D—": ibid.) and the sixth charity patient with obscured identity ("Louisa —": ibid., 111) were less anonymous than those of private patients, with full first names given rather than simply initials (as "Mr W.," above) or a descriptor.

It is clear that Bennett, while maintaining involved, genial relationships with his charity patients as well as those in his private practice, distinguished between the privacy granted the two in publication. The identities of private patients he invariably protected, while those of charity patients he most often provided to readers in full. A similar dichotomy is evident in Bennett's espousal of sensitivity to patients' desires in close proximity to his promise to hand charity patients' organs around the lecture theater. While Bennett's intentions are, to all appearances, beneficent, his actions draw a sharp distinction between the working-class recipients of charitable medical care and their wealthier contemporaries.

Bennett's ambivalent approach to working-class patients highlights the class tensions of the mid-Victorian era, illustrating the conflict inherent in middle-class efforts to "improve" a group defined to be beneath themselves. It is tempting to attribute the

dichotomy of Bennett's treatment of poor vs. wealthier patients to the hardening of attitudes toward the poor evident in Britain during this period, as evidenced by the Anatomy Act (1832), Poor Law Amendment Act (1834), and Poor Law (Scotland) Act (1845). That is, Bennett publicly displayed the names and body parts of his poorer patients because recipients of charitable medical care owed their benefactors a debt to be repaid through medical advancement and education. In this instance, however, such a motive seems unlikely given Bennett's apparently even-handed attitude toward his working-class patients.

Alternatively, it is attractive to explain the distinction between private and charitable patients as the result of increasingly crystallized distinctions between the middle and working class in the mid-Victorian era. Perhaps Bennett simply did not recognize an equal right to, or desire for, privacy among the working classes and those of higher socioeconomic status. He may have believed that broad knowledge of their illness would be more damaging to the reputations or respectability of his private patients, as these individuals he may have perceived as being more negatively affected by public perceptions. These individuals may also have been more likely to encounter socially the medical readers of his publications, occasioning embarrassment. Indeed, Bennett may have sought to protect his own professional reputation, as fee-paying patients may have chosen to avoid a physician likely to describe their health problems in print. It is difficult to distinguish among these possibilities given the information available, but the fact remains that patients receiving charitable care were subject to public exposure from which Bennett protected higher-class patients, in keeping with broader attitudes toward the poor during Bennett's career.

Bennett's attitudes toward patients — as with his perspectives on anatomy, physical diagnosis, the state of medical knowledge, and the uniquely perceptive lens through which practitioners viewed patients — encapsulate the transitions in medical practice of the mid-

nineteenth century. Physical diagnosis, while widely lauded as the future of medicine, continued to be resisted by the old guard. At the same time, more forward-thinking practitioners embraced the techniques, while acknowledging the difficulty in teaching and learning them as well as the shortcomings in pathological knowledge that prevented physicians from maximizing the diagnostic value of new technology. Despite the newly detailed anatomical portrait skilled practitioners could develop of a patient using a combination of older, discursive examination and new physical techniques, Bennett and his contemporaries remained incapable of effectively treating the diseases they could now diagnose more precisely. This was nowhere more apparent than in the case of TB and related disorders, a particular theoretical and therapeutic focus of Bennett.

TB AS A NUTRITIONAL DISORDER

By Bennett's time, medical practitioners had long considered TB and its historical analogues, particularly phthisis pulmonalis or pulmonary consumption, to be essential to the sufferer, arising from emotional imbalance or constitutional predisposition. Laënnec cited "depressing passions" as a cause of phthisis in Forbes's 1834 translation of *A Treatise on Diseases of the Chest*: "Among the occasional causes of phthisis, I know of none more assured operation than the depressing passions, particularly if strong and of long continuance" (Laënnec and Laënnec 1831, v. 2 119; Laënnec 1834, 302).[3] "The influence of the depressing passions in giving rise to diseases of the lungs, and particularly phthisis, has been noticed by many writers," agreed Forbes in a footnote (Laënnec 1834, 303: footnote by Forbes), citing both Morton (1720) and Auenbrugger (1824) on the subject.

[3] As in chapter four, quotations of Laënnec's works are from Forbes's translations for which I verified the accuracy by reference to the original French; for these and other specific passages I have cited, I include page numbers for the pertinent content in both the original French works and Forbes's English translations.

In the mid-1830s, medical writers similarly presented as settled the existence of a constitutional predisposition to phthisis. In response to Laënnec's discussion of the role of inflammation in the development of tubercles (1831, v. 2 57; 1834, 278), his nephew Dr. Meriadec Laënnec commented in an updated footnote, "The discussion in the text respecting the inflammatory or non-inflammatory origin of tubercles in the lungs, is now become idle, since all good observers are of accord on this point — that they are in all cases the consequence of a *predisposition* either congenital or acquired. … The only thing of importance is to … ascertain the predisposing causes of phthisis, as it is on this knowledge alone that any rational treatment of this dreadful disease can be founded." (ibid., footnote by M. Laënnec; emphasis retained) In the 1830s, such causes were both widely accepted and endorsed by sources considered authoritative on the subject of consumption.

By the mid-nineteenth century, Bennett's clinical observations had led him to diverge from these older, authoritative causes of tubercular disease, instead espousing a nutritional basis for these disorders. "The more [cases] are examined into," he wrote in his 1853 treatise on TB, "the more do I feel persuaded it will appear that the causes of phthisis are not hereditary influences, vitiated atmosphere, etc. etc., although *these may cooperate*, but almost invariably such circumstances as induce impoverished nutrition, resulting from an improper quantity, quality, or assimilation of food." (54; emphasis retained) Bennett argued that viewing nutrition as the ultimate cause of TB could explain salient aspects of the temporal and socioeconomic distribution of the disease:

> An observation of the circumstances which precede the disease, or its so-called causes, clearly indicate imperfect digestion and assimilation as its true origin. Thus phthisis is essentially a disorder of childhood and youth — that is, of a period of life when nutrition is directed to building up the tissues of the body. Diminish the proper quantity of food taken by a healthy man, tubercular diseases are not induced, but if this be attempted with children or young persons, they are a most common result. Thus scrofula and tubercle do not originate among the *able-bodied* men in

> armies and fleets, whatever [their] privations … but they may be observed to do so in the young of foundling hospitals, factories, and the poor and labouring classes of the community, and especially among tailors, sempstresses [i.e., seamstresses], and others who follow sedentary employments. (Bennett 1853, 52; emphasis retained)

What the nutritional etiology failed to do, however, was explain the presence, albeit less frequent, of tubercular disease among well-nourished classes of the population. As the extended quotation above indicates, among the lower classes Bennett attributed these disorders to inadequate food intake, particularly during development. This would seem to exclude the better-fed middle and particularly upper classes from susceptibility to the disease, necessitating a special explanation for the disorder among these groups. Bennett provided several: "In the higher classes [tubercular diseases] result from imperfect and insufficient lactation during infancy, or the irregular diet caused by carelessness or over-indulgence. No doubt they may frequently be observed in persons whose parents or relatives have been similarly affected." (Bennett 1853, 52)

Indeed, Bennett thus offered sharply divergent causes for tubercular disease among the lower and upper classes. "So long as misery and poverty exist on the one hand," he explained, "and dissipation and ennervating luxuries on the other, so long will the causes be in operation which induce this terrible disease." (Bennett 1853, 58) It is worth noting that these disparate sets of causes — misery and poverty, and dissipation and enervation — have distinct class connotations, the former working-class privations and the latter upper-class indulgences, suggesting Bennett was of the opinion that the middle classes ought not to suffer from tubercular disorders.

Such nutritional explanations for TB and its analogues were not unique to Bennett. While placing more emphasis on predisposition to TB-related disorders, Laënnec himself suggested the involvement of "an unknown alteration in the assimilation or nutrition" in the

disease (1831, v. 2 190; 1834, 339), as I have mentioned. Such early suggestions of nutrition's contribution to tubercular disease and to disease more generally, such as Bennett's 1842 paper on the relationship between "anormal nutrition" and tissue pathology (cited in "Obituary: John Hughes Bennett" 1875, 474), would have been bolstered by the European potato failure and the resulting "Hungry Forties." These likely drew medical attention to the effects of under- and malnutrition, such that by the publication of an 1855 *Lancet* report on the practice of James Edward Pollock of the Brompton Hospital for Consumption in London, the anonymous author described himself as convinced "that tubercle is a blood disease associated with … alterations of nutrition under depressed nervous influences" (388).

It is easy to see why the nutritional etiology gained broad support during the mid-nineteenth century. As a disorder of nutrition, tubercular disease retained an aura of essentialism; pulmonary tuberculosis was not a lung disease, but the local expression of a constitutional affection. This reinforced the long-recognized associations among scrofula, tuberculous caries of the bones and joints, and pulmonary consumption, as well as the new understanding that tubercles throughout the body resulted from a common cause. At the same time, it renewed the association between these disorders and TB's once-primary signifier, bodily wasting. As a result, the nutritional etiology favored by Bennett and his contemporaries linked traditional and newer understandings of the disease in a single explanatory framework.

COD LIVER OIL AND THE QUESTION OF CURABILITY

A preoccupying and longstanding question for the medical minds of this period, regardless of the postulated source of the disease, was whether TB-related disorders could be cured. An important distinction in this debate was the natural, as opposed to medical,

curability of these diseases. Many — but perhaps not all — mid-nineteenth-century medical authors agreed that recovery from consumption was possible. However, the problem of whether medical practitioners could effect recovery, and if so, what course of treatment should be employed to do so, remained contested by medical writers well beyond this period. "Is Consumption Curable?" asked the title of an anonymous 1887 *Science* article discussing the prospects for treatment in light of Koch's (1882a) research on the tubercle bacillus.

Laënnec was equivocal on the question of medical cure. "It is now … the general opinion of all those who are acquainted with the … pathology of diseases, that the tubercular affection, like cancer, is absolutely incurable, inasmuch as nature's efforts towards effecting a cure are injurious, and those of art useless," he stated (1831, v. 2 58–59; 1831, 279). Shortly thereafter, however, he added: "But while I admit the incurability of consumption in the early stages, I am convinced … that, in some cases, the disease is curable in the latter stages, that is, *after* the softening of the tubercles and the formation of an ulcerous excavation." (ibid.; emphasis retained)

For Forbes, cure was impossible, and medicine should instead focus on prevention of tubercular disease. "The result of all our knowledge of the pathology of phthisis, and of all our experience in the treatment of it, leading to the conclusion, that it is incurable by art, after tubercles are developed, — the only part of the subject that is really of any practical importance is, the plan to be adopted in individuals predisposed to the disease, with the view of obviating the formation of these extraneous bodies" (i.e., tubercles), he wrote (Laënnec 1834, 339: footnote by Forbes). "Indeed, all our facts and reasoning point not merely to the necessity of watching the very first and slightest symptoms of incipient consumption, but of

subjecting every child which seems predisposed to it … to a most rigid system of prophylactic discipline." (ibid.)

Bennett decried the notion that tuberculosis inevitably resulted in death. "So deeply rooted … has been the opinion of the necessarily fatal nature of this disease," he commented, "that the generality of practitioners have concluded, that, *because* phthisical cases recovered, that the disease was *not* phthisis; that is, they have rather distrusted their own diagnosis than ventured to oppose a dogma of general belief." (1853, 52; emphasis retained) Rather, he argued, the disease must be eminently curable by medicine, because cure occurred naturally: "Phthisis, in its incipient stage, may be considered a very curable disease; indeed, so much so, that cure is, as we have seen, spontaneously accomplished by nature, in a vast number of cases." (58)

In Bennett's view, early detection was critical to the possibility of cure. "[T]he general notion of [phthisis'] incurability is mainly attributable to the fact, that it is not recognised until it be far advanced," he lamented (1853, 54). Describing a case in which the chest was never examined, and thus phthisis never diagnosed, he remarked, "had the diagnosis of the disease been properly established at an early period, its onward march might have been arrested." (57) Thus, for Bennett, tubercular disease was most curable in the early stages: diagnosis of such disorders only in their later stages contributed to the mistaken belief that they were invariably fatal.

Accordingly, Bennett advocated the development of diagnostic skill in practitioners, in both his publications on tubercular disease and his teaching. The first section of his *Clinical Lectures on the Principles and Practice of Medicine* (1858) is dedicated to patient examination; while Bennett strongly encouraged the use of new techniques and technology — percussion, auscultation, microscopic observation — he also instructed students to

cultivate their skill in older, discursive methods. In diagnosing phthisis pulmonalis, Bennett continued to cite the utility of symptoms such as reduced appetite, acidity, and bowel irregularities in identifying the disease in its early stages (1858, 674–75). "Auscultation is only *one* of the means whereby we can arrive at a just diagnosis," he reminded students in speaking of the use of the stethoscope generally, "and should never be depended on alone." (56) Accurate diagnosis, in TB specifically and disease more generally, required the assimilation of data from traditional and new sources.

Although he cautioned students against relying on the stethoscope alone, Bennett clearly favored such new methods himself. Improper examination of the chest, or lack thereof, is a common theme in his discussion of misdiagnosed phthisis patients — that is, both patients wrongly considered to have phthisis and those with phthisis diagnosed as some other disorder (e.g., Bennett 1853, 54–57). Despite offering a brief mention of "the well-known general symptoms," Bennett nonetheless emphasized auscultatory signs in his criticism of those who fail to diagnose the disease in its early, more curable stages: "there is, perhaps, no disease which, by one practised in auscultation, may be more readily detected." (1853, 54)

Bennett's endorsement of percussion and auscultation went so far as to privilege practical training in physical examination above the classical scholarship that was a traditional hallmark of physician training. "[L]et no one undervalue percussion and auscultation," Bennett railed, excoriating writers who mocked practically trained medical students familiar with the gamut of auscultatory signs but unable to fathom Plato (1853, 58). "I am of the opinion that, if our university graduates can detect the *râles* above alluded to, and know their diagnostic value, it must be a matter of comparative indifference to mankind

whether they are able to 'unsphere the spirit of Plato,' or not." (58–59) In so stating, he may have expressed a view shared among Scottish medical instructors: Scottish medical training had long emphasized practical skills — training physicians in surgery, midwifery and pharmacy as well as medicine since the 1600s (Jenkinson 2012, 4) — and instruction at Edinburgh had been conducted in English rather than Latin since the late eighteenth century (Lawrence 2006, 214).

Indeed, it was practitioners' new emphasis on physical diagnosis and pathological anatomy that brought the question of phthisis' curability to the forefront during the early- to mid-nineteenth century. In 1841, Bennett remarked that Laënnec and other French practitioners had demonstrated beyond reasonable doubt the presence of healed TB lesions in the lungs (131). This he credited exclusively to anatomical study, commenting in 1853 that the recovery of pulmonary TB patients had "been established rather by the evidence of morbid anatomy than by the flattering accounts of individual practitioners." (125) He outlined the case for the curability of phthisis carefully and logically for his students in his 1858 textbook. Bennett demonstrated by analogies of substance and location that the "cretaceous and calcareous concretions, accompanied with puckerings" (670) observed in postmortem dissections of the lungs were in fact arrested tubercles, and that even advanced ulceration of the lungs showed postmortem evidence of healing (670–74).

Postmortem findings of healed or quiescent tubercular lesions bolstered claims during this period that tubercular disease could be cured, although the question remained how best a cure might be effected. In accordance with early- to mid-nineteenth century conceptions of TB and related disorders as constitutional, rather than local, diseases, those authors who admitted the possibility of medical cure suggested treatments should target the

constitutional defect at the root of the disorder. "In order to make a direct attack upon the disease, we ought probably to be able to correct an unknown alteration in the assimilation or nutrition," Laënnec ventured (1831, v. 2 190; 1834, 338–39). Similarly, the *Lancet* reporter on Pollock's Brompton Hospital practice suggested that natural cure occurred only when "the original [nutrition-related] blood disorder and blood contamination should have ceased," (1855, 389) implying that medical cures must also target this root cause.

In keeping with both his esteem for pathological and physiological approaches and his desire to restructure medical practice along more rational lines, Bennett favored medical treatments addressing the physiological basis of disease. Bennett expressed pessimism as to the effectiveness of drugs as then prescribed, and was particularly critical of bloodletting and what he saw as the overuse of mercury ("Obituary: John Hughes Bennett" 1875, 477). He dismissed such prevailing treatments as "discordant with [medical] theory" and "feeble or altogether inert" with respect to the pathological processes they purported to treat (1858, 9). Accordingly, he too favored constitutional rather than local treatment for TB, setting out three principles for successful therapy: "1st, that tubercular diseases will heal of themselves, if the faulty nutrition of the system can be removed; 2ndly, that with this object our efforts should be directed to the digestive rather than to the respiratory system; and 3rdly, that the kind of abnormal nutrition which exists is dependent on increased assimilation of the albuminous, and diminished assimilation of the fatty portion of the food." (1853, 9) Indeed, his emphasis on supplementing the patient's nutrition rather than applying depleting therapies such as bloodletting presaged both the rich diets typical of sanatorium treatment (e.g., Bardswell and Chapman 1908, 35–38) and today's emphasis on malnutrition as a contributing factor in developing tubercular disease (e.g., Paton et al. 2004).

For Bennett, the treatment that fulfilled these requirements was cod liver oil. Having observed the successful use of cod liver oil to treat scrofula, gout, and rheumatism during his studies in Germany, Bennett published a treatise on the substance upon his return to Scotland (Bennett 1841, vii). The medicinal use of cod liver oil was not unknown in the UK at the time — Bennett offered instances of its prior use in both folk and medical treatment for rickets and rheumatism (13–15) — but he intended to popularize the treatment (vii). Bennett's treatise outlined the use of cod liver oil in treating a wide range of diseases, but it was TB and related disorders for which he would emphasize its use in later publications.

With respect to tubercular disease, Bennett explained that cod liver oil functioned to correct deficiencies or inadequate absorption of oils in the diet. He taught that excess acidity in the alimentary canal rendered the digestive system incapable of either absorbing oil from the diet or producing it from other foods; this deficiency of fats generated a corresponding excess of albumen (protein) that was deposited in the lungs or other organs as tubercle (Bennett 1858, 677). The system turned to its own fat stores to provide the necessary substance, resulting in emaciation (ibid.). In some cases, the deficiency could be corrected by simply increasing dietary fat, leading, Bennett explained, to traditional cures such as asses' milk, dairy products, and fatty meats (678). Where the digestive system had become deranged or weakened, however, it was necessary to provide oil directly for rapid assimilation, and this was the strength of cod liver oil (ibid.). This accorded well with Bennett's promotion of treatments guided by physiology and pathology, and upon his introduction of the treatment in 1841, he expressed cautious optimism that cod liver oil might prove effective in broader trials (Bennett 1841, 152).

As evidenced by both his published accounts and ward journals, Bennett employed cod liver oil frequently in cases of TB-related disorders at RIE from the 1840s onward. He

acknowledged that patients may resist the substance due to its taste, but concluded that Britons "will swallow any substance which holds out the hopes of cure, after having suffered a few years" with rheumatism, gout, or tubercular disease (1841, 64). He further offered the somewhat dubious assertion, "Children soon become accustomed to it, and take it readily." (ibid.) He initially reported a usual adult dose of one to two tablespoonfuls up to four times daily (61), which by 1858 he had amended to one tablespoonful three to six times daily.

Such dosages would be considered toxic by modern standards: a single modern tablespoonful of cod liver oil contains 136 percent of the tolerable upper intake level of vitamin A and 34 percent of this level for vitamin D (United States Department of Agriculture 2011; Office of Dietary Supplements 2012; Office of Dietary Supplements 2011). Indeed, patients in my sample taking large doses of cod liver oil commonly developed nausea, vomiting, and other digestive symptoms; medical staff attributed this to the patient not tolerating the treatment, and may have resulted from hypervitaminosis A. Practitioners prescribed George R., for example, one tablespoonful of cod liver oil thrice daily beginning on January 2, 1865; one week later, on January 9, the clerk recorded that the patient "states that the cod liver oil is beginning to disagree with him." (LHB1/129/2/60, 140–41)

Medical staff did not use cod liver oil in every case of tubercular disease treated at RIE. In a sample of 55 TB-related cases treated at RIE between 1845 and 1875 for which the physician of record could be identified with confidence, 34 (61.8 percent) were treated with cod liver oil. Bennett was the physician of record for 32 of these (table 5.2). Bennett prescribed cod liver oil for two-thirds of his TB-related cases. It is unclear why he did not do so for all cases, but it may be that he considered the treatment contraindicated in patients with diarrhea and/or vomiting: these patients would likely be unable to retain the oil long enough to benefit from it.

Table 5.2. J.H. Bennett's and other physicians' treatment of TB-related disorders with cod liver oil

	Treatment		
Physician	Cod Liver Oil	No Cod Liver Oil	TOTAL
Bennett	32	16	48
Other	2	5	7
TOTAL	34	21	55

Note: P-value for the two-tailed Fisher exact test of differences is 0.0924.

Other physicians of record at RIE appear to have been somewhat less keen on the treatment than Bennett. In his 1858 textbook, Bennett criticized his colleagues for not prescribing cod liver oil. In the fatal 1851 phthisis case of Walter C., for example, "treatment … was conducted by my colleagues for four months before I saw him, on the palliative plan; and I may appeal to the facts it presents, in proof that such treatment produced no effect in any way checking the progress of the disease. In this respect it offers a marked contrast to … cases, in which the treatment was directed by the pathological principles … and had for its object increasing the nutritive powers," i.e., treatment with cod liver oil (Bennett 1858, 665).

This complaint is borne out in my sample to some extent. Only two of seven (28.6 percent) TB-related cases for which Bennett was not the physician of record received cod liver oil treatment (table 5.2). The distinction between cod liver oil prescribed in cases treated by Bennett and by other physicians is not significant at $p < 0.05$; however, at $p = 0.0924$, it does approach significance at $p < 0.1$. This lack of significance may simply be a result of the small number of TB-related cases for which I was able to identify the physician of record overall, or of the small number I identified with physicians of record other than Bennett. Alternatively, it may be that at least some other physicians practicing at RIE were

convinced of the therapeutic value of cod liver oil in cases of tubercular disease and did not prescribe it less often than Bennett himself.

In the treatment of TB-related disorders at RIE, the cod liver oil treatment Bennett championed was employed variably, even by Bennett himself. This suggests, as Bennett indicated in his 1858 text, that RIE physicians differed as to the best mode of care for these diseases. Bennett's endorsement of cod liver oil was rooted in his ascription to a nutritional etiology for these disorders, as well as his personal observation of outcomes of the treatment, first in Germany and later by himself. Despite Bennett's strong endorsement of the treatment, however, the prescription of cod liver oil at RIE contributed little to improving patient outcomes, as I will discuss in chapter six. Furthermore, neither a nutritional cause nor a nutritional cure for TB-related diseases was universally accepted even within the faculty of Edinburgh — evidence that a mosaic of medical constructs of these diseases was present even within a single infirmary during the mid-nineteenth century. Opinions among medical practitioners on the subject of phthisis' curability continued to differ well toward the turn of the century, suggesting that such polyvalent conceptions of TB-related disorders would continue for some time.

CONCLUSIONS

At the transition from essentialist to anatomical concepts of TB and related disorders, the Scottish medical profession was in a state of transition between discursive and physical diagnosis, between new and old modes of observation, and between collaborative and distant patient – practitioner interactions. The practice and writings of John Hughes Bennett in many ways encapsulate the tensions of medical practice during this mid-nineteenth-century period of change. Espousing patient sympathy while displaying charity

patients publicly and embracing new technology and physical diagnosis while continuing to support older, discursive modes of diagnosis, Bennett was in many ways exemplary of his time. Frustrated at the slow pace at which medical treatment followed pathological and physiological understandings of disease, he sought to introduce new treatments and encourage the use of technology in diagnosis. Despite Bennett's stature in the medical profession, however, such developments were not accepted universally, and medical practice would maintain conflicts regarding the causes of tubercular disease and its curability and treatment for many years to come.

CHAPTER 6

PHYSICAL DISEASE AND SOCIAL PATHOLOGY, 1841 – 1880

In chapter four, I addressed the adoption of physical diagnosis at GRI, a change coincident with and reliant upon medical access to poor patients' bodies both before and after death, and marked by increasing distance between patient and practitioner in patient case notes. Despite the near-universal use of auscultation and percussion at Glasgow during the period 1821 – 1840, however, practitioners' conversion to the new, physically and anatomically defined construct of TB and related disorders espoused by Laënnec (1831; 1834) and others remained incomplete. Rather, in diagnosing TB-related diseases, medical staff continued to use signs and symptoms characteristic of older, essentialist concepts of these disorders alongside symptoms observed through auscultation and percussion.

As I discussed in chapter five, the writings and practice at RIE of John Hughes Bennett from 1841 to 1874 exemplify the transitions and tensions associated with the advent of physical examination and widespread anatomical dissection. Although Bennett favored new technology and a new approach to TB treatment, his ideas about tubercular disease and cod liver oil were not universally accepted; the larger medical revolution he foresaw (Bennett 1855, 23) was similarly slow in coming. Bennett furthermore forged sometimes lasting, evidently congenial relationships with his charity patients, while nonetheless publicly revealing their medical details and their dissected body parts in ways he did not for wealthier patients. He thus reflected the ambivalence toward the working classes prevalent throughout the United Kingdom at the time, in which the middle classes' keen interest in "improving" the poor was accompanied by legislation such as the Anatomy Act (1832), Poor Law

Amendment Act (1834), and Poor Law (Scotland) Act (1845) that was increasingly punitive toward the recipients of public assistance.

In this chapter, I examine the ongoing development of medical authority, physical examination and constructs of TB-related disorders at GRI and RIE. My analysis of 45,135 admissions (GRI n = 15,867, RIE n = 29,268) and the case notes of 526 individuals (GRI n = 109, RIE n = 417) (table 6.1) shows that during the period 1841 through 1880 practitioner-patient relationships became increasingly marked by medical staff's exertion of authority over patients. At the same time, practitioners relied more strongly on technologically acquired clinical signs in diagnosing TB-related disorders; however, this did not preclude practitioners' still-continued use of the patient-reported symptoms that had long defined these diseases. As a result, constructs of TB and analogous disorders — including the nutritional etiology championed by Bennett and others — retained a stamp of essentialism. Coupled with mid-Victorian behavioral proscriptions and pathological conceptions of gender and class, this essentialism resulted in a new medical emphasis upon the contributions of immorality to ill-health, bolstering a growing conviction among the middle class that women and the working class posed a danger to the health of the social body.

PATIENTS, PRACTITIONERS, AND THE ROLE OF MEDICINE

As I have discussed in chapters three and four, GRI records from 1794 through 1840 reveal a growing clinical distance between patient and practitioner. While the earliest case notes show little distinction between practitioner and patient accounts and few instances of contention, in cases from 1821 through 1840 practitioners are more apt to distinguish their own statements from those of patients and to comment on patient behavior, although some

instances of a more collaborative relationship remain. These trends continue during the mid-Victorian period I address here, alongside evidence of an increasing sense of the importance of medical treatment in the minds of both patients and practitioners.

Table 6.1. GRI and RIE admission registers and case notes samples, 1841 – 1880

	GRI		RIE	
Diagnosis Groups	Admission Registers[a]	Casenotes[b]	Admission Registers[c]	Casenotes[d]
TB-Related	944	55	1861	211
Non-TB Pulmonary	1283	24	2484	94
Other	13640	30	24061	112
Total	15867	109	29268	417

[a] Sampled years include 1845, 1855, 1865 and 1875.
[b] Sampled years include 1860, 1870, 1875 and 1879.
[c] Sampled years include 1845, 1850, 1855, 1860, 1865, 1869 and 1875.
[d] Sampled years include 1845 – 47, 1850, 1855, 1860, 1865, 1869 and 1874 – 75.

THE IMPORTANCE OF MEDICAL CARE

During the period from 1841 through 1880, patients displayed increasing willingness to undergo medical treatment, in contrast to the fear and loathing with which hospitals were regarded in the period surrounding the 1832 Anatomy Act. Although an 1855 committee of contributors to RIE described to the board of management "an unwillingness on the part of the poorer classes to avail themselves of the benefits of the infirmary," they observed that "this feeling is gradually losing ground and as it is removed it is to be expected that the patients will apply to the infirmary in an earlier stage of disease."[1] (LHB1/1/18, 365) Despite this admittedly tepid description of working-class fervor for the infirmary, these classes did

[1] The committee of RIE contributors was ambivalent toward the growing willingness for the working-class to undergo infirmary treatment. Patients delaying admission "until it is too late to be of advantage" (LHB1/1/18: 365) resulted in shorter, less expensive stays, whereas earlier treatment entailed longer stays but would be less likely to end in the patients' deaths.

demonstrate interest in the institution's continuation: in the same year, donations to the infirmary included forty-four pounds, three shillings and sixpence from "Enginemen, firemen and workmen in locomotive department of North British Railway."[2] (486)

Indeed, some patients anxiously sought infirmary assistance, their concerns surrounding the hospital perhaps allayed by a greater fear of the poorhouse, previous positive experiences of treatment, or from financial and practical roadblocks to home treatment. The RIE clerk noted that Owen G., an 1855 phthisis patient, "is in great alarm interrupts enquiry by asking if he shall die — if there has ever been a case such as his — if he is to get any medicine — declares that if he doesn't get medicine he must go out — that if the doctors don't do something for him he doesn't know what is to become of him." (LHB1/129/2/12, 72) Other patients received ongoing medical treatment; patients at both GRI and RIE reported previous admissions to those institutions, as well as treatment from other sources. In chapter five, I discussed Patrick B., whom RIE medical staff treated multiple times between 1849 and 1852 (LHB1/129/2/5, –7, –9; Bennett 1853, 101–106), and Robert K., who allowed Bennett to examine his lungs in a stairwell eighteen months after his discharge from RIE (Bennett 1853, 110–11). The mother of William P. — an Edinburgh child treated for tapeworm — described his having received treatment as an outpatient at RIE as well as from the doctor of her husband's regiment (LHB1/129/2/12 1855, 87–88). Although the prescription provided by the former had proven successful several times, she sought admission at the infirmary "partly because the medicine is too dear

[2] This was a donation rather than a subscription that would have entitled them to hospital treatment; the workers' motive was not recorded, but the gift may have related to the large number of railway laborers cured of scurvy at RIE during the late 1840s. For example, in my complete sample of the journal of Clinical Medical Ward 1 — in which I included every third case — four of 18 sampled cases admitted between January 25 and May 27, 1847 were scorbutic railway laborers (LHB1/129/2/1, front matter).

for her to buy, and partly because she is not successful in getting the child to take physic." (ibid.)

Prior treatment was not always by orthodox practitioners, however. William P.'s mother had also dosed him with laxatives "(recommended by a female friend)" (ibid.); James B., an RIE pneumonia patient, reported having had "Mustard plaisters [*sic*] applied over back and front of chest and a dose of senna and salts [a laxative] administered by his landlady." (LHB1/129/2/71 1869, 90). Of George M., suffering from sexual hypochondriasis, the RIE clerk noted, "'Two years ago he was thrown into low spirits by reading a quack treatise entitled 'The Errors of Youth' …. He has been treated by numerous medical men and also been in the hands of all sorts of quacks" (LHB1/129/2/26 1860, 66).

Perhaps to counter the willingness of their patients to be treated outside the auspices of the infirmary, the GRI board of management waxed enthusiastic about their working-class subscribers. The board unanimously approved an 1865 motion "that a special vote of thanks be awarded … to the working-classes, warehouse employ'es [*sic*], and seamen, for their continued and liberal subscriptions in aid of this institution." (HB14/1/13, 10) Working-class contributors were specifically touted in the GRI annual reports of 1865 and 1875, albeit in a manner intended to reassure middle-class donors that those benefitting from hospital treatment were both grateful and worthy. The 1865 report acknowledged, "'To the working-classes [*sic*] the Institution is deeply indebted for the constant and liberal support which it has received from them," followed by the paternalistic note, "the Managers trust that the efforts of these classes will not be relaxed." (HB14/2/7, 9) A decade later, the report described "the strong hold which the Institution has on the Working Classes, and the interest which they take in it as evidenced by their contributions" (HB14/2/8, 8) — i.e., they earned enough, and were public-minded enough, to make their own contributions.

THE PRACTITIONER – PATIENT RELATIONSHIP

Practitioners, too, exhibited some esteem for patients, demonstrating like Bennett obliging relationships at times. Disabled patients could be accommodated gracefully. Peter McG., a Glasgow phthisis patient, was "deaf and dumb," a fact the clerk only considered notable for the change it necessitated to the standard auscultatory examination: in being assessed for vocal resonance, Peter McG. spoke his name rather than the usual "twenty-one – twenty-two" owing to his speech difficulty (HH67/1/1 1870, 258). The RIE clerk noted at the opening of Jane B.'s record, "This patient is deaf and dumb since infancy, but appears to be rather intelligent. She expresses herself and answers questions by writing on a slate."[3] (LHB1/129/5/20, 60) The case reports of Peter McG. and Jane B. are otherwise indiscernible from those of hearing patients, suggesting a matter-of-fact approach by practitioners to patients' special needs.

In some cases, medical staff at RIE went beyond infirmary regulations to assist their poorest patients. Both GRI and RIE were intended by their founders and boards of management to serve the working poor, with the health of "paupers" the responsibility of the parish. The distinction between paupers and the "worthy poor" was fluid, however, and became more consequential with the Poor Law (Scotland) Act of 1845. This law upheld the prohibition against parish assistance to the able-bodied, a feature of Scottish poor laws since the fifteenth century, while encouraging parochial boards to establish poorhouses like those of the 1832 Poor Law Amendment Act of England and Wales. That is, after 1845 the able-bodied continued to be refused parish relief, while paupers were increasingly subjected to punitive poorhouses.

[3] The comment, "but appears to be rather intelligent," while patronizing, was not isolated to this disabled individual: for example, Mary McQ., an RIE chronic pneumonia patient, was described as exhibiting "even less intelligence than usual among her class." (LHB1/129/5/22, 1855: 68)

Patients not uncommonly bestrode the boundary between able-bodied and pauper, a point recognized by the RIE board. Just months after enactment of the 1845 Poor Law (Scotland) Act, the RIE board resolved to facilitate patients' access to poor relief where appropriate: "it would be a proper and crowning act of charity to poor persons who had been in the Infirmary, on its appearing that they were entitled to Parochial relief, if the Treasurer-Superintendent sent a memorandum along with them to the Inspector of the poor of the City stating that he is supposed to be poor and entitled to parochial relief and recommending his case be enquired into." (LHB1/1/14 1845, 282) The impetus behind this resolution may have been to limit patient admissions and length of stay, as a liminal patient could be something of a hot potato between the infirmary and parochial board: "John W[…] is not a pauper and as he states he will not become such, his circumstances apart from his wish forming a barrier against pauperism. I have therefore to request you will say on what ground John W[…] is refused admission to the Royal Infirmary," demanded the city Assistant Inspector of the Poor of the RIE board of management (LHB1/1/18 1855, 391).

Medical staff were known to stretch a point with respect to admission and treatment of the very poor, however. Philip G., an 1869 RIE chronic bronchitis patient, stated at intake that although he had already seen a doctor and improved under treatment, "he … applied to the Infirmary as he was not quite well and had no place to live." (LHB1/129/3/20, 50) The hospital accommodated him as an inpatient for fifteen days. Medical staff furthermore acknowledged to the board of management that they delayed discharging recovered patients due to poverty:

> Dr Sanders remarked … If patients are sent out too soon, that is just recovered from an acute attack and before they have got strength to work, … they either become victims of some serious chronic disease and soon die or become invalids and hence a burden to the community. On the whole it seems at once bad policy and something cruel and harsh to send out patients of this description before they are perfectly

> strong and completely recovered. Dr Bennett stated that in several cases he found that patients completely recovered could not be sent out for want of clothing or similar necessaries. (LHB1/1/22 1865, 563–64)

The board, concerned with the increase in per-patient cost from extended stays, remained unmoved, commenting, "This it is to be observed is not a valid reason for retaining patients in the Infirmary; as there are always means … of supplying these requisites; either from the Parochial Board if the patient is a pauper, or from some other source." (564) Medical staff, then, went so far as to assist patients beyond the stated limits of their institution, contrary to the insistence by others of the practitioners' class — the managerial board — that patients should be assisted only medically.

At the same time, however, clinicians demonstrated little faith that patients would relate their conditions accurately. As during the late-Georgian/early-Victorian period I addressed in chapter four, during the mid-Victorian period practitioners at both GRI and RIE continued to express reservations regarding patient accounts by explicitly distancing themselves from patients' statements. Such disavowals frequently centered on symptoms strongly associated with tubercular disorders. "She is said to have become emaciated, although she is still tolerably fat," noted the clerk of RIE phthisis and heart disease patient Jane B. (LHB1/129/5/20 1855, 60). Of blood expectorated by GRI phthisis patient Sarah McC., the clerk recorded, "she says it obstructed her breathing as if it come up her windpipe" (i.e., not from her esophagus; HH67/8/4 1875, 1).

RIE medical staff were ambivalent as to the diagnosis for another Robert K., recording "bronchitis?" in the ward journal index (LHB1/129/3/1, 1845) and "phthisis" in the admissions register (LHB1/126/30 July 1845, entry 2562). "*He describes himself* as being of a healthy constitution, and *says* his parents and most of his relatives were also … " wrote the clerk. "There is no fever at any period of the day, and little tendency to sweating. He has,

however, severe and long continued cough, with copious expectoration of white and frothy sputa …. *He says* that he never expectorated any blood." (LHB1/129/3/1, 97–98; emphasis added) The statements that the clerk attributed to Robert K. are those pertaining to symptoms closely associated with phthisis — hereditary constitutional weakness and hemoptysis. The unattributed symptoms are those not indicative of tubercular disorders, or not necessarily so: the former include the patient's white sputum and lack of fever and sweating, the latter his severe, long-continued cough.

Such cases indicate that practitioners were hesitant to underwrite symptoms that could tip the scales toward a diagnosis of phthisis; instead, they made it clear that they could not vouch for their patients' veracity on these points. By disclaiming and deemphasizing the symptoms classically associated with TB-related diseases, practitioners emphasized the reliability and importance to diagnosis of clinical signs detectable by themselves alone, reiterating the clinician's unique perspicuity from the turn of the nineteenth century. What the patient observed about herself was uncertain, but the doctor's understanding of him or her — what his trained gaze encompassed — was not in question. In this way, medical staff constructed a scientific detachment from the diagnostic process, taking legible pains to distinguish certainty from hearsay. Their greater distrust of diagnostically loaded symptoms such as those associated with consumption tends to reveal rather than deny bias, however. It is a performance not of detachment, but of reluctance to leave unverified the symptoms leading to a momentous diagnosis. Only the clinician could ascertain diagnoses of TB-related diseases.

Notably, however, medical staff did not disavow only diagnostically significant symptoms; similar doubt surrounded patients' self-reported temperance and morality. Of Andrew M. — an 1855 RIE phthisis patient who dated his illness to what was characterized

by the clerk as a drunken "debauch" and "spree" (LHB1/129/2/54, 167) — the clerk noted, "patient has not (he says) tampered with his constitution by the practice of venery." (169) "She does not acknowledge any excesses or irregularities in food or drink," commented the clerk of RIE bronchitis patient Ann H. (LHB1/129/5/34 1860, 24); upon feeling ill, RIE pleurisy patient John F. "took warm foot bath — also warm gruel," according to the clerk, "(but he says no whisky)." (LHB1/129/2/12 1855, 91) RIE phthisis patient Mary R. "state[d] that she has always been well-fed and well-kept, and little exposed [i.e., to the elements]," an innocuous report the clerk called into question due to her rumored character: "but she is reputed as a femme du pavé," or prostitute, he continued (LHB1/129/5/22 1855, 105).

Through rhetorical moves emphasizing that certain statements originated with the patient, practitioners demonstrated circumspection in diagnosing TB-related disorders, but also reluctance to credit working-class patients' reports of salubrious behavior. The former echoes the 1822 *Edinburgh Medical and Surgical Journal* stethoscope reviewer, who expressed concern that phthisis be diagnosed beyond a doubt in private patients owing to its incurability (457). In chapter four, I noted that the "mature deliberation" the reviewer advocated before applying the stethoscope to a private patient contrasted with his assurance that "no bad consequence can follow" from the instrument's use in hospital practice, distinguishing the consideration practitioners afforded private and charity patients. GRI and RIE practitioners' hesitation to credit TB-related symptoms suggests medical staff made less distinction in practice. In contrast, however, medical staff's doubt of patients' temperance and healthy living situations reveals a fixed belief in their working-class patients' departure from middle class norms of behavior. In exhibiting empathy for their working-class patients while simultaneously casting doubt upon their accounts of their symptoms and behavior,

practitioners at GRI and RIE echo Bennett's ability to form lasting relationships with working-class patients while revealing their identities and bodies.

MEDICAL AUTHORITY

Despite evidence of empathy, there is no doubt that the relationship between practitioner and patient was unequal: patients were expected to submit to medical authority, and the goals of practitioners were primary. Medical staff demonstrated a belief in the importance of orthodox treatment in the maintenance of health. In one example of this, the GRI clerk recorded Bridget K., an 1860 phthisis patient, as having "Caught a cold which she neglected to have relieved or cured and which consequently became rapidly worse." (HB14/15/16, 229) The clerk directly and causatively linked her neglect — not of self-care, but of medical care, having not had her disorder relieved or cured for her — to her present, grave disease.

In treatment, too, increasing medical authority was apparent. Whereas during the period 1821 through 1840 practitioners still acquiesced with good grace to patient's wishes respecting treatment, this was no longer the case by the mid-Victorian period. "The gallic acid has been suspended from patient imagining that it increased the abdominal pain and torminae," sniffed the clerk of hysteralgia patient Susan C. (LHB1/129/5/4 1850, 94). It was more usual than previously for medical staff to respond harshly to patients' refusal of treatment. Mary C., who sought treatment at Edinburgh for pain in her head, was summarily dismissed for refusing her prescribed blister: "This treatment … she refused to undergo. She was accordingly dismissed from the infirmary," noted the clerk (LHB1/129/5/52 1869, 122) Alexander D., an 1865 RIE phthisis patient, left the infirmary after a four-day stay during which medical staff never fully examined him. The clerk reported that Alexander D. "was

recommended … as a fit person for the enjoyment of the charity of the hospital and was admitted because so recommended …. The patient, however, absconded before his case was properly taken." (LHB1/129/2/57, 206)

Practitioners at RIE engaged in deception of patients while expecting full disclosure from them. In one instance of the latter, the clerk recorded syphilis patient Catherine G.'s reluctance to reveal early symptoms of the disease, noting, "Patient states that five years ago she had a sore throat and great difficulty of swallowing, but is not very explicit on the subject." With respect to the former, Bennett (1858), after exhorting students to use "language proportioned to the intelligence of the individual" (20) to gain information from patients, advises the opposite when practitioners exchange information about the patient within the patient's hearing: "In conversing on, or discussing, the circumstances of the case at the bed-side, we should always use technical language … instead of saying, a man has a cavern at the top of the lung, we should speak of a vomica under the clavicle." (21)

Medical staff further used subterfuge to test patients' veracity. "[S]he complains of pain across the whole of the abdomen …. On pressure on the abdomen if she was on her guard she invariably screamed out with pain but if pressure were made when she was not looking she took no notice of it if it were not very forcible," reported the clerk of 1855 RIE phthisis patient Georgina McD. (LHB1/129/5/21, 146). Of hemoptysis patient Jane McG., the clerk wrote, "The fits have been fewer within the last twenty-four hours than previously," apparently tracing this reduction to an incident he related immediately afterward: "D'r [*sic*] Bennett announced in the patient's hearing that a red hot iron is to applied to the back when fits come on." (LHB1/129/5/23 1855, 22) As this treatment is neither recorded for any other patient nor noted to have ever been administered in this case, it is likely that Bennett never actually intended the iron to be applied, but rather sought to

ascertain whether Jane McG.'s "fits" were under her control by threatening a painful response should they continue.

It is furthermore evident that medical staff thought themselves entitled to dissect patients of interest. Wilhelmina F., suffering from gastric ulcers, miscarried a first-trimester fetus in the hours before her death, surprising the practitioner who had identified only an abdominal tumor (LHB1/129/5/23 1855, 18). A gynecological examination subsequent to delivery revealed the presence of fluid in the abdominal cavity, which together with the previously-identified tumor seems to have generated an intense desire to autopsy the patient: her record ends with the large notation, "Postmortem refused!!" (25; emphasis retained), expressive of frustration. Indeed, in 1875 RIE medical staff sought the right to dissect any patient without permission: "The medical and surgical officers with the view of increasing the number of post-mortem examinations suggest that there should be withdrawn from the regulations the necessity of a written permission from the friends of deceased patients," recorded the minutes of the managers' meeting on October 11. The committee to review the rules regulating the pathologist refused, stating that this would "infallibly and not unjustly lead to remonstrances and dissatisfaction from the public" (LHB1/1/27, 148).

In all, the picture emerges of a practice increasingly governed by medical authority. The relationship between practitioner and patient was not entirely contentious; rather, patients increasingly reported seeking medical treatment while practitioners in some ways treated patients respectfully and sought to assist them beyond medical treatment. Nonetheless, during the period 1841 through 1880 practitioners more commonly and more explicitly regarded the patient as an unreliable source of information with respect to his or her own health and history, with practitioners treating themselves and each other as more trusted narrators of patients' bodily status. Medical staff furthermore performed a more

authoritative role, expecting patients to conform to hospital treatments and procedures. This expectation extended even to patients' deaths, after which practitioners could be displeased to be refused permission for autopsy — the traditional requirement for which they actively sought to end in 1875. As I will show in the following sections, it is the conjunction during this period of medical authority, the importance of medical care, and practitioners' special knowledge of their patients that granted medical practitioners significant voice in arbitrating behavioral and social health as well as bodily health and disease.

AUTHORITY, TECHNOLOGY, AND DIAGNOSIS

The increasing detachment with which practitioners viewed their patients during the mid-Victorian period — in many cases despite their evident investment in patients' recovery and health — is in keeping with a more formalized practitioner-patient relationship favored by a more technological and scientific approach to patient examination. Case notes during this period, particularly at RIE, reflect a ritualistic (cf. Kennedy 2010, 10) patient examination in which standard observations were made regardless of patient complaint. At both institutions, patient records opened with the patient's account of his or her illness, followed by his or her family history and physical examinations of the respiratory, circulatory, integumentary (skin), nervous, genitourinary, and digestive systems.

Beyond this, however, the systems of examination varied between these institutions. At RIE, patient examination notes accorded closely with the procedures recommended by Bennett (1858, 20–22). Highly structured, these records contained subheadings indicating patient history and each bodily system. Clerks recorded both normal and abnormal findings. At GRI, intake notes were less constrained. Often lacking subheadings demarcating the various bodily systems examined and indeed often lacking notes for some systems

altogether, GRI case notes indicate a more variable and less thorough approach to such records in the institution overall. Nonetheless, GRI case notes from 1841 – 1880 — particularly the 1870s — do show more internal organization than those from earlier periods, demonstrating not only a difference in procedure between RIE and GRI, but a larger temporal change in GRI practitioners' approaches to the creation of these records.

PATIENT NARRATIVES

Perhaps the most notable change in patient case notes since the early nineteenth century was the decreased emphasis on patient history. As I have discussed, over the period from 1794 through 1840, the diagnostic process transitioned from the practitioners' astute questioning and interpretation of the patient's account to incorporate techniques of physical examination to identify hidden pathology. The continuation of this trend during the period 1841 through 1880 saw multiple strategies through which practitioners sought to bring patients' self-reported histories into alignment with the increasingly standardized form and self-consciously scientific stance of patient examination. I have already discussed practitioners' strategies to distance themselves from patient statements they found dubious; medical staff also rhetorically underscored those statements they found justifiable.

Clerks might restate pertinent points from the patient's statement verbatim under the examination notes. RIE phthisis patient Ann S. described "a pain which shoots through from the base of the left chest anteriorly to the shoulder of the same side … and micturition … accompanied by pain in the lower part of the abdomen and the small of the back, and when seized with a fit of coughing [urine] is passed involuntarily." (LHB1/129/5/35 1860, 65) Of her physical examination, the clerk noted, "Nervous system …. pain shooting from the base of the left chest anteriorly to the shoulder of the same side …. Genito-urinary

system. Urine normal, but passed … with pain in the lower part of abdomen, and small of the back, and involuntarily when seized with a fit of coughing." (66–67; emphasis retained) Such statements are thus doubly co-opted by medical staff, first by translating the patient's narrative into medical terms, and then by interleaving elements of this narrative with signs observed by the practitioner.

In this way, practitioners reorganized patients' narratives to conform to medical procedures and thought processes, whereas in earlier periods the practitioner only guided such narratives. That is, in addition to eliciting detailed information from patients, medical staff selectively validated those aspects of patient experience deemed most pertinent. This could be explicit, as in the case of Thomas P., treated for chronic pleurisy at RIE in 1855: "By himself the malady is traced to the deleterious influence of confinement with other … workmen in a small hot workshop," the clerk recorded, adding approvingly, "reputed indeed to be unhealthy." (LHB1/129/2/54, 95) Such commentary and selective doubt or validation of patients' own accounts serve to underscore medical practitioners' increasingly authoritative knowledge — while the patient may relate many aspects of his history to his present condition, only the medical staff can know what is *really* important. Indeed, Bennett was renowned for pruning patient accounts of "irrelevant matter": "If there were no direct relation between an attack of measles at 8 years of age and an attack of pneumonia at 50, the reference to the former was at once repressed," commented the author of Bennett's obituary (1875, 475).

This emphasis on the practitioner as authoritative gatekeeper for patient-reported symptoms is in keeping with the increasing importance of the sign rather than the symptom in nineteenth-century Western medical diagnostic practice described by Foucault (1963, 159–70). The primacy in patient examinations of physical examination and those aspects of the

patient's account the practitioner could verify himself minimizes the extent to which practitioners needed to rely upon their patients — whom they increasingly treated as unreliable narrators of their own conditions — to arrive at an appropriate diagnosis. In distancing themselves from certain of their patients' statements, then, medical staff not only performed scientific detachment, but also in a sense removed the patient from his or her own case history (cf. O'Neal 1998, 481). As I will show, this removal was by no means complete in practice, however, and it is this retention of older symptomatic definitions of TB and its analogues alongside the rise of anatomical signs that allowed the conflation of individual and social disease that I discuss below.

SIGNS, SYMPTOMS, AND DIAGNOSIS

Quantitative analysis of the signs and symptoms forming diagnoses of TB-related and non-TB-related diseases for the period 1841 – 1880 supports the continued coexistence of older symptom-based constructs of these disorders alongside new anatomical definitions. TB and its analogues are sharply distinguished from both non-TB pulmonary and non-TB, non-pulmonary disorders at both GRI (table 6.2) and RIE (table 6.3), and more strongly so at GRI than in earlier periods (tables 3.3 and 4.3). At GRI, TB and its analogues differed significantly ($p < 0.05$) from those of non-TB, non-pulmonary disorders in the categories of appearance and comportment, circulatory, dermatological, digestive, genitourinary, medical history, pain, and miscellaneous clinical indicators (for a complete listing of clinical indicators and their frequencies, see appendix E). Differences approached significance ($p < 0.10$) for an additional category, general, which comprised systemic signs and symptoms such as impaired general health or sleep, temperature alterations, and thirst. Respiratory indicators again failed to distinguish these two disease categories, most likely owing to the continued low frequency

with which such signs and symptoms were recorded in cases of non-TB, non-pulmonary disorders. Compared to the period 1821 – 1840 (table 4.3), three categories gained significance or near-significance: circulatory, which had also been significant in the period 1794 – 1820, genitourinary, and general. Mouth and throat lost near-significance.

Table 6.2. P-values for Fisher's exact tests of differences in clinical indicators at GRI, 1841 – 1880

Signs and Symptoms, GRI	TB-Related vs. Non-TB Pulmonary	TB-Related vs. Other
1. Appearance and Comportment	0.0070**	0.0082**
2. Circulatory	0.0038**	0.0013**
3. Dermatological	0.0088**	6.14×10^{-5}***
4. Digestive	0.0035**	0.0141*
5. General	0.1905	0.0908†
6. Genitourinary	0.0046**	0.0022**
7. History, Medical	0.0004***	1.76×10^{-6}***
8. History, Personal	0.0005***	0.3173
9. Mouth and Throat	0.1304	0.2186
10. Pain	0.0252*	0.0040**
11. Neurological	1	0.1793
12. Respiratory, General	0.0006***	0.4092
13. Respiratory, Auscultation/Percussion	9.73×10^{-5}***	0.659
14. Respiratory, Chest Form/Sensation	2.65×10^{-6}***	0.5471
15. Miscellaneous	0.3761	0.0059**

Note: See appendix E for a complete list of signs and symptoms comprising each category.
† $p < 0.10$
* $p < 0.05$
** $p < 0.01$
*** $p < 0.001$

TB-related diagnoses were distinguished significantly from other pulmonary disorders in the categories appearance and comportment, circulatory, dermatological, digestive, genitourinary, medical history, personal history, pain, general respiratory, respiratory auscultation/percussion, and chest form/sensation (table 6.2). Compared to the

earlier cases discussed in chapter four (table 4.3), two categories — genitourinary and chest form/sensation — gained significance, while none lost significance.

Table 6.3. P-values for Fisher's exact tests of differences in clinical indicators at RIE, 1841 – 1880

Signs and Symptoms, RIE	TB-Related vs. Non-TB Pulmonary	TB-Related vs. Other
1. Appearance and Comportment	0.0015^{a}**	0.0005^{a}***
2. Circulatory	0.1509^{a}	0.0005^{a}***
3. Dermatological	0.0015^{a}**	0.0005^{a}***
4. Digestive	0.1743	0.0010^{a}**
5. General	0.3924	0.2457
6. Genitourinary	0.0020^{a}**	0.0025^{a}**
7. History, Medical	$0.0985^{a\dagger}$	0.0005^{a}***
8. History, Personal	0.0005^{a}***	0.0005^{a}***
9. Mouth and Throat	0.1054^{a}	0.0325^{a}*
10. Pain	0.1564	0.0020^{a}**
11. Neurological	0.3769	0.9218
12. Respiratory, General	0.0005^{a}***	0.0005^{a}***
13. Respiratory, Auscultation/Percussion	0.0005^{a}***	0.0005^{a}***
14. Respiratory, Chest Form/Sensation	2.75×10^{-5}***	1.93×10^{-5}***
15. Miscellaneous	0.0202*	0.0021**

Note: See appendix F for a complete list of signs and symptoms comprising each category.
[a] P-value obtained through Monte Carlo simulation using 2000 replicates.
† $p < 0.10$
* $p < 0.05$
** $p < 0.01$
*** $p < 0.001$

In comparison to GRI, at RIE TB-related disorders were differentiated from non-TB, non-pulmonary disorders in more categories of clinical indicators, but fewer categories distinguished the former from non-TB pulmonary disorders. At RIE, all categories except general and neurological significantly distinguished TB-related disorders from non-TB, non-pulmonary diagnoses (table 6.3; appendix F lists these clinical indicators in detail, along with their frequencies). Compared to the same period for GRI (table 6.2), personal history, mouth

and throat, and all three respiratory categories gained significance, while general signs and symptoms lost near-significance.

With respect to non-TB pulmonary diagnoses, TB-related disorders were differentiated significantly in eight categories: appearance and comportment, dermatological, genitourinary, personal history, all three respiratory categories, and miscellaneous. An additional category, medical history, approached significance (table 6.3). Compared to GRI (table 6.2), only miscellaneous signs and symptoms gained significance, but three lost significance: circulatory, digestive, and pain.

Across time at Glasgow, then, I have found a net gain of sign and symptom categories differentiating between TB-related disorders and both other pulmonary diseases and non-TB, non-pulmonary diagnoses. Some fluctuation in categories attaining significance may be attributable to sample size — the 1841 through 1880 GRI total sample comprises 109 patients, an increase of 24 percent and 114 percent over the periods 1821 – 1840 and 1794 – 1820, respectively. This is not likely to have been the only contributor, however: the total number of clinical indicators recorded for 1841 – 1880 was 1650, a six percent decrease from that of the period 1821 – 1840.

Of the changing signs and symptoms contributing to diagnosis, most notable was the first appearance of genitourinary indicators among the categories differentiating TB and its analogues from both of the other disease categories. This was in part driven by a new interest in detailed urinalysis (e.g., Beale 1864; Parkes 1860; Roberts 1862), supplanting the simple visual examination of urine performed at GRI prior to the mid-nineteenth century. Urinalysis results at GRI — with the exception of a single instance each of urine density, sugar, and albumen (the former two in cases of TB-related disorders) — were recorded exclusively in cases of non-TB, non-pulmonary disorders. As a result, urinalysis techniques

contributed little to the GRI clinical construct of TB-related diagnoses, except inasmuch as cases of the latter either did not require urinalysis for diagnosis or returned normal urinalysis results that were not recorded.

In contrast, during the period 1841 – 1880, the preponderance of female patients in my GRI sample resulted in two to three times the instances of amenorrhea recorded by medical staff, and like the period 1821 – 1840, they recorded amenorrhea most often among patients diagnosed with TB-related disorders (table 6.4). That recorded amenorrhea remained constant from 1794 through 1880 and across institutions for the period 1841 through 1880 indicates a continued concern with female reproductive anatomy and its relationship to disease. That practitioners recorded amenorrhea most often in the mid-Victorian period at both GRI and RIE in association with TB-related diagnoses suggests a growing conceptual link between gynecological and tubercular disorders. This is not to say that amenorrhea did not really occur more frequently in cases of TB and analogous disorders. Rather, their co-occurrence — like the significance of nearly all sign and symptom categories rather than respiratory categories only — underscores the conception of tubercular disease as a systemic disorder.

Unlike many other clinical indicators, of course, amenorrhea applies only to female patients. As no sign or symptom of comparable frequency is specifically male, female patients are uniquely pathological, part of a larger social phenomenon I will discuss in greater detail below. For the present discussion, the observation of feminine, but not masculine, pathology meant that only gynecological problems could be observed in conjunction with other disorders. This supported the widespread construction of feminine anatomy as uniquely sensitive to health insults, and feminine pathology as uniquely associated with systemic disease, including most often in this sample TB and analogous conditions.

Tubercular disorders thus come to reinforce the construction of women as essentially pathological.

Table 6.4. Amenorrhea, 1794 – 1880

	Diagnosis Groups			
Time Period	TB-Related	Non-TB Pulmonary	Other	TOTAL
1794 – 1820, GRI				
Female Patients	9	5	6	20
Amenorrhea	2	0	3	5
Proportion Amenorrhea	0.22	0.00	0.50	0.25
1821 – 1840, GRI				
Female Patients	12	8	9	29
Amenorrhea	4	1	2	7
Proportion Amenorrhea	0.33	0.13	0.22	0.24
1841 – 1880				
GRI				
Female Patients	46	21	25	92
Amenorrhea	15	0	4	19
Proportion Amenorrhea	0.33	0.00	0.16	0.21
RIE				
Female Patients	99	40	53	192
Amenorrhea	38	8	13	57
Proportion Amenorrhea	0.38	0.20	0.25	0.30

The differences I observed between RIE and GRI may result for the most part from the more detailed examination protocol I have discussed at RIE; however, those differences not attributable to this reveal important distinctions between concepts of disease at these institutions. The increased level of detail in RIE's case notes is marked. For this period, my RIE case notes sample included 3.8 times more patients than GRI (GRI n = 109, RIE n = 417), but 6.4 times more signs and symptoms (total signs and symptoms: GRI n = 1650 (appendix E), RIE n = 10,601 (appendix F)). The difference amounts to about ten additional

clinical indicators per patient at RIE, on average (table 6.5), largely due to the RIE standard of examining every bodily system for every patient. RIE practitioners also tended to take a more detailed record of the patient's account of his or her history and present state.

Table 6.5. Clinical indicator category popularity at GRI and RIE, 1841 – 1880

	Observations					
	Total		Per Patient [a]		(RIE – GRI)[b]	
Signs and Symptoms	GRI	RIE	GRI	RIE	Value	Rank
1. Appearance and Comportment	118	743	1.07	1.78	0.71	6
2. Circulatory	186	938	1.69	2.25	0.56	9
3. Dermatological	92	624	0.84	1.50	0.66	8
4. Digestive	145	734	1.32	1.76	0.44	11
5. General	37	372	0.34	0.89	0.56	9
6. Genitourinary	75	1020	0.68	2.45	1.76	1
7. History, Medical	211	860	1.92	2.06	0.14	13
8. History, Personal	163	915	1.48	2.19	0.71	6
9. Mouth and Throat	83	757	0.75	1.82	1.06	5
10. Pain	130	966	1.18	2.32	1.13	4
11. Neurological	12	194	0.11	0.47	0.36	12
12. Respiratory, General	229	1473	2.08	3.53	1.45	2
13. Respiratory, Ausc./Perc.	236	1492	2.15	3.58	1.43	3
14. Respiratory, Form/Sensation	30	174	0.27	0.42	0.14	13
15. Miscellaneous	66	254	0.60	0.61	0.01	15
TOTAL	1650	10601	15.00	25.42	10.42	

[a] Per-patient values are the total observations divided by the total number of patients for each institution (GRI $n = 109$, RIE $n = 417$).
[b] (RIE – GRI) values are the differences in per-patient values rather than total observations.

The RIE practice of complete examination reveals in its practitioners both a scientific stance and assurance in the patient's incomplete knowledge of him- or herself. As I discussed in chapter five, Bennett taught his students the importance of scientific medicine (1858, 1–2), exhorting them to complete dissection in order to thoroughly test their antemortem hypotheses regarding the patient's pathology (22). In examination, too, he noted

in his *Clinical Lectures*, "it may appear to you that such a minute examination is unnecessary; but … ignorance of a fact frequently leads to error" in diagnosis (20). His advocacy of complete examination also sought to avoid false confirmation of the patient's prior assumptions: "We must guard against the preconceived views of the patient." (21) In the complete and minutely detailed case notes I sampled, it is clear that the RIE medical staff agreed with Bennett, demonstrating an institutional construct of disease hidden to the patient but not the careful practitioner. This construct renders diagnosis practitioner-driven rather than patient-driven, assuming as it does that examination of the patient may reveal a medical problem other than that she or he sought admission to address, and that the practitioner's priority is to discover and treat this "real" disease rather than that the patient has preconceived.

This increased level of detail likely drives the newly significant differences at RIE but not GRI between TB-related and non-TB, non-pulmonary disorders. As respiratory examinations were made routinely of every patient at RIE, these categories of clinical indicators were more populated for non-TB, non-pulmonary disorders at RIE than GRI. RIE non-TB, non-pulmonary cases yielded 262 total observations across all three respiratory sign and symptom categories — or 2.34 per non-TB, non-pulmonary patient — in comparison with 20 total observations at GRI, or 0.67 per patient in this diagnosis category. The categories mouth and throat and personal history were markedly more populated at RIE as well. These rank fifth and tied for sixth, respectively, in the magnitude of difference between RIE and GRI in the number of observations per patient across diagnostic categories (table 6.5); those categories with greater differences in the instances observed per patient distinguished TB-related and non-TB, non-pulmonary diagnoses at both institutions. As a result, it appeared that the greater number of clinical indicators recorded at RIE allowed

discrimination between TB-related and non-TB, non-pulmonary disorders in a greater number of symptom categories, emphasizing the distinctive nature of the former diagnoses and their dependence upon symptoms beyond the pulmonary. At both institutions, TB and its analogues were perceived as systemic diseases, not just lung diseases.

Despite this similarity, however, quantitative analysis comparing TB-related to other pulmonary diagnoses revealed important differences in the constructs of TB-related disorders between the two institutions. Circulatory, digestive, and pain indicators significantly differed between TB-related and other pulmonary diseases at GRI but not RIE; miscellaneous indicators were significantly different at RIE but not GRI. This latter is likely an effect of sample size: miscellaneous indicators comprised a poorly populated category at both institutions, but the fourfold greater absolute number of observations at RIE would have allowed statistical differentiation (table 6.5).

With respect to circulatory signs and symptoms, pulse indicators — particularly descriptive characteristics — appear to drive the significant difference between TB-related and non-TB pulmonary disorders at GRI. At GRI, a feeble or weak pulse is by far the most common circulatory sign recorded by practitioners in TB-related cases, followed by a very rapid pulse (115 beats per minute or more); these signs are less common among non-TB pulmonary cases, ranking third and tenth, respectively (appendix E). At RIE, a feeble pulse is also the most common circulatory sign recorded in cases of TB and its analogues, but it is also the most common in cases of other pulmonary disorders. Pulse rates in patients with TB-related diagnoses at RIE are more evenly distributed than at GRI, although a larger proportion of very rapid pulse rates occurred among TB-related cases than other pulmonary cases (appendix F). It thus appears that practitioners at GRI emphasized feeble and very

rapid pulses in TB-related diagnoses, while these did not figure prominently in such diagnoses for RIE medical staff.

Among digestive indicators, constipation and diarrhea likely drive the disparate significance between GRI and RIE. At GRI, constipation was less common among cases of TB-related disorders compared to other pulmonary diseases (recorded in 25 percent and 54 percent of cases, respectively), whereas the opposite is true for diarrhea (22 percent and zero percent of cases, respectively). At RIE, constipation was nearly equally frequent in both diagnosis categories (recorded in 28 percent and 27 percent of cases, respectively) but medical staff more commonly recorded diarrhea with TB-related diagnoses than other pulmonary diagnoses (17 percent and 7 percent of cases, respectively). This suggests that for GRI practitioners, diarrhea indicated and constipation contraindicated a TB-related diagnosis, whereas for RIE practitioners, only the former was true — and that incompletely so.

Pain symptoms, too, significantly differed between TB-related and other pulmonary disorders at GRI but not RIE. At GRI, the difference appears to have been driven by acute pain and pain in the chest and thorax, both of which are underrepresented among cases of TB-related diseases (appendix E). At RIE, these differences are present, but less exaggerated (appendix F), suggesting that for RIE practitioners, the presence of acute pain or pain in the chest or thorax did not as strongly indicate a non-TB pulmonary disease.

Altogether, these differences indicate adherence in some respects to older constructs of TB and its analogues at GRI that had been lost at RIE. Medical staff's emphasis on pulse characteristics in diagnosing these disorders at GRI hearkens to the outmoded external examination and Buchan's (1790) description of the disease I discussed in chapter three, in which abnormalities of pulse figured prominently (e.g., 176). RIE practitioners de-

emphasized these pulse characteristics, in keeping with the newer focus on pathological anatomy and physical examination in diagnosis. With respect to digestive indicators, RIE practitioners may have regarded constipation in patients manifesting TB-related disorders to be a negative symptom (cf. Bennett 1858) — "Symptoms which are usually present in the disease, but are absent in the particular case" (21) — a deviation from the overall pattern of clinical indicators that the practitioner should note, but not one salient enough to warrant a non-TB-related diagnosis.

In any case, at intake both bowel indicators and pain were symptoms rather than signs, their presence or absence the word of the patient alone. That RIE medical staff relied less on these clinical indicators in diagnosing TB and its analogues indicates that RIE practitioners more generally adopted the scientific, sign-oriented diagnostic procedures advocated by Bennett, while GRI medical staff continued to hew somewhat more closely to older, less physical and anatomical modes of examination. This is not to say that GRI practitioners did not avail themselves of new technology and new techniques: as I discussed in chapter four, their adoption of the stethoscope and percussion indicates the opposite. Nor is it true that RIE medical staff entirely abandoned older modes of examination: classic signs and symptoms of TB and its analogues, as well as careful questioning of the patient, continued to figure prominently in diagnosis at Edinburgh. However, GRI practitioners do not seem to have transitioned to newer modes of diagnosis as rapidly nor as thoroughly as RIE medical staff.

This distinction may be attributable in part to GRI's decline in stature beginning in 1874. With the university's relocation to Glasgow's West End, the Western Infirmary replaced GRI as the training hospital affiliated with the University of Glasgow. GRI continued to offer medical training — first as the GRI School of Medicine beginning in

1876 and as St. Mungo's College of Medicine from 1888 — but as this constituted study independent of a university degree, the institution lost prestige and students to the new Western Infirmary. The change may have reduced GRI practitioners' impetus to implement new technologies and new approaches in the latter part of the period I address here.

TECHNOLOGY AND MEDICAL PERCEPTION

Qualitative differences, too, distinguish diagnosis of TB-related disorders at RIE from that at GRI. Practitioners at GRI tended to characterize their auscultatory findings by diagnosis, such that they described patients as having a bronchitic, pneumonic, or tubercular crepitus. As I discussed in chapter four, this institution-specific shorthand departs from Laënnec's work by distilling the unstated suite of sounds observed into a diagnosis. This practice elides the diagnostic process: by noting the set of sounds observed in a particular patient as tubercular, rather than describing the sounds that together indicated tuberculosis, GRI medical staff reported the examination's result but not its content. In so doing, GRI practitioners reveal an emphasis on diagnostic endpoint rather than diagnostic procedure, and thus perhaps on treatment rather than research and training.

In contrast, auscultation by RIE medical staff was recorded in a manner unequivocally emphasizing the processes of examination and diagnosis. RIE clerks recorded stethoscopic findings in minute detail. For example, the clerk recorded the auscultatory examination of Elizabeth K., RIE bronchitis and phthisis patient, as follows:

> Under right clavicle, inspiration is harsh, attended with a small sized bubbling and also with sonorous ronchus [*sic*] — expiration is prolonged and attended by sonorous ronchus. Below the level of the second rib inspiration is harsh and expiration is almost inaudible — after a cough, however, its place is taken by a loud sibilant and sonorous ronchus. On left side under clavicle both expiration and inspiration sounds are at first attended with loud sibilant and sonorous ronchi, but with no moist ronchus — after a fit of coughing they become dry and normal. In the lower part of

> the left side in front dry sibilant and sonorous ronchi are heard, but no moist ronchus. … Right supra-scapular region presents on auscultation nearly the same phenomena as the right infra-clavicular, but in the scapular and infra-scapular regions very fine sub-crepitant rhonchus may be heard, in the former place mingled with sibilant and sonorous rhonchus. On the left side, posteriorly, with the exception of some sibilant and sonorous ronchi, there is nothing abnormal in the respiratory sounds. Vocal resonance slightly marked on both sides, appearing greater on left than on right. (LHB1/129/5/22 1855, 77–78)

Compare to this the GRI clerk's notes of the auscultation performed on Elizabeth A., phthisis patient: "The stethoscope communicates to the ear — a roughened respiratory mur [i.e., murmur] found under both clavicles — doubtful tubercular crepitation under the right. Elsewhere over the whole chest the breathing is for the most part tubular and as a general rule absence of expiratory murmur." (HB14/5/16 1860, 231)

Concern with medical training and replicability of results contributed to the lengthy descriptions of auscultation at RIE, as was illustrated by instances in which Bennett made corrections to the clerks' auscultation notes. In the case of John R., an 1855 phthisis patient, a "peculiar metallic crepitation" and "metallic crepitation" on the left side of the chest noted by the clerk at John R.'s initial examination were struck out and replaced with "friction sound." (LHB1/129/2/48: 104) The clerk evidently made this correction after Bennett's own examination the following day: "Visited today by Prof Bennett who heard distinct friction under left … clavicle." (105) In the case of Jessie D., double pneumonia patient, auscultatory findings noted by the clerk on December 6, 1855 were contradicted in a different hand on December 8. At the bottom of the page is the addendum, in the same hand as the latter and signed JHB (i.e., John Hughes Bennett), "NB Clerk's reports of the respiratory system here defective." (LHB1/129/5/22: 96)

Indeed, the style of auscultatory examination at RIE bears Bennett's stamp even in cases he did not examine personally. Bennett expressly warned students against considering

auscultatory signs pathognomonic for particular diseases: "we should regard a crepitating rattle, not as distinctive of this or that so-called disease, but simply of fluid in the smaller air-passages, and an increased resonance of the voice, as indicating hollow spaces with vibrating walls, or increased induration of the pulmonary textures, and not as diagnostic of phthisis, pneumonia, and so on." (Bennett 1858, 56) Accordingly, RIE medical staff recorded the signs observed rather than their interpretation, in stark contrast to GRI. It is important to note that Bennett's opinion was not always the last word, however: "Cardiac sounds normal (such is D'r [*sic*] Bennett's opinion, but others who have listened to them declare that there is a reduplication of the second sound.)" noted the clerk in the 1860 phthisis case of Bridget W. (LHB1/129/5/34, 115) While GRI practitioners had betrayed some hesitancy in their auscultation during the period 1821 – 1840, by the mid-Victorian period RIE practitioners clearly were confident enough in their own auscultatory ability to challenge the expert Bennett.

The stethoscope was just one of a suite of technologies in use during this period at both RIE and GRI, including urinalysis and to a lesser extent clinical applications of the thermometer and microscope. Urinalysis beyond unaugmented visual inspection of the urine — chemical analysis for pH or components such as albumen, or microscopic and chemical analysis of precipitates — gained popularity in the mid-nineteenth century, and a number of texts from this period instruct practitioners in its protocols and applications (e.g., Beale 1864; Parkes 1860). Beyond uses such as identifying protein or sugar in the urine, however, the diagnostic utility of urinalysis lagged behind the degree of detail with which urine could be described: Parkes (1860) described for three pages the characteristics of urine in cases of phthisis before summarizing, "Altogether, the condition of the urine in phthisis seems to deviate little from the normal" (318).

Nonetheless, practitioners at RIE and to a lesser extent GRI did utilize these analyses across disorders. At GRI, in keeping with that institution's more pragmatic approach to patient examination, practitioners for the most part limited urinalysis reports to density — one earlier instance of which had also been reported in my early-Victorian sample — pH, and the diagnostically salient presence of albumen or sugar. Clerks recorded fifteen instances of urinalysis results for the 109-patient sample as a whole, twelve of which occurred in non-TB, non-pulmonary cases (table 6.6), indicating that medical staff limited the use of these analyses — or at least recording their results — to assist diagnosis in cases for which a particular test was indicated, such as suspected diabetes.

Table 6.6. Urinalysis beyond visual inspection

	Diagnosis Groups			
Time Period	TB-Related	Non-TB Pulmonary	Other	TOTAL
GRI				
Patients	55	24	30	109
Urinalysis Signs	2	1	12	15
Proportion Urinalysis	0.04	0.04	0.39	0.14
RIE				
Patients	210	95	112	417
Urinalysis Signs	312	170	199	681
Proportion Urinalysis	1.49	1.79	1.78	1.63

At RIE, practitioners performed detailed urinalysis as a matter of course. On average, medical staff recorded per patient more than one sign derived from such analyses, with similar frequencies of such clinical indicators across diagnostic categories (table 6.6). Urinalysis notes were also more extensive, including multiple urine components beyond albumen and sugar, indicating that RIE practitioners performed a standardized urinalysis —

and recorded the results — for nearly every patient. Indeed, such detailed analyses were performed despite the relative non-utility of the results, particularly in cases of TB-related disorders, to diagnosis and treatment — likely from either the institutional bent for doing things thoroughly, or out of investigative interest seeking to understand the variation in urine components not yet associated with disease states. Thus, for urinalysis as well as auscultation, GRI's practitioners exhibited an emphasis on those elements of examination most pertinent for diagnosis on a case-by-case basis, while RIE medical staff performed standard, more extensive examinations of every patient.

Two additional technologies, the thermometer and microscope, warrant brief discussion. These clinically newly-acquired tools were not used extensively at either RIE or GRI, but their use is nonetheless informative. Thermometer readings were recorded by medical staff in only eighteen cases overall — two at GRI (1.8 percent of cases) and sixteen at RIE (3.8 percent of cases). Notably, in all cases across both institutions, recorded thermometer readings ranged from the high end of the normal range upwards, with values between 99°F and 107°F. While it may be that practitioners measured temperature regularly but only recorded those found to be elevated, the propensity of RIE to record negative and normal observations makes this unlikely for RIE, at least. Indeed, at neither institution did medical staff record a temperature for every patient reporting fever or heat. Rather, this suggests that medical staff used the thermometer only to determine the degree of elevation — and that only in certain cases — after they ascertained an elevated body temperature by other means.

Notations of microscope use were only present at RIE, where its use was perhaps to be expected owing to Bennett's early-career experience as an independent instructor of microscopy and histology. "Sputa has pink masses under microscope shows many pus cells,"

noted the clerk without fanfare in one example, the case of phthisis patient Peter G. (1/129/3/16 1865, 28). Conversely, the clerk in William G.'s scrofula case recorded, "the matter vomited … has not yet been examined microscopically, owing to there not having been any microscope in the ward til yesterday." (LHB1/129/2/47 1855, 38) Medical staff did not record the use of microscopes frequently — and indeed this rarity, along with GRI practitioners' terser case notes, may explain its absence at Glasgow. The tenor of the rare mentions of microscopy at RIE indicate that practitioners turned to the instrument as a matter of course, although the results of such examination did not figure prominently in case notes.

Overall, a greatly increased level of detail characterized RIE in case notes compared to GRI. Thus, auscultation notes were brief and summarizing at GRI but extensive and detailed at RIE; similarly, the results of examinations such as urinalysis were recorded in greater detail and more frequently at the latter institution. This may relate in part to GRI's loss of stature as a training institution, discussed above. However, as brief auscultation notes like those I quoted above in the case of Elizabeth A. (1860) predated the 1874 establishment of the Western Infirmary and as only 30 percent of sampled cases for this period were admitted after 1874, this does not wholly explain these differences. Rather, RIE clinical instruction was dominated by Bennett, who, as I have discussed, forcefully advocated detailed case notes and the use of technologies like the stethoscope and microscope. Bennett's scheme of clinical examination and note-taking was described in his obituary as "Irksome at first … [but] always appreciated by the better class of students," going on to describe Bennett as deeply annoyed by imprecision ("Obituary: John Hughes Bennett" 1875, 475), representing him as a demanding instructor who required such complete case records. This likely drove the differences I have discussed here.

The mid-Victorian emphasis at both GRI and RIE on technology, physical examination, and signs originating with the practitioners' observations rather than patients' symptoms reveals a diagnostic practice increasingly reliant upon figuratively seeing into the patient. This pathological bent follows from the increased knowledge of and access to patient anatomy I discussed in chapters four and five. This was at times explicit: in 1863, the *Lancet* reported that surgeon Christopher Heath exhorted Westminster Hospital students, "the creature he would have to treat in disease would be a man whom he would not be able to dissect literally to find out the malady; but would have, as it were, to pierce the tissues with his mental eye, and discover the secret mischief by his cultivated senses." (Heath 1863, 401) "Seeing" pathology represented medical practitioners' continued reliance upon their unique perspicuity. By the mid-Victorian period, however, this perspicuity rested not only in practitioners' trained but unaugmented senses and mental acuity, but also in their mastery of instruments that augmented perception. Together, these allowed practitioners to see within the patient's body as well as uncovering the hidden diagnostic meaning in his or her statement and outward appearance (cf. Foucault 1963). In practice at RIE and GRI as well as in theory, then, medical perception became during the nineteenth century both more authoritative — taking precedence over that of the patient — and more penetrating.

It is notable, however, that the growing importance of technology and pathology in medical practice I have described was far from universal during this period. As I discussed in chapter four, in 1863 the surgeon Sydney Jones's remarks to students of St. Thomas' Hospital included encouragement to master the use of technologies like the stethoscope, as he was convinced of their utility despite having heard them "not unfrequently" dismissed as "lumber." (400) Heath followed his remarks on mental dissection above with a reminder not to neglect earlier forms of examination: "External form and general physiognomy both in

health and disease were also urged upon the student's attention," wrote the *Lancet* of his address (401). Indeed, as I will show in the following section, practitioners' new diagnostic emphasis on technology and signs rather than symptoms continued, as in the period 1821 – 1840, to complement rather than replace older essentialist constructs of TB and its analogues.

THE PERSISTENCE OF ESSENTIALISM

I have discussed the ways in which diagnostic procedures at GRI and RIE during the mid-Victorian period increasingly utilized technology to reveal pathology invisible to the unaugmented eye, whether through the mental dissection of physical examination or the scrutiny of urinalysis and microscopy. Examination and diagnosis were furthermore ritualized and practitioner-driven, with medical staff confident that thorough knowledge of their patients could reveal hidden pathology — perhaps even that unknown to the patient. While practitioners like Bennett argued that the identification of pathology was central to understanding and diagnosing disease, implying an anatomically-based construct of TB and its analogues, the continued significance to these disorders' diagnosis of clinical indicator categories located throughout the body speak to other practices. Indeed, during the period 1841 – 1880 practitioners at GRI and RIE demonstrated their continued reliance on systemic, even essentialist, etiologies for TB-related diseases.

NUTRITIONAL ETIOLOGY IN PRACTICE

In chapter five, I described Bennett's (1853) explanation of TB and related disorders as fundamentally diseases of nutrition, originating with the body's inability to assimilate fats. This view was not unique to Bennett: numerous practitioners during the mid-Victorian

period presented a nutritional origin of these diseases as given. Horace Dobell of London's Royal Hospital for Diseases of the Chest opened his explanation of the logical basis for his new pancreatic emulsion treatment with, "assuming a defect to exist in the natural power of digesting and assimilating fats[.]" (1865, 561–62) A variation on Bennett's nutritional etiology was presented in the *Lancet* by the Brompton Hospital for Consumption ("Brompton Hospital: Physical Signs of Phthisis" 1855), anonymous representatives of which described their "conviction that tubercle is a blood disease associated with … alterations of nutrition under depressed nervous influences; [and] mal-assimilation in its secondary processes of organic matters in the food."[4] (388)

Nutritional explanations for TB and its analogues were appealing in part because they explained the emaciation that had long been a hallmark of these disorders: mal-assimilation of fats required the consumptive's body to draw on its own fat stores. This perhaps drove the continued prominence of emaciation and weight loss in diagnoses of TB-related diseases at both RIE and GRI during the mid-Victorian period, recorded by practitioners in 52 percent and 62 percent of TB-related cases, respectively. Medical staff furthermore drew a parallel between this deficiency of fat digestion and a deficiency of fat intake. Both the latter and the former were implicated in the origin of these diseases: particularly at RIE, practitioners recorded a history of poor diet — insufficient or improperly balanced — in association with TB-related diagnoses. Clinicians did record such history across disorders, suggesting a belief in the contribution of poor nutrition to other diseases as well, albeit most prominently to cases of TB-related disorders.

[4] Malnutrition is indeed still today considered a predisposing factor for TB (e.g., Lönnroth et al. 2010); recent research has indicated a role for dietary fats in limiting TB infection, although it was omega-six fatty acids found in plant foods rather than the omega-three found in cod liver oil that were suggested to be beneficial (McFarland et al. 2008).

In keeping with the larger environment of behavioral explanations for TB and related disorders, which I discuss below, practitioners also read patient agency into deficient fat intake. Edinburgh-trained Fothergill lamented in his lay health guide (1874), "It is painful to see children at table permitted to reject every particle of fat …. Still more painful is it to know that the absurd caprice, if persisted in, will in all probability lead to such a condition as may result in tubercle. For there is no doubt … that this abstinence from fatty food too commonly paves the way for consumption." (41) In practice, RIE medical staff recorded patients' preference against fatty foods as well as their inability to eat fat — the latter presumably an indication of difficulty digesting the substance — only in cases of TB and related disorders, albeit infrequently. Nonetheless, practitioners accepted the contribution of dietary fat deficiency to the development of TB-related diseases broadly enough that RIE practitioners asked some phthisis patients directly about their taste for fatty foods: "she had no disinclination for fats," (LHB1/129/5/54, 1869: 139) noted the clerk in Margaret C.'s case history, a statement unlikely to have arisen unprompted from the patient.

The acceptance of nutritional etiologies for TB and related disorders is further evident in the frequency with which medical staff prescribed cod liver oil to treat these diseases at both GRI and RIE, despite these institutions' otherwise disparate prescribing practices. At GRI, treatments in the categories of antispasmodic, stimulant and unclassified drugs — which included cod liver oil — significantly ($p < 0.05$) differentiated between TB-related and non-TB, non-pulmonary disorders, while diuretics and drugs of indeterminate action approached significance ($p < 0.10$: see table 6.7 for a summary of quantitative analysis of treatments at GRI and appendix J for a complete listing of GRI treatments and their frequencies). Significantly differentiating TB-related diagnoses from other pulmonary diseases were diaphoretics, stimulants, and unclassified drugs. As no categories of treatment

for the period 1794 – 1820 differentiated among diagnoses (table 3.4) and I found only two significant differences in treatments for the period 1821 – 1840 (table 4.5), these results suggest a shift to more specific prescribing over time at GRI.

Table 6.7. P-values for Fisher's exact tests of differences in treatments at GRI, 1841 – 1880

Treatments, GRI	TB-Related vs. Non-TB Pulmonary	TB-Related vs. Other
1. Anodyne	—	—
2. Antacid	0.3333	1
3. Antispasmodic	0.1215	0.0373*
4. Astringent	0.2333	1
5. Bleeding	—	—
6. Cathartic	0.4803	0.5695
7. Corrosive	0.2657	0.1818
8. Demulcent	0.25	1
9. Diaphoretic	0.0138*	0.693
10. Diuretic	1	0.0909†
11. Emetic	0.2364	0.2222
12. Emollient	—	—
13. Expectorant	1	0.4
14. Mercury	1	0.4
15. Opium	1	1
16. Sedative	0.2143	0.5921
17. Stimulant	0.0093**	0.0020**
18. Tonic	0.7725	0.6074
19. Indeterminate Function	0.1181	0.0600†
20. Unclassified Drugs	0.0141*	0.0009***

Note: See appendix J for a complete list of the treatments comprising each category.
† $p < 0.10$
* $p < 0.05$
** $p < 0.01$
*** $p < 0.001$

Table 6.8. P-values for Fisher's exact tests of differences in treatments at RIE, 1841 – 1880

Treatments, RIE	TB-Related vs. Non-TB Pulmonary	TB-Related vs. Other
1. Anodyne	—	—
2. Antacid	0.8674	0.9395
3. Antispasmodic	0.1188	0.639
4. Astringent	0.8383	0.0987†
5. Attenuant	1	1
6. Bleeding	0.4974	0.7044
7. Cathartic	0.1026	0.2057
8. Corrosive	0.5219	0.0019**
9. Demulcent	0.502	0.2565
10. Diaphoretic	0.0034**	0.399
11. Diuretic	1	0.9042
12. Emetic	0.0050**	0.1236
13. Emollient	—	—
14. Expectorant	0.1246	0.1697
15. Mercury	0.48	0.5731
16. Opium	0.1447	0.3174
17. Sedative	0.3906	0.0336*
18. Stimulant	0.0166*	0.0845†
19. Tonic	0.0831†	0.2844
20. Indeterminate Function	0.4385	0.2624
21. Unclassified Drugs	0.0005[a]***	0.0005[a]***

Note: See appendix K for a complete list of treatments comprising each category.
[a] P-value obtained through Monte Carlo simulation using 2000 replicates.
† $p < 0.10$
* $p < 0.05$
** $p < 0.01$
*** $p < 0.001$

At RIE, treatments for TB and its analogues were similarly differentiated from those for other categories of disease (table 6.8; see appendix K for the full listing of treatments). Treatments in cases of TB-related disorders were significantly different from non-TB, non-pulmonary diseases for corrosive, sedative, and unclassified drugs; astringents and stimulants approached significance. TB-related cases differed significantly from non-TB pulmonary disorders for diaphoretic, emetic, stimulant and unclassified drugs, while tonics approached

significance. Thus, RIE practitioners added emetics — an unpopular category of drug at GRI — and to a lesser degree tonics to the categories of drugs differentiating TB-related from other pulmonary diseases at GRI. In contrast, RIE practitioners showed little overlap with their GRI colleagues in the categories of drug distinguishing treatment of TB-related disorders from non-TB, non-pulmonary diseases. Only stimulant and unclassified drugs share significance or near-significance, underscoring the differences between these institutions in approaches to treatment.

Most notable among the treatments recorded in cases of TB and its analogues is the distinction between this and other disease categories across institutions in the prescription of unclassified drugs, driven by cod liver oil. Having been introduced to orthodox UK practitioners by Bennett in 1841, medical staff did not record cod liver oil in my sample prior to the period 1841 – 1880. During the mid-Victorian period, however, medical staff at both GRI and RIE prescribed it commonly, and almost exclusively in cases of TB-related disorders (table 6.9).

Table 6.9. Cod liver oil prescriptions, 1841 – 1880

	Diagnosis Groups			
Time Period	TB-Related	Non-TB Pulmonary	Other	TOTAL
GRI				
Patients	55	24	30	109
Cod Liver Oil	16	1	1	18
Proportion CLO	0.29	0.04	0.03	0.16
RIE				
Patients	210	95	112	417
Cod Liver Oil	103	5	2	110
Proportion CLO	0.49	0.05	0.02	0.26

The drug seems to have been prescribed in Bennett's ward at RIE as a matter of course: "no medicine yet ordered but cod liver oil," noted the clerk two days after Norman McK.'s admission for phthisis (LHB1/129/2/12 1855, 41). Medical staff diagnosed Peter H. with pleurisy upon his admission May 25, 1855 (LHB1/126/34, entry 2499); a month later, on June 23, the clerk recorded the results of Peter H.'s examination by Bennett, ending with the remark, "Phthisis?" (LHB1/129/2/48, 109, 116) Four days later, on June 27, practitioners prescribed the patient three teaspoons of cod liver oil daily (116). The drug's popularity reached beyond Scotland: Norman McK. reported having been treated with cod liver oil at Newcastle Infirmary (LHB1/129/2/12, 39). Despite its widespread use, however, cod liver oil does not seem to have improved outcomes for TB-related disorders (table 6.10). Relief and death continued as the most common outcomes for these diseases — which included not only pulmonary TB and phthisis, but also skin, gland, and bone and joint TB — at both GRI and RIE, in significant contrast to other pulmonary disorders and non-TB, non-pulmonary disorders, both of which were most often cured or relieved.

Nutritional etiologies and treatments were more frequently recorded by practitioners at RIE than GRI. This, like the increased level of detail in RIE case notes, likely follows from Bennett's commitment to a nutritional construct of TB and related disorders. As Bennett espoused such an etiology prior to his employment at RIE, it is unlikely that an existing institutional bent toward such explanations influenced Bennett; rather, both coincided or Bennett influenced the institution. That RIE and GRI differed suggests both institutional differences in the importance of nutritional etiology to diagnosis and treatment, and the persistence of multiple explanations for tubercular disease — and disease more generally — in Scottish medical practice during the mid-Victorian period.

Table 6.10. Outcomes, 1841 – 1880

	GRI			RIE		
Outcomes	TB-Related	Non-TB Pulmonary	Other	TB-Related	Non-TB Pulmonary	Other
Advice	9	0	80	69[a]	47	707
By Desire	21	12	98			
Cured	95	706	9310	257	1414	15311
Died	240	157	1564	590	431	1848
Improper	0	0	7	22[b]	35	502
Irregular	15	24	151			
Relieved	391	322	1635	754	492	4258
Other	179	60	760	157	45	1184
[Blank]	0	1	30	12	20	251
P-Values[c]		0.0005***	0.0005***		0.0005***	0.0005***

[a] RIE staff collapsed the categories "advice" and "desire" under the single outcome "advice or desire" in the admission registers for the years 1860, 1865, 1869, and 1875. To accommodate this change, I treated these two categories as one for the years 1845, 1850, and 1855 as well.

[b] RIE staff similarly collapsed "improper" and "irregular" under the single outcome "improper or irregular" in the admission registers for the years 1860, 1865, 1869, and 1875. These categories were thus treated as for "advice or desire."

[c] All p-values were obtained through Monte Carlo simulation using 2000 replicates.

*** $p < 0.001$

HEREDITY, ENVIRONMENT, BEHAVIOR, AND DISEASE

The evident favor in which practitioners held the new nutritional explanations for TB and related disorders did not prevent the maintenance, and even resurgence, of older etiologies. One such was heredity, which tubercular diseases shared with other disorders held to result from an inborn diathesis, including nervous disease and hysteria (Rosenberg 1997, 29). Hereditary etiologies for these diseases had a long history: both Morton (1720, 66) and Buchan (1790, 176) listed an inherited predisposition among causes for consumption. "[E]verybody knows very well, that those who come of Consumptive Parents, are apt to fall into the same Distemper," commented the former (Morton 1720, 66). By the mid-Victorian

period, Fothergill (1874) was ambivalent regarding heredity, writing, "But tubercle is nothing unique or new, it is merely an alteration of normal nutrition, to which, however, some families are infinitely more liable than others. Under proper care and supervision many children of what are called consumptive families survive growth and reach old age, but much more frequently members of healthy families succumb to tubercle as a consequence of neglect of trivial complaints or of the commonest laws of health." (50) Despite his endorsement of nutritional etiology and his confidence that "No one, almost, can feel any certainty that he or she may not some day become the subject of tubercle," (ibid.) Fothergill maintained a hereditary construct of the disease.

The practitioners at GRI and RIE, too, continued — and even revived — hereditary explanations for TB and its analogues. Whereas my samples of prior periods at GRI yielded no mentions of family history of these diseases, during the mid-Victorian period practitioners at both RIE and GRI regularly recorded these details in TB-related cases. Practitioners' preoccupation with TB-related heredity was evident through both positive and negative notations: at both GRI and RIE, patients with TB-related diagnoses were more likely to be described as having a family history of such disorders and as not having such (table 6.11). Indeed, compared to patients with non-TB pulmonary disorders, those with TB-related diseases were more like to have a family history of any disorder noted.

Alternatively, records of family members' illnesses and causes of death could have represented practitioners' adherence to other etiologies — including contagious and environmental sources of disease. While this was the case for diseases considered contagious at the time such as cholera and smallpox, prior to 1880 evidence that tubercular diseases were contagious was largely rejected by the British medical establishment, particularly Bennett, as I will discuss in chapter seven; during the mid-Victorian period I saw no

evidence comparable to that I discuss in the next chapter regarding practitioners' perception of TB and its analogues as contagious. Environmental etiology during this period largely comprised two modes: constitutional weakening through unhealthy exposure to the elements as I discuss below and miasmatic infection through exposure to bad air and filth (e.g., Gavin 1847, 10). While both of these figured in practitioners' records of patients' histories, as I will address below, these were most often recorded in addition to family history. It is thus most likely that, except for disorders established as contagious during this period, clinicians inquired into and recorded patients' family histories out of concern surrounding hereditary predisposition — which, as I will discuss in the next chapter, became preoccupying in the late Victorian era — rather than contagion or environmental etiologies.

Table 6.11. Family histories of disease, 1841 – 1880

	Diagnosis Groups			
Institution	TB-Related	Non-TB Pulmonary	Other	TOTAL
GRI				
Patients	55	24	30	109
TB Heredity Total	10	1	2	13
TB-Related	*7*	*1*	*2*	*10*
TB-Related Unknown	*3*	*0*	*0*	*3*
Proportion TB Heredity	0.18	0.04	0.06	0.12
Family History, Other	10	1	7	18
Proportion, Other History	0.18	0.04	0.23	0.16
RIE				
Patients	210	95	112	417
TB Heredity Total	39	9	8	56
TB-Related	*23*	*3*	*2*	*28*
TB-Related Unknown	*16*	*6*	*6*	*28*
Proportion TB Heredity	0.19	0.09	0.07	0.13
Family History, Other	27	7	13	47
Proportion, Other History	0.13	0.07	0.12	0.11

This indicates a conceptual framework for TB and its analogues, shared by practitioners at both institutions, in which family health history mattered — patients suspected of having these disorders were questioned about their families' histories. Whether family members did or did not have similar disorders was worth noting by medical staff, the former owing to a possible hereditary origin of the patient's present disease, the latter as a negative symptom. In a number of cases, medical staff simply noted a negative family history as "unimportant," that is, the presence of similar disease in family members would have been important to diagnosis and treatment. Some practitioners seemed invested enough in hereditary roots of diseases like phthisis to express frustration when they could find no family history: "Family History Could get nothing of any importance No history of phthisis," noted the RIE clerk in the case of Eliza P. (LHB1/129/6/10, 1875: 184; emphasis retained), for example. Clearly, practitioners retained — and even increased — the causative role of heredity in their concepts of TB and related disorders into the mid-Victorian period, despite the development of newer, physiologically-based explanations for these diseases.

Practitioners similarly continued to invoke environmental explanations for disease across disorders. With respect to TB-related disorders specifically, environmental causes were considered, at least by the Royal Medical and Chirurgical Society (1861), to be perfectly obvious. Upon Henry McCormac's presentation of his paper "On the True Nature and Absolute Preventability of Pulmonary Consumption," (1861), a Dr. Chambers complained the paper "simply stated that close rooms, foul air, and other injurious influences, favoured the production of consumption. All knew this before, and it was a waste of time to enlarge upon a truism." (Royal Medical and Chirurgical Society 1861, 434) A Dr. Tanner added, "Dr McCormac's views on the subject of phthisis … were more than five hundred years old," after which a Dr. Little asserted, "The Society ought to be protected against the reading of

such productions." The discussion concluded with the Society's formal refusal to thank the unfortunate Dr. McCormac for his contribution (ibid.).

At GRI and RIE, medical staff and patients similarly accepted environmental etiologies. Across disorders, patients regularly traced their disease to exposure to unhealthy environments. I have already discussed the case of Thomas P., in which patient and practitioner agreed on the unhealthiness of the patient's confined, hot workshop (LHB1/129/2/54, 95). More commonly, damp and cold figured in the causation of TB and other disorders. Medical staff recorded patients' exposure to wet and/or cold in relation to the onset of their disorders in eight percent of all sampled cases at GRI and 40 percent of cases overall at RIE; the difference between institutions likely relates to RIE's more detailed case notes.

As in Thomas P.'s case, it was clear that environmental explanations for disease were not held by patients alone. In several instances, medical staff specifically asked regarding adverse environmental exposure. "[S]he has been exposed to cold and wet, but does not remember it being so particularly before she was taken ill," recorded the clerk in the diarrhea case of Mary S. (LHB1/129/5/34, 1860: 53); "had not been previously exposed to wet and cold and is unable to account for the attack," read the clerk's account of George McC.'s rheumatic fever case (LHB1/129/2/59, 1865: 9). In these and similar notations, the patients' negative statements regarding adverse exposure are likely to have been prompted by the examining practitioner — in reporting the history of one's illness, it would be unusual to state what did not make one ill. It is rather more likely that practitioners shared with patients the long-held construct of disease caused by adverse environments.

Nutritional, hereditary, and environmental etiologies have in common a construct of TB and its analogues as diseases of the whole body. Despite the increasing emphasis

clinicians placed on identifying pathological anatomy in understanding and diagnosing disease, in both theory and practice systemic disease concepts persisted. Newly emphasized by practitioners in such constructs during the Victorian era, however, was the patient's complicity in disease. Clinicians extended nutritional etiology to include not only the body's failure to process dietary fat, but also the patient's failure to eat it. Hereditary causes for these diseases entailed a responsibility for self-control in those affected: as I noted previously, John Forbes, in a footnote to his 1834 translation of Laënnec, urged "a most rigid system of prophylactic discipline" for children predisposed to tubercular disease (339). Environmental influences, as I will discuss below, could be guarded against, leaving those who failed to do so culpable for the resulting pathology. Medical staff at GRI and RIE revealed — more so than in earlier periods — belief in the role of patient behavior in the development of disease, rendering TB and related disorders pathologies not just of the whole body, but of the self.

Patient case notes from the period 1821 – 1880 at GRI and 1841 – 1880 at RIE demonstrated a qualitative increase in practitioners' concern with patient morality viewed through the lens of clinicians' own middle-class mores. Whereas my earliest sample (1794 – 1820) from GRI bore very little mention by medical staff of patients' behavior, from 1821 – 1840 and 1841 – 1880 practitioners noted patients' immoral behavior in nine percent and two percent of cases, respectively. At RIE, the period 1841 – 1880 yielded practitioner notations of patients' immorality — including intemperance in alcohol consumption or sex, sleeping out of doors, and imprisonment — in 15 percent of cases. This was an objectively small percentage, but notable in that mentions of morality increased with, rather than being replaced by, a more self-consciously scientific clinical posture.

GRI practitioners' comments on immorality are more limited than those at RIE: the majority of these for the period 1821 – 1840 concern sexual behavior and occur in cases of sexually transmitted disease, while those for the mid-Victorian period are few in number. This is likely the effect of a lesser social distance between patient and practitioner at GRI: the 1835 GRI annual report touted the affordability of that institution's medical training, boasting of its clinical lectures, "This important part of medical instruction is here very complete, at least equal to what is obtained at the London and Edinburgh schools, and is afforded to the pupils at much less expense. What is got for seven guineas in Glasgow, would in London cost above fifty, and in Edinburgh nearly twenty pounds." (HB14/2/4, 5) Crucially, however, even GRI clinicians' rarer notations of patient behavior contributing to disease are limited to the periods after 1820, marking practitioners' concern with patients' morality as a phenomenon of the circum-Victorian Era.

A relatively minor form of deviance, yet one practitioners felt compelled to note across disorders, was patients' failure to adequately protect themselves from adverse environmental conditions, most often cold and damp, but also night air. RIE phthisis patient Helen M., "having incautiously exposed herself to cold in the evenings," noted the clerk, "was suddenly seized with sharp cutting pain in left side" (LHB1/129/5/4 1850, 76). A decade later an RIE clerk commented of phthisis patient John P., "having imprudently exposed himself to wet … he caught a severe cold, which brought on the cough and expectoration more severe than it was before." (LHB1/129/2/29 1860, 147) In such instances, the patients' imputed responsibility for the exposure is made clear by medical staff.

In other cases, the patient's culpability is implied by the exposure's proximity to other irresponsible acts. Phthisis patient John R. stated, according to the RIE clerk, "he was always a strong healthy man till twelve months ago [i.e., June 1854] when in consequence of

getting tipsy and sleeping all night in a field he caught a severe cold which he neglected." (LHB1/129/2/48 1855, 103) Similarly, another John R.'s phthisis originated in January 1859 when he "caught cold, while attending Burn's [*sic*; i.e., Robert Burns] centenary, having stood in cold and got wet that night. The day after this he felt wearied and tired; does not remember whether he had any shivering, but he had been drinking all night." (LHB1/129/2/26, 91) Both of these patients compounded their exposure to the elements — itself considered risky, at least by medical staff — by excessive drinking.

Medical staff commonly associated intemperance in the use of alcohol with ill health. In cases of TB-related disorders, practitioners invoked excessive drinking even by patients' spouses to explain disease origins. The RIE clerk reported of phthisis patient Catherine S., "Three days previous to admission … her husband, a man of drunken habits, came home in the evening, abused her, took the bed clothes away and sold them. Not long after she was seized with rigors …. Next day the cough became more violent and for the first time she noticed she spat blood." (LHB1/129/5/22 1855, 55) Clinicians in some cases struggled to discover intemperance in patients' histories. John D. asserted twice during his intake examination that he was not a heavy drinker, the clerk noting, "States he has been regular in his habits …. he says he had not been drinking." (LHB1/129/2/47 1855, 113) Apparently doubting the patient's own report, after his death medical staff also asked his father about John D.'s drinking habits: "He [the patient's father] is certain he [John D.] had not more whisky than a glass or so for some weeks past." (117)

Practitioners also linked sexual and reproductive excesses and improprieties to disease. Indeed, medical staff could consider these diseases in and of themselves. George M. — the sexual hypochondriasis patient who had fallen into the hands of quacks — was said by the clerk to have become "addicted to self-abuse" at age fifteen, a fact related by close

juxtaposition to his having had scarlatina at twelve years of age (LHB1/129/2/26 1860, 66). At GRI, the clerk admitted Matthew T. under the diagnosis "masturbation" (HH67/9/3 1868, front matter): his entire intake record read, "[Patient] has been an habitual masturbator for eighteen yrs. His intellect and his bodily strength are undermined by the long indulgence of this habit. He is also affected by varicosele and by eczema scroti." (244) Notably, both Matthew T. and George M. were affected by bodily pathology — George M. was also treated for tapeworm, and Matthew T., as his clerk noted, had scrotal symptoms — but were admitted under diagnoses invoking their sexual conduct. In instances such as these, behavior trumped pathological anatomy in diagnosis.

For women, recovery from childbirth was a risky time, which medical staff regarded as requiring careful self-management to avoid disease. The RIE clerk, for example, traced Mary Ann N.'s phthisis to "About three months ago, while confined to bed after the birth of her sixth child," during which time "she unadvisedly sat up for some time one day." (LHB1/129/6/10 1874, 10) Practitioners during the mid-Victorian period also evidently shared Buchan's (1790) inclusion of "giving suck too long" (175) among the causes of TB-related disorders: of RIE pulmonary tubercle patient Alice C., a clerk disapprovingly noted, "she has been nursing her child up to the time she came into the infirmary, though it is now seventeen months old." (LHB1/129/5/44 1865, 42) Here, as for appropriate shelter from the elements and intemperance in alcohol or sex, practitioners attributed bodily pathology to behavior deviating from middle-class limits.

The relationship evident at RIE and GRI between physical pathology and behavior deviant from a middle-class perspective revealed the retention of an essentialist disease concept. Physical pathology rooted in behavioral pathology constructs disease as an expression of systemic rather than local influences: for Foucault (1963), the clinical sign is

local, while symptoms "refer … to a pathological essence." (161) TB and analogous conditions in my mid-Victorian sample were not diagnosed by pathological anatomy alone, despite the influence of anatomical reformers like Bennett. Rather, the combination of pulmonary pathology with an older, more symptomatic conception of these disorders and practitioners' reassertion of earlier behavioral etiologies resulted in a systemic disease construct. In this conception, physical and behavioral pathology together created a disease of the total self, the physical body and social actor. As I will discuss in the final section of this chapter, this conflation of pathological anatomy with behavioral pathology defined by middle-class norms of behavior also classified deviation from these norms as a disease of the nation's social body.

DISEASE IN THE SOCIAL BODY

As I have discussed, practitioners' increased understanding of anatomical lesions in TB and related disorders did not preclude their continued attachment to systemic models of these diseases in practice. Indeed, even new constructs of these disorders, such as the nutritional etiology favored by Bennett (1853), were rooted in essentialist disease concepts incorporating anatomical and behavioral pathology. Beyond holistic disease constructs — like that of TB and its analogues as disorders of nutrition and heredity — Bennett (1858), Heath (1863) and others advocated total examination of patients. Bennett advocated complete dissection (23); Heath encouraged medical students to consider the whole body rather than its parts in isolation (401). In examination, Bennett (1858) too insisted on the continued use of symptoms as well as signs in diagnosis, telling students, "Auscultation is only *one* of the means whereby we can arrive at a just diagnosis, and should never be depended on alone." (56; emphasis retained) Medical practitioners actively championed this

systemic view of disease while simultaneously asserting their authoritative knowledge of the body through technologies and increased access to postmortem dissection that allowed them to see within.

At the same time, practitioners' professional and class concerns led them to define their work as integral to the social fabric. The period from 1841 through 1880 was a high point in medical practitioners' professional anxieties. Bennett (1858) described to his students the challenges facing the profession: decrying a lack of public approbation and acclaim for the work of medical practitioners and scientists, Bennett lamented that wealth was the only "prize" available, and that open to charlatans as well as orthodox practitioners (9–16). Indeed, in Scotland many physicians and surgeons operated apothecary shops to make ends meet, a practice which in Glasgow and Edinburgh precluded membership in professional fellowships over concern it would degrade the status of the medical profession (Jenkinson 2012, 10).

In response to these concerns, as has been established by existing research, medical practitioners and sanitary reformers drew on the middle-class touchstones of work and philanthropy to cement their status. Middle-class masculinity was defined in part by work integral to the nation's well-being; middle-class identity relied on philanthropy toward the working classes (Joshi 2004, 355, 357). By portraying medicine as ministering to the poor in order to maintain working-class productivity and protect the health of the nation (cf. Brown 2010; Joshi 2004), practitioners aligned themselves with both of these pillars of middle-class identity. Thus, in addition to practitioners' dominance in relationship to their patients, discussed above, the mid-Victorian period was marked by practitioners' increasing social importance — well-placing them as arbiters of healthy and unhealthy behavior.

Unsurprisingly, the behaviors medical practitioners championed as the best means to maintain health coincided with middle class male ideals. Fothergill (1874), in his health guide "aim[ed] at the inculcation of those principles which ought to guide us in our search after health," (vii) returned frequently to the unhealthiness of industrial work and industrial workers. Their ill-health and behavior for Fothergill formed a vicious circle in which industrial work fostered poorly regulated behavior damaging to physical health: "Where man is left too much to his mere muscular efforts, without the mind being engaged, we find disease engendered …. [T]he mental lethargy entailed by a form of labour making no demand upon the intellectual powers, leave the persons engaged in such labour a prey to every form of excitement when the work hours are over. Drunkenness, political and theological agitation, bursts of excitement, and a sensational literature of the lowest order, are part of the price mankind pays for the development of industrial enterprise." (65)

Such intemperance became heritable, in that Fothergill traced its propagation across generations. Working-class children were subjected to the damaging effects of industrial work "too frequently in order to permit their father to spend a larger portion of his earnings in drunkenness and vice." (48) Such children would be doomed to perpetuate the cycle: "with the thriftless habits and gross improvidence of many of our working populace, … an improvement in the habits and in the thoughts of the parents must precede any general improvement." (48–49) In a work aimed at a middle-class audience, such information can only have been intended to inspire in readers a sense of their essential — not just professional or behavioral — difference from the pathological working classes, and their responsibility to maintain this difference for the health of future generations.

Women were the focus of much social anxiety, perceived as possessing both inherently shaky morals and unique influence over the moral and physical health of future

generations. Poovey (1986), for example, recounts mid-Victorian medical arguments for the essential pathology of women relative to men, summarizing, "hysteria is simultaneously the norm of the female body taken to its logical extreme and a medical category that effectively defines this norm as inherently abnormal." (146–47) Medical theorists considered reproductive events, in particular, as inflection points from which women could descend into psychological and physical disease (ibid.) — as was the case for medical practice.

As for the working class, the health of future generations depended on the physical and mental health of women. Children could inherit disordered nerves from their mothers' mental disturbance during pregnancy and lactation (Fothergill 1874, 34), an idea predating the anatomical revolution in medicine. "On the constitution of mothers depends originally that of their offspring. No one who believes this will be surprised, on a view of the female world, to find diseases and death so frequent among children," wrote Buchan on the subject in 1790 (7). Prevention of such inherited disease lay, of course, in adherence to middle-class norms of domesticity, which were even granted a medical function: Mrs. John Sandford, in her American paean to the domestic role of women, wrote approvingly of the "sedative" effects of "the duties which home involves."

Pathology was inherent for both women and the working class, then, in both medical theory and practice. In women and the working class lurked dangers to their own health and that of the population as a whole from which middle class men were, by their very definition — their self-definition — exempt. Immorality comprised "foul blood" passed to future generations, in the words of the social reformer Judith Butler (1879, 10) — a rare voice on the social cost of middle-class male deviance. Behavior engendered systemic pathology that spread from the individual to the entire social body. Just as the site of tubercular pathology was the lung, that of social pathology was women and the working class. But as tubercle in

the lung heralded the presence of systemic disease, so too was the pathology of these groups the bellwether for the health of society as a whole. The essential pathology of women and the working class was the manifestation of a diseased social body.

Mitigation of disease in the social body through "improvement" of the poor in particular formed a raison d'être for the Victorian middle class (e.g., Joshi 2004), albeit one for which enthusiasm paled as the Victorian Era wore on. Parliament expressed increasingly punitive attitudes toward the poor in the 1832 Anatomy Act (2 and 3 William IV c. 75), the 1834 Poor Law Amendment Act (4 and 5 William IV c. 76) and the 1845 Poor Law (Scotland) Act (8 and 9 Victoria c. 83). Private individuals, however, continued optimistic about aiding the poor. Donations to the infirmaries in the form of subscriptions formed one way to do so. Another was donations aimed at instilling working-class infirmary patients with religion: in 1855, the RIE board of managers received a donation from a well-intentioned but ill-informed individual intending to enable the infirmary to engage a missionary (LHB1/1/18, 474). In fact, RIE already employed two chaplains, and so the management board instead turned the funds to "suitable books for the patients." (ibid.)

During this period, however, middle class frustrations were mounting with the refractory working class. "If the artizan [*sic*] would but take somewhat more care of himself, his death rate would not be nearly so high, but how he is to be brought to see this, it is not so easy to say," commented Fothergill (1874, 69). Medical men criticized the indiscriminate charity provided by voluntary hospitals (e.g., "Where to Draw the Line" 1857; Coley 1858). D. Campbell Black, a Glasgow surgeon, struck a Scrooge-like note on the subject, criticizing voluntary hospitals for leaving the working class with money to spend on vice: "Stop this [hospital] charity and the working man will not be poor, he will not be improvident, and he will not be drunken … and the parochial board, the only defensible form of charity, will be

otherwise employed than in begetting pauperism, black-guardism, and crime." (1872, 60) By the mid-Victorian Era, medical writers, at least, were decidedly pessimistic on the possibility of curing the social disease wrought by the working class.

CONCLUSIONS

The combination of behavioral proscriptions and constructs of women and the working class as inherently pathological during the mid-Victorian Era influenced a new focus by practitioners on morality and health, both of the individual and the social body. While relationships between patient and practitioner increasingly privileged the practitioner in the years 1841 – 1880, medical staff turned to technology to "see" pathology within the patient. At the same time, the continued use of older, symptomatic constructs of TB and its analogues revealed essentialist models in which these diseases were systemic rather than localized. So, too, was the imagined social pathology that was thought to arise from the poorly regulated behavior of women and the working class. The resistance of the working class, in particular, to middle-class efforts to cure this disease of the social body led to pessimism regarding middle-class "improvement" of the working class. This in turn led — as I will discuss in the following chapter — to notions of the working class as degenerate and incapable of reclamation to middle-class ideals.

CHAPTER 7

DEGENERATION AND THE TUBERCLE BACILLUS, 1881 – 1905

On March 24, 1882, Robert Koch presented to the Berlin Physiological Society evidence that he had isolated the bacillus responsible for tuberculosis. In retrospect, many have hailed Koch's discovery as revolutionary; modern authors relate with relish the silence that greeted Koch at the conclusion of his presentation, casting the audience as "astounded" (Grange and Bishop 1982, 3) or quietly conscious of the weight of the occasion (Sakula 1982, 248). Indeed, at the time burgeoning support for germ theory in Europe rendered many medical practitioners and scientists keenly interested in such emerging evidence for microorganisms as a cause of disease, particularly a disease as significant as TB. In the United Kingdom, the reaction to Koch's tubercle bacillus was accordingly swift: both the *Lancet* ("Saturday, April 22" 1882) and the *British Medical Journal* (*BMJ*: "Recent Researches on Bacteria: I" April 29, 1882) reported the news within weeks of the April 10 publication of Koch's research in the *Berliner klinische Wochenschrift* (1882a). Both British publications emphasized Koch's methodology rather than his conclusions, however, suggesting that the British medical establishment approached Koch's revolution cautiously.

During the first half of the nineteenth century, practitioners' constructs of tubercular diseases began to incorporate internal lesions owing to the insights into pathological anatomy gained as a result of increased access to cadavers for dissection and applied using technologies like the stethoscope. Nonetheless, clinicians continued to use older, essentialist definitions of TB and analogous conditions such as scrofula and tabes mesenterica in which these diagnoses comprised systemic disorders — diseases of the body rather than of the lungs, glands, or digestive system alone. Thus, in Scottish hospital practice during the mid-

Victorian era, the rise of pathological constructs of TB and related disorders was accompanied by an increased emphasis on patient behavior, reflecting the increased concern of the Victorian middle class with the behavior of women and the working classes.

Here, I discuss GRI medical practitioners' incorporation of germ theory into the existing constructs of TB and related disorders I have described in prior chapters. Between 1881 and 1905, practitioners' acceptance of the tubercle bacillus accrued slowly, with the microorganism's utility in diagnosis outpacing its role in recasting phthisis as a contagious disease. My analysis of 18,606 admissions and the case notes of 258 individuals (table 7.1) reveals that medical examinations during the late-Victorian era became increasingly technology-driven, although new techniques like bacteriology complemented rather than replaced the suite of systemic and pulmonary signs and symptoms long associated with TB and its analogues. Continuing the mid-Victorian trend, at the close of the nineteenth century medical staff's scrutiny of their patients' private lives intensified, with practitioners recording details of with whom their patients lived, what their living conditions, and how they behaved. Such concerns were characteristic of members of the middle class interacting with the working class during the late-Victorian era, when evolutionary thought and pessimism regarding the global stature of the United Kingdom combined to cast the working classes as a degenerate race unto themselves (e.g., Luckin 2006; Pinell 2001; Rosenberg 1997; Stedman Jones 1971, c. 6, c. 16), which I discuss in greater detail below. Incorporating earlier views of tubercular disorders as hereditary, environmental, or nutritional into the new germ theory, medical practitioners began to depict the working classes — complicit in their own degeneration by their or their ancestors' rejection of middle-class values — as soil ripe for germination of the seeds of disease such as tubercle bacilli.

Table 7.1. GRI Admission registers and case notes samples, 1881 – 1905

Diagnosis Groups	Admission Registers[a]	Casenotes[b]
TB-Related	1605	124
Non-TB Pulmonary	1506	64
Other	15495	70
Total	18606	258

[a] Sampled years include 1885, 1895 and 1905.
[b] Sampled years include 1885, 1893, 1895, 1900 and 1905.

THE TB BACILLUS IN BRITAIN

Support by British medical practitioners for a contagious etiology of TB and related disorders began to accrue prior to Koch's 1882 identification of the tubercle bacillus, albeit gradually. During the 1860s, Villemin's research in France (collected in Villemin 1868) had demonstrated that inoculation with tuberculous material could induce tubercles in animals, a finding that medical men received with interest but not immediate acceptance in the UK. It was not until 1867, two years after Villemin's initial presentation of his findings, that the *Edinburgh Medical Journal* (*EMJ*) reported on Villemin's original experiments and those he had performed since, noting reservedly, "M. Villemin believes that his experimental researches have substantiated these inductive hypotheses," i.e., his hypotheses regarding the infectious nature of TB ("Cause and Nature of Tuberculosis" 1867, 757). In the same year, debate at the Pathological Society of London regarding the nature of tubercular material was informed by Andrew Clark's attempted replication of Villemin's experiments, in which Clark identified in experimental animals tubercles resulting from inoculation with tubercular as well as non-tubercular material ("What is Tubercle?" 1867). Following a similar experimental approach, Wilson Fox confirmed Clark's result the following year (Fox 1868).

It is likely that the homegrown challenges to Villemin's findings by Clark, Fox and others dampened early British enthusiasm for contagion as a cause of TB and its analogues, as did opposition from John Hughes Bennett, by then a leading authority on tubercular disease. Bennett took the opportunity of contributing the article on phthisis pulmonalis for J. Russell Reynolds' *System of Medicine* (1871) to flatly declare that with respect to phthisis, contagion and infection "have received no support from the [medical] profession." (Bennett 1871, 547) Despite this assertion, Bennett felt compelled to address Villemin's findings, requesting of Reynolds in a letter dated December 23, 1870 the opportunity to "add a Paragraph as to the inoculability of Phthisis, a subject which [h]as been developed since I wrote the Article." (Bennett 1870) In what is most likely the added paragraph, Bennett cited the work of Clark, Fox and others in drawing a parallel between tubercle formation and a generalized physiological response to putrid material, "in no way support[ing] the hypothesis that Phthisis Pulmonalis is contagious or infectious." (547–48)

By the years immediately preceding Koch's isolation of the tubercle bacillus, however, British practitioners had begun to warm somewhat to the idea of a contagious phthisis. In 1880, William Murrell of the Royal Hospital for Diseases of the Chest presented a case in the *Lancet* of "phthisis, probably of contagious origin" (802) in a young woman without prior phthisical history who developed the disease shortly after her husband's death from it. "It is certainly a question worth considering," concluded Murrell, "whether in cases of advanced consumption with copious expectoration there is not some danger of infection, however slight." (ibid.)

Murrell's diffidence was justified in light of the sometimes-vitriolic opposition to contagion theory at the time: in November 1881 William Dale of Norfolk wrote to the *Lancet* to rail against "the renewed, — daily renewed, and totally unproven views [of

contagion] which find utterance in the present literature of phthisis." (Dale 1881, 1108) "We, who have practised our profession upwards of three decades," Dale wrote, "on recalling our professional experience during all these years we believe we have not seen a single case, or barely a single case, which even suggested the idea of contagion. I cannot help thinking that these ever-recurring statements respecting the contagiousness of phthisis depend either on the lack of a full history … being obtained, or the ignoring of the constitutional and predisposing causes which are present." (ibid.)

Just four months later, on March 24, 1882, Koch presented his findings on the tubercle bacillus in Berlin. His presentation was published on April 10 (Koch 1882a); the *Lancet* ("Saturday, April 22" 1882) and the *BMJ* ("Recent Researches on Bacteria: I" 1882; "Recent Researches on Bacteria: II" 1882) reacted within weeks, publishing detailed synopses of Koch's methods along with commentary on the results. First to press on the subject on April 22, the *Lancet* ventured that Koch's findings "may be the inauguration of a new era" in disease etiology and prevention (655). Even so, however, the *Lancet*'s editors would not go so far as to acknowledge the tubercle bacillus as the ultimate cause of tubercular disease: "The pathological importance of the discovery of the *proximate* cause of this frightful scourge of the human race cannot be over-estimated." (656; emphasis added)

The *BMJ* divided their coverage into two articles under the unassuming heading "Recent Researches on Bacteria." The first, on April 29, described Koch's methods in depth, commenting only circumspectly on his results: "He [Koch] is a worker on whose observations and accuracy the most implicit reliance can be placed, and those who have had the pleasure of seeing him at work will hesitate before they find fault with his statements." (656) The second, on May 13, again summarized Koch's "beautiful" experiments but went a step further, stating that tuberculosis was infective "beyond a doubt" (706) and concluding,

"[Koch's] investigations seem very definite, and only require careful confirmation to enable us to accept the matter as proven." (707)

Despite the *BMJ*'s hearty endorsement of infective TB, the *Lancet*'s provisional acceptance of the tubercle bacillus as *a* cause — but not *the* cause — of tubercular disease was more characteristic of discussion in the English medical literature for decades following Koch's announcement. Michael Worboys (2000, 193–233; 2001) has traced these debates in British medical literature, identifying continuity rather than revolution in both theory and practice following Koch's 1882 announcement. As practitioners viewed the tubercle bacillus through the lens of their existing constructs of TB and analogous disorders, Worboys argues, they brought contagion into alignment with older models of these diseases rather than abandoning these models in favor of bacterial etiology.

Indeed, within a year of Koch's announcement British practitioners could be found vociferously endorsing the tubercle bacillus as fact while their attitudes surrounding its role in disease causation remained ambivalent. In August 1882, C. Theodore Williams of the Brompton Hospital for Consumption presented to the British Medical Association data on the paucity of tubercular disease among hospital staff despite the surely dense environmental distribution there of "our friends the bacilli." (Williams 1882, 619) "Phthisis is not, in the ordinary sense of the word, an infectious disease; the opportunities for contagion being most numerous, while the examples of its action are exceedingly rare," (620) he concluded.

In response, Clifford Allbutt — President of the Medical Section of the Association and recent inventor of the clinical thermometer (Rolleston and Bearn 2004) — criticized Williams' guarded language in discussion of the bacillus. "[I]t was scarcely correct to speak of the 'bacillus theory': it was the bacillus fact. Whatever the inference that might be drawn from it, the fact was perfectly indisputable, that there was not merely a bacillus, but a

peculiar phthisis bacillus, which belonged to the disease, and was not found accidentally." (Allbutt 1882, 621) Despite this energetic defense of the bacillus, however, Allbutt admitted, "How far this would help in understanding the spread of phthisis was, of course, another thing." (ibid.) In response, Williams explained that "he did not question the existence of the bacillus," but that in his conception, the bacillus caused inflammation secondary to the underlying consumptive disease (Williams 1882, 621).

By February 1883, Williams had confirmed the presence of tubercle bacilli in patient sputum and offered a proof of concept for contagion by demonstrating their abundance in the ventilation system of the Brompton Hospital (Williams 1883). Reiterating that phthisis was not contagious "in the ordinary sense of the word," instead "requiring a previous weakening of the constitution," he nonetheless continued to acknowledge its role in disease and began to advocate "the disinfection and removal of the sputum" as a preventive measure (313). Williams claimed this measure "was carried out at the Brompton Hospital long before Koch's discovery of the bacillus," (ibid.) although in August of the previous year he had reported, "The spittoons of the patients are changed two or three times a day; but, until lately, unless the odour was unpleasant, no attempt was made to disinfect them." (Williams 1882, 619) This revisionist view of practices at Brompton reveals that Williams, at least, was anxious to position his institution at the cutting edge of research on tubercular disease by claiming long adherence to anti-infective practices — confirming a growing role for contagion in British constructs of tubercular disease in spite of dissemblance regarding its relative unimportance.

Debate over the "true causation" (N. W. 1886, 87) of tubercular disease persisted to the end of the nineteenth century (Worboys 2000, 231), and included many of the major etiologies popular prior to Koch's discovery: inflammation, heredity, and nutrition.

Hearkening to older inflammatory models for the disease and making only a passing reference to bacilli, R. J. Shepherd of St. Olave's Union Infirmary in London insisted, "*Irritation* … is the *cause* of phthisis." (1888, 1000; emphasis retained) Heredity, too, retained its place in clinical diagnosis. "The account she gave of her family history was very indefinite — that is to say, it was impossible to fix an hereditary taint," lamented the house physician in an 1889 case related to the *Lancet* by Dr. Evershed of the North London Consumption Hospital (852). Neither Shepherd (1888) nor Evershed (1889) mentioned any contribution of contagion to phthisis.

Indeed, by the turn of the century, some English practitioners had heard quite enough about tubercle bacilli and contagion. In 1894, J. K. Fowler wrote the *Lancet* to criticize a recent Royal Medical and Chirurgical Society presentation (Squire 1894) minimizing the role of heredity in phthisis. "The general tone of the discussion clearly showed a tendency to ignore the importance of the hereditary factor and to insist upon infection as the only point worthy of consideration," Fowler complained. "I hold that it will be a grave error if the profession should be led by the fascination of this element to the problem to cast aside the experience derived from centuries of observation." (Fowler 1894, 1514)

Similar pleas continued into the twentieth century. In 1901, William R. Gowers of University College London echoed Fowler, writing, "There has come to be a strong popular impression that an inherited influence in the causation of tuberculosis has been disproved by the discovery of its dependence on organisms received from without. The belief is apparently due to some sweeping unqualified statements which have been made that we must discard the idea of actual inheritance of the disease. This, of course, is true," he admitted, but nonetheless advocated continued attention to heredity: "the facts still remain

which have led to the belief that an inherited condition influences the occurrence of the disease." (Gowers 1901, 1007)

W. Roger Williams of Bristol hastened to second Gowers' opinion. In his *Lancet* letter, which ran just one week after Gowers', Williams "emphatically … endorse[d]" Gowers' views (1901, 1080) and went on to argue for the continued importance of nutrition in preventing tubercular disease, downplaying the recent concern surrounding zoonotic TB acquired from tainted beef and dairy. "Because a few living tubercle microbes were found in a roll of beef that had been cooked in the ordinary way a learned commission somewhat hastily concluded that the disease was therefore spread by eating such meat, &c. … Plenty of good meat — which generally contains some tubercle bacilli — is one of the chief safeguards of crowded urban populations against tuberculosis." (ibid.) The essentialist etiologies of the nineteenth century, then, were carried into the twentieth by English practitioners' acceptance of tubercular contagion as contingent upon inflammation, heredity and nutrition.

In published literature at the start of the twentieth century, practitioners continued to attribute TB and its analogues primarily to factors within the patient, and only secondarily to bacilli "received from without." (Gowers 1901, 1007) Despite having rapidly accepted a role for contagion and the tubercle bacillus following Koch's 1882 announcement, however, the English medical establishment continued to debate the nature of that role for decades.

BACTERIOLOGY COMES TO GRI

In Scotland, the immediate response to Koch's discovery was quieter. Neither the *Glasgow Medical Journal* (*GMJ*) nor the *EMJ* reviewed Koch's (1882a) publication on the tubercle bacillus; indeed, although the editorial staff and readers of both certainly were

familiar with Koch's findings, neither printed anything on the subject until July 1882. In that month, the printed proceedings of a Glasgow Southern Medical Society meeting held on the first of June included a presentation by Dr. N. Carmichael, the society's president, on the pharmacology of oil of gaultheria (i.e., wintergreen; acid methyl salicylate) and oil of spiraea (i.e., meadowsweet; salicylous acid, a precursor to aspirin) (1882). In the discussion that followed, a Dr. A. Napier — most likely Alexander Napier, a *GMJ* editor — commented that these oils' putative antiseptic action "would prove most valuable in the treatment of cystitis and of phthisis. The recent announcement of Koch's views as to the pathology of tubercle would make us resort more largely to the use of antiseptic inhalation in the treatment of this disease." (67) In September 1882, Napier summarized refinements by British and German researchers to Koch's staining process to visualize tubercle bacilli, developed in the six months since Koch's initial announcement (1882a). Because this piece opens, "Since Koch's views on tuberculosis have come to be so generally adopted, or at least looked upon favourably," (221) and because Napier reported that Koch himself now favored one of the new methods (ibid.), it is clear from these two pieces that the *GMJ*'s editors were closely following scientific developments surrounding the tubercle bacillus and assumed their readers wished to stay abreast of the most current methods for their detection.

The *EMJ* responded to Koch's findings in a markedly different tone. The editors' earliest commentary was an oblique reference in September 1882 in the form of a satirical piece, reprinted from Philadelphia's *Medical News* (1882), purporting to detail the efforts of a Wisconsin physician to isolate the "parasite which infests the human trousers and causes broken legs." ("New Parasites," *Medical News* 1882, 286) The unnamed author characterized this effort as "rather more valuable than the discovery of Prof. Koch," (285) at least — the piece winkingly noted, congratulating its readers on their more cultivated understanding —

"So far as the non-scientific mind can perceive." (286) The following month, a reviewer of Frederick Treves' *Scrofula and its Gland Diseases* referred in passing to the "established" (1882, 324) link between bacteria and tubercle — the author thus assuming familiarity with Koch's research and some acceptance thereof among the *EMJ*'s readers.

The *EMJ* made no further comment on the subject until December, by which time the editors' jocular mood had returned: they reprinted a story from New York's *Medical Record* (Shrady 1882) ostensibly describing a Chico, California physician's isolation of a bacillus causing "love madness" and the humorous results of his inoculation of test subjects — a fifty-year-old bachelor, a "young lady of forty-five," and a male teenager — therewith ("A New Disease," *EMJ* 1882, 573). The editors of the *EMJ*, then, assumed their readers' interest in and awareness of Koch's findings as did the editors of the *GMJ*, although the *EMJ* left earnest reports on the subject to other publications.

Lacking from the pages of both the *GMJ* and *EMJ* is the immediate, lively debate over Koch's discovery and its import evident in the *Lancet* and *BMJ*. Both Scottish publications printed a number of pieces on tubercular disorders — case studies, book reviews, and new treatments — early in 1882 (*GMJ*: Napier 1882a; Young 1882; Main 1882; J. A. A. 1882; Steven 1882) (*EMJ*: "Davos Platz" 1882; Craig 1882). However, these cease after February 1882 in the *EMJ* and May 1882 in the *GMJ* and do not reappear in 1882 with the exception of the five described above (i.e., Napier's comment on Carmichael 1882; Napier 1882b; "New Parasites," *EMJ* 1882; "Scrofula and its Gland Diseases" 1882; "A New Disease," *EMJ* 1882). Only three of these Scottish pieces on tubercular disease published in the latter half of 1882 comment substantively on the topic — that is, Napier's comment on Carmichael's presentation (1882), Napier (1882b) and the review of *Scrofula and its Gland*

Diseases (1882) — the latter of which addresses a work published prior to Koch's announcement.

By the 1890s, Scottish physicians were again discussing the tubercle bacillus, joining in national conversation on the subject in the *Lancet*. In October 1893, the *Lancet* reported on a public address by Dr. Anderson, Medical Officer of Health for Dundee, in which he criticized a lack of government and individual action against the spread of TB and urged his audience to undertake disinfectant measures to prevent the disease ("Tuberculosis and its Prevention" 1893). The previous year, members of the Medico-Chirurgical Society of Glasgow had written the *Lancet* with a resolution, passed by the group in December 1891, stating human TB to be infectious and urging that public health measures be taken against it as they had for bovine TB (Coats et al. 1892). Characteristic of British understanding of the disease at the time, however, the authors of the resolution did take care to note, "There are doubtless other elements in the causation" aside from the microbe (218). In Scotland as well as England, then, medical discourse acknowledged a role for contagion in TB and related disorders — and even actively campaigned for the control of infection — while often at the same time upholding older, non-contagious etiologies for these diseases.

DIAGNOSTIC CONTINUITY IN THE BACTERIOLOGICAL ERA

The tensions evident in British medical literature surrounding the growing experimental support for contagious tubercular disease are also present in Scottish medical practice in the period 1881 – 1905. This practice revealed practitioners incorporating infective etiology into their constructs of TB and related disorders as modification of, rather than replacement for, more essentialist explanations. My quantitative analysis illustrates the continued dependence of tubercular diagnoses on signs and symptoms distributed

throughout the body — neither strictly localized to the affected organs nor strictly dependent upon bacteriological findings after sputum examination commenced in the 1890s.

During the late-Victorian period, TB and its analogues were highly distinct from non-TB, non-pulmonary disorders (table 7.2), and were significantly differentiated from these diagnoses in more categories of clinical indicators than in earlier periods at both GRI (tables 3.3, 4.3 and 6.2) and RIE (table 6.3): see appendix M to visually compare significance across institutions and time periods. However, the opposite was true of tubercular diseases with respect to non-TB pulmonary disorders, indicating the progressive de-emphasis of several signs and symptoms critical for diagnosis of the former prior to the increasingly internalized, anatomical constructs of disease that gained favor over the course of the nineteenth century.

Cases bearing TB-related diagnoses differed significantly ($p < 0.05$) or near-significantly ($p < 0.10$) from those diagnosed as non-TB, non-pulmonary disorders in all but two categories of clinical indicators: personal history and mouth and throat. I obtained significant differences in the categories appearance and comportment, circulatory, dermatological, digestive, general, genitourinary, medical history, pain, neurological, general respiratory, and miscellaneous (see appendix G for a complete list of clinical indicators and their frequencies); the remaining two respiratory classes, auscultation/percussion and chest form and sensation, approached significance. Compared to the period 1841 – 1880 at both GRI and RIE, two categories — neurological and general — gained significance or near-significance. With respect to GRI, general respiratory also gained significance while respiratory auscultation/percussion and chest form and sensation gained near-significance. In comparison with the mid-Victorian period at RIE, personal history and mouth and throat

lost significance, and the respiratory classes auscultation/percussion and chest form and sensation went from significant to near-significant.

Table 7.2. P-values for Fisher's exact tests of differences in clinical indicators, 1881 – 1905

Signs and Symptoms	TB-Related vs. Non-TB Pulmonary	TB-Related vs. Other
1. Appearance and Comportment	0.0005[a]***	0.0005[a]***
2. Circulatory	0.0220[a]*	0.0005[a]***
3. Dermatological	0.0001***	0.0009***
4. Digestive	0.5106	0.0118*
5. General	0.2951	0.0196*
6. Genitourinary	0.1919[a]	0.0425[a]*
7. History, Medical	0.0250[a]*	0.0075[a]**
8. History, Personal	0.0005[a]***	0.1359[a]
9. Mouth and Throat	0.5248	0.7372
10. Pain	0.0350[a]*	0.0005[a]***
11. Neurological	3.89×10^{-5}***	0.0283*
12. Respiratory, General	0.0005[a]***	0.0005[a]***
13. Respiratory, Auscultation/Percussion	0.0475[a]*	0.0795[a†]
14. Respiratory, Chest Form/Sensation	0.1977	0.0832[†]
15. Miscellaneous	0.0006***	0.0020[a]**

Note: See appendix G for a complete list of signs and symptoms comprising each category.
[a] P-value obtained through Monte Carlo simulation using 2000 replicates.
[†] $p < 0.10$
* $p < 0.05$
** $p < 0.01$
*** $p < 0.001$

In comparison with non-TB pulmonary diagnoses, TB-related disorders were distinguished significantly by signs and symptoms in the categories appearance and comportment, circulatory, dermatological, medical history, personal history, pain, neurological, general respiratory, respiratory auscultation/percussion, and miscellaneous (table 7.2). This represents a gain in significance compared to the mid-Victorian period at both GRI and RIE in neurological signs and symptoms, and a loss in significance in the categories genitourinary and chest form and sensation. Compared to the period 1841 – 1880

at GRI, digestive signs and symptoms also lost significance. With respect to the mid-Victorian RIE, GRI in the late-Victorian period gained significance in circulatory and pain indicators and went from near-significant to significant in medical history.

Some of these differences from earlier periods at GRI and RIE are likely attributable to variations in sample size. The total clinical indicators per patient in my late-Victorian GRI sample was 23.3, comparable to the 25.4 indicators per patient at RIE for the period 1841 – 1880 and greater than earlier values at GRI: 16.5 (1794 – 1820), 19.9 (1821 – 1840), and 15.0 (1841 – 1880). Thus, the loss of significant difference in mouth and throat symptoms between TB-related and non-TB, non-pulmonary diagnoses at GRI in the period 1881 – 1905 compared to RIE in the period 1841 – 1880 likely results from the relative popularity of these clinical indicators at RIE, at which physicians tended to record more detailed and anatomically complete examination notes than did practitioners at GRI, perhaps owing to the reduced importance of GRI as a teaching institution beginning in 1874.

Similarly, the pattern in which pulmonary auscultation and percussion signs between TB-related and non-TB, non-pulmonary diagnoses range from insignificantly different at GRI in the mid-Victorian period to approaching significance in the late-Victorian GRI sample and significant at RIE reflects the frequency of these signs in the three samples. Physicians relatively rarely recorded auscultation and percussion findings in non-TB, non-pulmonary cases in all three settings, but did so most frequently under the RIE's thorough examination protocol (0.89 indicators per patient), followed by the similarly dense examinations at the late-Victorian GRI (0.80) and the brief case notes of the mid-Victorian GRI (0.26). Thus, it is likely only in the former two settings that the number of observations allowed statistical differentiation.

Others of these quantitative findings are more informative as to GRI practitioners' concepts of TB and its analogues during the late-Victorian period. Medical staff continued to note the classic, systemic indicators of TB-related disorders alongside detailed pulmonary signs and symptoms: still frequently recorded among these diagnoses are weight loss, debility, night sweats, and anorexia. Some newer systemic indicators lost diagnostic utility in the period 1881 – 1905, however. Contrary to the mid-Victorian period, genitourinary symptoms significantly distinguished TB-related diagnoses from non-TB, non-pulmonary disorders but not from other pulmonary diseases.

This distinction in the significance of genitourinary indicators between the mid- and late-Victorian periods likely results in part from a decline during the latter in the frequency with which medical practitioners recorded female patients' amenorrhea overall, but particularly in TB-related cases (table 7.3). In the early-Victorian period, GRI practitioners recorded amenorrhea for one-third of female patients with TB-related disorders and nearly one-fourth of sampled female patients overall; values for the mid-Victorian period at GRI were similar, with the records of one-third of female TB-related patients and 21 percent of sampled female patients overall bearing mentions of amenorrhea by medical staff. At the mid-Victorian RIE, physicians recorded this symptom for 38 percent of female TB-related patients and 30 percent of female patients overall. During the late-Victorian period, however, GRI practitioners recorded amenorrhea in only 23 percent of records for female patients with tubercular diagnoses and 18 percent of sampled female patients overall. The reason for the decline is unclear, but may indicate a reduced reliance by practitioners on disease constructs related to humoral flow, imbalance of which could stop the menses.

Table 7.3. Amenorrhea, 1794 – 1905

Time Period	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
1794 – 1820, GRI				
Female Patients	9	5	6	20
Amenorrhea	2	0	3	5
Proportion Amenorrhea	0.22	0.00	0.50	0.25
1821 – 1840, GRI				
Female Patients	12	8	9	29
Amenorrhea	4	1	2	7
Proportion Amenorrhea	0.33	0.13	0.22	0.24
1841 – 1880				
GRI				
Female Patients	46	21	25	92
Amenorrhea	15	0	4	19
Proportion Amenorrhea	0.33	0.00	0.16	0.21
RIE				
Female Patients	99	40	53	192
Amenorrhea	38	8	13	57
Proportion Amenorrhea	0.38	0.20	0.25	0.30
1881 – 1905, GRI				
Female Patients	44	17	27	88
Amenorrhea	10	3	3	16
Proportion Amenorrhea	0.23	0.18	0.11	0.18

Thus, while practitioners continued to report amenorrhea in clinical records with appreciable frequency — indicating physicians' continued interest in female pathology — in the late-Victorian period instances of amenorrhea were distributed more evenly across diagnosis categories. This suggests that by 1881 – 1905, medical staff associated amenorrhea with a broader range of disorders than earlier in the Victorian Era. In this respect, then, TB and its analogues became less distinctive in the late-Victorian period: both tubercular and non-TB pulmonary disorders could produce the systemic perturbations resulting in

amenorrhea. Whether this change resulted from clinicians viewing non-TB pulmonary diseases as increasingly systemic or perceiving female patients as generally more susceptible to reproductive disorder is unclear. Nevertheless, practitioners' relatively recent zeal for recording amenorrhea in tubercular cases in particular had faded somewhat by the 1880s while many older systemic indicators for these diseases retained their popularity, indicating that classic descriptions of TB-related disorders retained their clinical utility.

Localized signs and symptoms long held to be indicative of TB-related diagnoses also continued to be so, as in the case of hemoptysis. Practitioners continued to associate expectoration of blood almost exclusively with TB and its analogues when they deemed it coughing blood, whereas less severe instances — bloody or blood-streaked sputum — they tended to class with non-TB pulmonary cases, continuing a trend established in the mid-Victorian period.

While medical staff thus significantly utilized in diagnosis signs and symptoms localizing these diseases within individual anatomical systems (i.e., the lungs in pulmonary phthisis or the glands in scrofula), they nonetheless continued to conceptualize these disorders as diseases of the whole body, systemic rather than local. Indeed, in this period practitioners recorded a history of strumous diseases and disorders suggestive of non-pulmonary TB — including glandular swellings, knee or spine disease, and hydrocephalus — with greater frequency than previously (table 7.4), which in part drove the slight gain in medical history's significance between TB-related and non-TB pulmonary disorders at GRI in this period compared to RIE. This further indicated practitioners' understanding of TB and analogous conditions as related clinically as well as pathologically: having experienced one TB-related disorder was increasingly pertinent to the diagnosis of a second. Contemporaneous Scottish views of bovine TB were similar; an 1889 Glasgow court case

found on the strength of testimony from Scottish veterinarians, physicians and surgeons that when TB was found in meat, the whole carcass should be condemned as unsafe for consumption rather than just the affected parts (reviewed in Worboys 2000, 223).

Table 7.4. Strumous history

	Diagnosis Groups			
Time Period	TB-Related	Non-TB Pulmonary	Other	TOTAL
1841 – 1880				
GRI				
Patients	55	24	31	110
Strumous History	2	0	1	3
Proportion Strumous	0.04	0.00	0.03	0.03
RIE				
Patients	210	95	112	417
Strumous History	7	0	1	8
Proportion Strumous	0.03	0.00	0.01	0.02
1881 – 1905, GRI				
Patients	124	64	70	268
Strumous History	15	4	2	21
Proportion Strumous	0.12	0.06	0.03	0.08

Note: Medical staff recorded no instances of strumous history in my sample prior to 1841.

Tubercular etiology at GRI during the period 1881 – 1905 also continued to reflect essentialist disease concepts. Practitioners' concern with tubercular heredity was particularly evident. Their notation of the presence or absence of TB-related disease and unspecified hereditary taint in patients' families — absent from my sample prior to 1841 — increased across all classes of diagnosis from 0.12 instances per patient at GRI and 0.13 at RIE in the mid-Victorian period to 0.47 instances per patient at GRI in the late-Victorian period (table 7.5). Unsurprisingly, medical staff recorded a tubercular family history most frequently in TB-related cases, illustrating a role for heredity in the clinical etiology for these diseases.

However, practitioners were nearly equally likely to record the absence of hereditary taint in general or of tubercular history specifically in any diagnosis category: this likely drives the loss of significance in personal history symptoms between TB-related and non-TB, non-pulmonary disorders at the late-Victorian GRI relative to RIE. This concern with family history furthermore demonstrates a preoccupation of medical staff with hereditary explanations for disease across disorders, as well as a pervasive concern with hereditary susceptibility to tubercular disease in particular, both of which relate to the broader social concern with degeneration of the British population I discuss below.

Unlike hereditary explanations for TB and analogous disorders, the nutritional etiology favored by John Hughes Bennett and others seems to have borne relatively little clinical weight at GRI in the late-Victorian period. Digestive symptoms, for example, lost significance with respect to non-TB pulmonary disorders compared to earlier Glasgow practice. This category had significantly differentiated tubercular cases from both non-TB pulmonary and non-TB, non-pulmonary cases at GRI from 1821 through 1880, likely owing to the underrepresentation of constipation and overrepresentation of diarrhea among TB-related cases (table 7.6). However, during the period 1881 – 1905, as at RIE in the mid-Victorian period, only the frequency of diarrhea — increased among tubercular patients — differed appreciably between TB-related and non-TB pulmonary disorders. GRI medical staff in the late-Victorian period furthermore recorded diarrhea relatively infrequently: clinicians noted this indicator in only six percent of cases across disorders at the late-Victorian GRI, compared with 12 percent and 14 percent at the mid-Victorian GRI and RIE, respectively.

Table 7.5. Family histories of disease, 1841 – 1905

Time Period	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
1841 – 1880				
GRI				
Patients	55	24	30	109
TB Heredity Total	10	1	2	13
TB-Related	*7*	*1*	*2*	*10*
TB-Related Unknown	*3*	*0*	*0*	*3*
Proportion TB Heredity	0.18	0.04	0.06	0.12
Family History, Other	10	1	7	18
Proportion, Other History	0.18	0.04	0.23	0.16
RIE				
Patients	210	95	112	417
TB Heredity Total	39	9	8	56
TB-Related	*23*	*3*	*2*	*28*
TB-Related Unknown	*16*	*6*	*6*	*28*
Proportion TB Heredity	0.19	0.09	0.07	0.13
Family History, Other	27	7	13	47
Proportion, Other History	0.13	0.07	0.12	0.11
1881 – 1905, GRI				
Patients	124	64	70	268
TB Heredity Total	72	26	27	125
TB-Related	*24*	*6*	*3*	*33*
TB-Related Unknown	*48*	*20*	*24*	*92*
Proportion TB Heredity	0.58	0.41	0.39	0.47
Family History, Other	63	29	25	117
Proportion, Other History	0.51	0.45	0.36	0.44

Note: Medical staff recorded no mentions of such heredity in my sample prior to 1841.

Table 7.6. Bowel function

Time Period	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
1794 – 1820, GRI				
Patients	25	13	13	51
Constipation	7	4	3	14
Proportion Constipation	0.28	0.31	0.23	0.27
Diarrhea	7	0	1	8
Proportion Diarrhea	0.28	0.00	0.08	0.16
1821 – 1840, GRI				
Patients	38	25	25	88
Constipation	3	8	12	23
Proportion Constipation	0.08	0.32	0.48	0.26
Diarrhea	9	1	1	11
Proportion Diarrhea	0.24	0.04	0.04	0.13
1841 – 1880				
GRI				
Patients	55	24	31	110
Constipation	14	13	13	40
Proportion Constipation	0.25	0.54	0.42	0.36
Diarrhea	12	0	1	13
Proportion Diarrhea	0.22	0.00	0.03	0.12
RIE				
Patients	210	95	112	417
Constipation	59	26	27	112
Proportion Constipation	0.28	0.27	0.24	0.27
Diarrhea	36	7	14	57
Proportion Diarrhea	0.17	0.07	0.13	0.14
1881 – 1905, GRI				
Patients	124	64	70	268
Constipation	32	17	22	71
Proportion Constipation	0.26	0.27	0.31	0.26
Diarrhea	12	2	3	17
Proportion Diarrhea	0.10	0.03	0.04	0.06

The reduced clinical interest in diarrhea at GRI in the period 1881 – 1905 most likely reflects practitioners' diminished concern with deranged digestion as a cause of tubercular disease, or at least as a clinically useful indicator thereof. Alternatively, this could result from a more general decline in clinicians' use of humoral flux and stasis — and an attendant obsession with metabolism and bowel function — to conceptualize disease. Late-Victorian GRI practitioners continued to record constipation at a similar frequency to medical staff at GRI prior to 1841 and at RIE (table 7.6), however, indicating their continued belief that the state of the bowels had bearing on their patients' health across diagnosis categories. Rather, clinicians recorded diarrhea less frequently in cases of TB-related disorders in particular, suggesting a reduced role for this indicator in tubercular disease specifically.

GRI practitioners' general lack of concern regarding patients' dietary preferences further indicates their disinterest in nutritional explanations for tubercular disorders. In the period 1841 – 1880, GRI clinicians had recorded no instances of patient dietary preferences (appendix E). From 1881 through 1905, medical staff noted dietary preferences in one (one percent) TB-related case, in which the patient reported the least difficulty digesting sour milk (HH67/8/18 1905, 407), and two (three percent) non-TB pulmonary cases, in which the patients reported sickness from meat and fat (HH67/7/11 1905, 377) and vegetables agreeing best (HH67/12/5 1900, 175, 181) (appendix G). At RIE, such mentions by medical staff were not overwhelmingly frequent — occurring in eleven TB-related cases (five percent) and two non-TB, non-pulmonary cases (two percent) (appendix F) — but were more common and in tubercular cases tended to hew to the dislike of fat associated by Bennett (1871, 554), Fothergill (1874, 41), and others with the deranged digestion characteristic of tubercular disease.

In practice at GRI, then, clinicians do not seem to have acted upon the nutritional etiology for TB and its analogues that Bennett espoused at Edinburgh. This is not likely to have resulted from a loss of nutritional explanations for these disorders in Britain more generally: British medical writers continued to reference nutrition's contribution to tubercular disorders into the twentieth century, as in W. Roger Williams' (1901) hearty endorsement of beef-eating to prevent tubercular disease discussed above. Instead, the place of nutrition in British practitioners' conception of TB and its analogues was likely changing in a manner tending to de-emphasize its utility in diagnosis. By 1905, the author of an American volume on dietetics stated confidently, "The [tubercle] bacillus produces from the tissue or 'soil' in which it grows a toxin or poisonous substance … which modifies nutrition" (Thompson 1905, 462). As the cause rather than the effect of deranged nutrition, infection with the bacillus in this view would become the ultimate cause of TB-related disorders; as such, it would also become the sine qua non of diagnosis.

THE INTRODUCTION OF BACTERIOLOGICAL METHODS

British medical literature during the late-Victorian period was dominated by ambivalence rather than Thompson's (1905) confidence regarding the role of the bacillus in tubercular disease; a similar uncertainty was revealed in GRI clinicians' interactions with the tubercle bacillus. It is clear from Napier's (1882b) methodological overview in the *GMJ* that within months of Koch's presentation of his discovery, Glasgow practitioners would have had some familiarity with the bacillus and its detection in patients' sputum. Nonetheless, the earliest mention of tubercle bacilli in my sample did not occur until the January 1893 case of Bernard McK., an Irish laborer diagnosed with phthisis. "Sputum is abundant, frothy and

muco-purulent and slightly tinged with blood," noted the clerk, adding on a separate line, "Tubercule [*sic*] Bacillus found." (HH67/7/5, 3)

It is likely that this decade-long delay between Koch's announcement and the application of bacteriological techniques in practice at GRI stems in part from the poor preservation of GRI patient records for the 1880s and 1890s. No GRI case notes volumes are extant for 1882; for the period 1883 – 1888, only four surgical volumes are preserved, in which medical staff recorded extremely brief diagnostic notes. The period 1889 – 1891 again lacks preserved records. The scarce extant records for patients admitted in 1892 largely comprise continuation of treatment rather than intake, and thus include no diagnostic notes. Intake records for the late-Victorian period were therefore not available in sufficient detail or quantity to assess the presence of bacteriological testing prior to 1893.

From 1893 through 1905, however, practitioners' use of bacteriological techniques occurred in my sample with increasing frequency. As I show in table 7.7, Bernard McK. was one of only three patients in my 1893 sample for whom medical staff recorded bacteriological results, whether positive or negative. In that year's sample, practitioners noted such results only for patients with TB-related disorders. Clerks recorded no bacteriological observations among my small 1895 sample, but in 1900 and 1905 microscopic examination of the sputum — or rarely, the feces or urine — for bacilli was much more commonplace. By late 1900, the staff of a dedicated bacteriological laboratory conducted testing for tubercle bacilli, pneumococci, and Widal's reaction — indicative of typhoid — and communicated the results to ward clinicians by means of pre-printed forms completed with the patient's personal information, bacteriological observations, and the bacteriologist's signature (e.g., HH67/8/9 1900, attachment to page 71). Practitioners in 1900 and 1905 noted bacteriological results in 40 percent or more of TB-related cases (table 7.7). Clinicians

furthermore extended bacteriology to 15 percent of non-TB pulmonary cases in my sample beginning in 1900 and 30 percent of such cases in 1905. By 1905, 27 percent of all cases in my sample bore some mention by medical staff of bacteriological examination.

Table 7.7. Bacteriological examination

	Diagnosis Groups			
Year	TB-Related	Non-TB Pulmonary	Other	TOTAL
1885				
Patients	3	0	3	6
Bacteriology Recorded	0	0	0	0
Proportion Bacteriology	0.00	0.00	0.00	0.00
1893				
Patients	36	20	19	75
Bacteriology Recorded	3	0	0	3
Proportion Bacteriology	0.08	0.00	0.00	0.04
1895				
Patients	5	4	2	11
Bacteriology Recorded	0	0	0	0
Proportion Bacteriology	0.00	0.00	0.00	0.00
1900				
Patients	30	13	18	61
Bacteriology Recorded	14	2	0	16
Proportion Bacteriology	0.47	0.15	0.00	0.26
1905				
Patients	50	27	28	105
Bacteriology Recorded	20	8	0	28
Proportion Bacteriology	0.40	0.30	0.00	0.27

Bacteriology was not the only new examination method popular in the late-Victorian period. The gains in significance I observed in general and neurological signs and symptoms were largely driven by the new popularity among medical staff of thermometry and reflex-testing, respectively (appendix G): clinicians recorded thermometer readings in 44 percent and reflex observations in 22 percent of all sampled cases. Taken together, the frequency

with which practitioners utilized bacteriological techniques, thermometry, and/or reflex testing represent a continued trend from earlier periods, in which medical staff increasingly relied upon instrument-mediated signs in diagnosis. Unlike thermometry and reflexes, however, bacteriology linked diagnosis with etiology, offering unique insights as to the ways in which practitioners incorporated Koch's discovery into their concepts of tubercular disease.

Medical staff employed bacteriological testing in TB-related cases primarily in confirming rather than identifying tubercular disease, indicating that the bacillus was secondary to pathological anatomy in practical constructs of these disorders. Practitioners reported examination of the sputum, urine and/or feces for bacilli — whether bacilli were found or not — most commonly in association with TB-related diagnoses, secondarily in cases of non-TB pulmonary disease, and never in cases of non-TB, non-pulmonary disorders (table 7.7). Furthermore, all but one case in which clinicians reported examining the sputum, urine and/or feces multiple times bore tubercular diagnoses (table 7.8).

Table 7.8. Single and multiple bacteriological examinations

	Diagnosis Groups			
	TB-Related	Non-TB Pulmonary	Other	TOTAL
Patients	124	64	70	258
One Examination	26	9	0	35
Two Examinations	9	0	0	9
Three Examinations	2	1	0	3
Proportion Single Exam	0.21	0.14	0.00	0.14
Proportion Multiple Exams	0.09	0.02	0.00	0.05

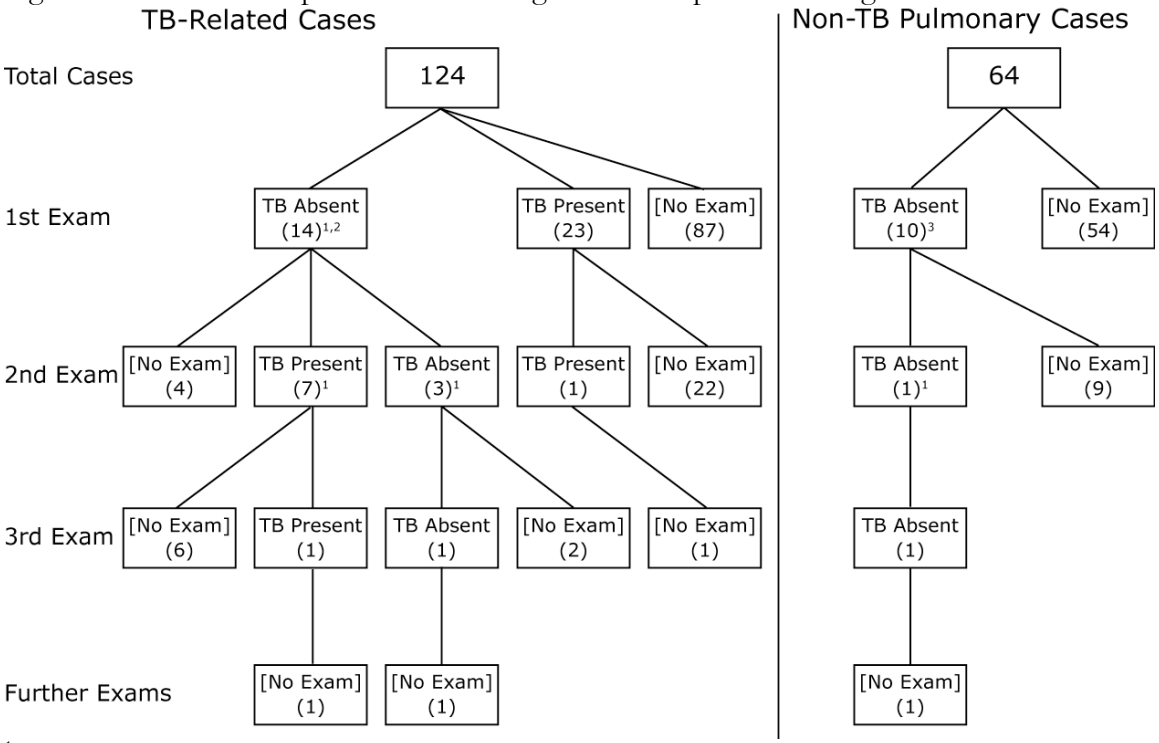
Figure 7.1 illustrates the pattern of bacteriological testing and retesting for TB-related and non-TB pulmonary disorders. For the most part, GRI practitioners utilized bacteriological examination to confirm the presence of tubercle bacilli assumed to be present following clinicians' physical examination of patients. As a result, medical staff tended to downplay negative bacteriological results in cases of tubercular disease. A practitioner might explain away a negative result in order to strengthen his case in favor of the positive reexamination. "The sputum was examined on Oct[ober] 25th, but no TB [i.e., tubercle bacilli] were found owing to quantity of blood present," reported the clerk in the hemoptysis case of Charles O'N., "but on the 29th, when there was much less blood in the sputum which was muco-purulent in places [and] had the characteristic discrete appearance, TB were found present." (HH67/5/3 1900, 367) Medical staff also tended to cast the absence of tubercle bacilli as provisional, noting not that these organisms were unequivocally absent but that the bacteriologist failed to locate them in the particular sample examined: "The sputum was examined on the 28th when no TB were found," for example. (HH67/12/6 1900, 49)

This bias against negative bacteriological findings in cases of tubercular disease was further revealed in clinicians' replication of bacteriological tests most commonly where they had anticipated but failed to confirm the presence of tubercle bacilli. Of the 14 TB-related cases with negative bacteriological findings upon the first examination, GRI medical staff reexamined 10; three of these were again negative on the second examination, of which clinicians examined one a third time (figure 7.1). Thus, of the 18 bacteriological examinations in TB-related cases yielding no tubercle bacilli, 11 (61 percent) were repeated.

In contrast, medical staff reported TB bacilli absent from 12 sputum samples of non-TB pulmonary patients, only two of which (17 percent) were repeated (figure 7.1). In fact, both retests were performed upon the same individual, the lung cancer patient James J.

Finding no tubercle bacilli in the first sputum sample on September 16, 1905, the bacteriologist examined a second sample on September 19 with negative results. On September 26 he reported finding no tubercle bacilli in a third sample through both direct examination and the sole 48-hour culture noted in my sample.

Figure 7.1. Schematic representation of single and multiple bacteriological examinations



[1] Pneumococci were reported in one case.
[2] Streptococci and diplococci were reported together in one case.
[3] Pneumococci were reported in four cases.

James J.'s case history explains the GRI clinicians' unusually persistent scrutiny of his sputa. "The patient's appearance is not that of ordinary bronchitis," (HH67/12/20, 145) summarized the ward physician, J. Lindsay Steven, on September 15. "The clinical history … and the physical signs first recorded are on the whole very suggestive of a neoplasma maligna of the right lung." (157) That medical staff went on despite this assessment to examine James J.'s sputa thrice for tubercle bacilli is not surprising; his case history and

examination also overlap with those of tubercular cases in a number of key points. Upon the patient's admission on September 8, the clerk noted "expectoration of blood, loss of flesh and debility," (139; emphasis retained), as well as prior hospitalization for a digestive disorder (ibid.), all of which are suggestive of TB-related disorders. The clerk also recorded in James J.'s family history a brother who "died at the age of thirty-five of 'inflammation of the lungs following a cold' after a year's illness" (141; emphasis retained): the brother's age, the length of his illness, its origin in a cold, and its pulmonary location are together strongly indicative of phthisis. The clerk's emphasis upon this point in particular, alongside his underscoring the blood in the patient's sputum and his weight loss and debility, make it clear that tubercular disease would need to be ruled out — hence the assiduous efforts by medical staff to confirm the absence of tubercle bacilli in James J.'s sputa.

In the case of Alexander L., GRI's clinicians again sought to rule out tubercular disease — in this instance unsuccessfully. Admitted December 12, 1900, Alexander L.'s initial diagnosis of chronic bronchitis was later amended to phthisis, as evidenced by the apparent insertion of the latter in the casebook index (HH67/12/5, front matter). His diagnosis posed some difficulty: in an initial summary of the patient's case, the practitioner noted, "Chronic bronchitis of eighteen years duration in all characterised latterly by somewhat frequently recurrent attacks of haemoptysis. … No positive evidence of tubercular infection of the lung, although crackling rales are apt to be present over the right upper lobe than elsewhere." (166) Clearly considering tubercular disease among Alexander L.'s possible diagnoses, the clerk also recorded results of a sputum examination at admission, reporting diplococci and streptococci present but no tubercle bacilli (169). The possibility of tubercular disease in Alexander L.'s case was rejected more forcefully upon reflection. Two days after his admission, an entry attributed to "D'r [*sic*] Steven" — almost certainly J.

Lindsay Steven, recorded in the volume's front matter as the ward's visiting physician — noted, "The case is evidently one of chronic bronchitis …. The rales in the right infraclavicular region disappear with coughing and it is possible that they may be of bronchial origin. The patient's aspect is that of a chronic bronchitis the expression being anxious and the lips rather livid." (177)

Despite these two repudiations of tuberculosis, Dr. McCrorie (i.e., the staff bacteriologist David McCrorie: HB14/2/11 1905, 4) examined Alexander L.'s sputum again on April 2, 1901, at which time he reported having found tubercle bacilli (HH67/12/5, 275). While it is not certain whether McCrorie's findings precipitated the change in diagnosis noted in the volume's index, this is distinctly likely. The clerk recording the April 2 entry noted that Alexander L.'s general condition, hemoptysis and physical signs of the chest all remained similar to prior notes. Over the course of his treatment, these had remained largely unchanged from admission, when medical staff had insisted twice that they were indicative of chronic bronchitis — once explicitly denying tubercular infection (166). Alexander L.'s case, then, may represent a sole sampled instance in which bacteriological findings superseded physical examination in diagnosis.

The overall pattern of bacteriological testing indicates that GRI medical staff believed negative results among TB-related cases — but not non-TB pulmonary cases — warranted retesting; in contrast, positive results were duplicated for only two patients in my sample. In the first of these exceptions, phthisis patient Christina B.'s sputa were retested more than two months after her March 6, 1905 admission, evidently to ascertain whether her condition required further treatment. The bacteriologist found "TB abundant" in Christina B.'s sputum on March 8 (HH67/10/5, attachment to page 52); on May 31, examination of her chest having yielded "no physical sign of disease" (63), medical staff again sent her

sputum for analysis and again found to contain tubercle bacilli (attachment to page 52). Apparently on the strength of this result, she remained at GRI for another three weeks — despite her "very much improved" (front matter) health — before being dismissed on June 20 to a sanatorium (63).

A second instance of practitioners replicating positive bacteriological findings occurred in the case of Francis M., admitted December 15, 1905. "Microscope exam'n [examination]. No TB have been found so far," (HH67/12/20, 357) noted the clerk at Francis M.'s admission, making clear his expectation that they would be. The clerk linked this comment to an addendum on the facing page, reporting the result of a second examination the following day: "A few TB found." (356) Four days later, an entry headed, "Bacteriological report on examination of sputum" strengthened this finding, noting less equivocally, "TB present." (ibid.) Thus, in both Christina B.'s and Francis M.'s cases, medical staff had particular reasons for duplicating positive bacteriological results: in the former case clinicians wished to assess Christina B.'s need for further treatment, while in the latter they strengthened an equivocally positive result by replication.

The evident assumption by GRI medical staff that negative results in TB-related cases were less trustworthy than positive was shared by practitioners at other institutions. Of bacteriological methods at the Brompton Hospital for Consumption, C. Theodore Williams wrote, "the rule adopted has been in the case of a negative result to repeat the examinations two, three, or four times so as to ensure accuracy." (1883, 312) Rosemary Wall (2011) identified a similar approach in her analysis of bacteriological methods at St. Bartholomew's Hospital in London and Addenbrooke's Hospital in Cambridge: at these elite institutions, as at GRI, negative bacteriological results did not preclude a TB-related diagnosis (786). However, Wall's London and Cambridge practitioners also tended to mistrust such negative

findings. Medical staff examined the sputum of an 1894 pulmonary tuberculosis patient at St. Bartholomew's six times before the bacilli were identified on the seventh attempt, while clinicians at Addenbrooke's Hospital performed nine examinations of an 1889 tubercular patient's sputum before obtaining a single positive result (787).

By extension, practitioners at GRI as well as the Brompton, Addenbrooke's, and St. Bartholomew's Hospitals considered positive bacteriological results inherently accurate, demonstrating that clinicians held false negatives to be a greater limitation of bacteriological testing for TB than false positives. This in turn encouraged clinicians' continued reliance on physical examination techniques rooted in pathological conceptions of TB-related disorders. GRI medical staff during the late-Victorian period therefore incorporated the microscopic identification of tubercle bacilli as a tool to confirm TB-related diagnoses at which they had arrived through observation of signs and symptoms associated with these disorders earlier in the nineteenth century, using long-established methods of physical and discursive examination. As a result, the clinical constructs of these diseases at GRI remained overwhelmingly defined by pathological anatomy rather than Koch's bacillus.

The strong retention at GRI of pathological constructs for TB-related disorders could stem from the loss of institutional prestige in the 1870s I discussed in chapter six. Despite GRI's loss in stature, however, the institution's clinicians kept pace with their colleagues at the Brompton, St. Bartholomew's and Addenbrooke's Hospitals in their adoption of bacteriological techniques. Wall (2011) reports that in each year between 1881 and 1905, St. Bartholomew's medical staff tested from none to all pulmonary tuberculosis patients for tubercle bacilli, with this value most often ranging between 50 and 70 percent; at Addenbrooke's Hospital, the proportion of pulmonary tuberculosis patients examined bacteriologically ranged from five to 50 percent (786, figure 2). While the former outstripped

the zero to 47 percent I observed at GRI (table 7.7), the latter comfortably overlaps GRI's values, indicating comparable levels of interest in bacteriological examination at GRI and Addenbrooke's Hospital, at least.

Taken together, the establishment of a bacteriology laboratory with dedicated staff along with the frequency with which clinicians called upon this facility to examine patients' sputum, feces or urine speak to the commitment of GRI's medical staff and board of management to the bacteriological methods introduced in the late nineteenth century. The manner in which these methods were applied, however, demonstrates that Koch's discovery did not substantially alter clinical constructs of tubercular disorders at this institution. Diagnosis of these diseases remained rooted primarily in pathological anatomy retaining clear vestiges of much older essentialist disease concepts, with bacteriology contributing only secondarily. Failure of medical staff to identify tubercle bacilli does not seem to have affected diagnosis: the differential treatment of negative results in TB-related and non-TB pulmonary cases demonstrates that clinicians had arrived at their diagnoses before bacteriological results were returned. In practice as well as theory, practitioners for the most part adapted the tubercle bacillus and bacteriological findings to existing constructs of tubercular disease rather than the reverse.

CONTAGION, CURABILITY AND TREATMENT

Contagion was the most notable exception to clinicians' tendency to reframe the tubercle bacillus within existing disease constructs. Villemin's research in the 1860s had ignited debate among British medical practitioners regarding the possibility of a contagious etiology for tubercular disease; Koch's 1882 identification of the tubercle bacillus offered a mechanism through which such contagion could occur. Nonetheless, it was not until 1900

that practitioners at GRI incorporated contagion into their recorded discussion of patients' tubercular disease. By December 1900, at least one clinician referred to "tubercular infection," (HH67/12/5, 166) in the case of Alexander L. described above — the casual nature of this reference indicating that tubercular infection was a concept familiar to GRI medical staff. At least some patients, too, ascribed to an infectious origin for TB-related disorders at this time. Of Mrs. W., admitted December 17, 1900, the clerk recorded, "She attributes the onset of the illness to infection from her husband. The husband suffers from pulmonary tuberculosis and most of his family also suffer from this disease." (HH67/8/9, 379) The clinician may not have wholeheartedly accepted this explanation, recording only, "Question of contagion" regarding the topic in the case summary (388).

The visiting physician for both of these cases was J. Lindsay Steven, long in favor of a contagious etiology for tubercular disease: eight years prior, Steven had cosigned the Medico-Chirurgical Society of Glasgow's resolution on the infectiousness of TB communicated to the *Lancet* (Coats et al. 1892). Even when familiar with the concept of tubercular infection and under the direction of a physician with a record of supporting a contagious tubercular etiology, then, GRI medical staff continued to express reservations regarding an infectious origin for tubercular disease. This underscores the trepidation with which Glasgow practitioners incorporated bacteriology into their existing clinical constructs of TB-related disorders, even as late as the turn of the century. Indeed, explicit references to tubercular contagion were exceedingly rare in my sample, further supporting a clinical construct of TB and its analogues overwhelmingly characterized by older concepts rooted in pathological anatomy.

Like diagnostic procedures, the approaches to treatment employed by GRI practitioners in cases of TB-related disorders did not change radically in response to Koch's

discovery of the tubercle bacillus. During the period 1881 – 1905, antispasmodics, expectorants, and unclassified treatments significantly ($p < 0.05$) differentiated between TB-related and non-TB, non-pulmonary disorders; cathartics, diaphoretics, and sedatives approached significance ($p < 0.10$: see table 7.9 for quantitative results regarding treatments and appendix L for a complete listing of treatments and their frequencies during this period). With respect to non-TB pulmonary disorders, TB-related cases bore significantly different frequencies of cathartic, sedative, tonic, and unclassified treatments, while expectorants approached significance.

The results shown in table 7.9 represent a small net increase in the number of treatment categories significantly differentiating between TB-related and non-TB diagnoses, whether the latter were pulmonary or non-pulmonary (see appendix N to visually compare significance across institutions and time periods). This suggests a further refinement in treatment specificity compared to earlier periods at GRI and RIE. The late-Victorian period also saw a reduction in prescriptions for treatments that had long been standbys: bleeding, alcohol, and opiates. During the period 1821 – 1840 — the peak of its popularity in my sample — GRI practitioners prescribed bleeding in a total of twenty-three (61 percent) TB-related cases and sixty (68 percent) cases overall. By the mid-Victorian period, medical staff prescribed bleeding much more rarely, likely owing to the decline of humoral etiologies and, at RIE, John Hughes Bennett's opposition to the practice. GRI clinicians employed these treatments in only one (two percent) TB-related case, while RIE medical staff prescribed bleeding in seventeen (eight percent) TB-related cases and fifty-two (12 percent) cases overall. The frequency with which GRI practitioners prescribed bleeding in the late-Victorian period was comparable to their practice in the immediately preceding period, with

no TB-related patients receiving these treatments and only two (less than one percent) patients prescribed leeches overall.

Alcohol and opiates, too, became less popular treatments in the late-Victorian period (table 7.10). Prescriptions for alcoholic beverages were rare prior to 1841 both in TB-related cases and across disease categories, having been prescribed in 10 percent or fewer of all cases; however, the mid-Victorian period saw a dramatic increase in practitioners' use of these treatments, which rose to include more than one-fourth of TB-related cases. By the late-Victorian period, however, GRI clinicians had again cooled toward alcohol, prescribing its use in only one in ten cases of TB and its analogues and 13 percent of cases overall. Opiates had been extremely popular throughout the nineteenth century, practitioners having never prescribed them in fewer than half of TB-related cases until the late-Victorian period. During the period 1881 – 1905, however, only twenty-two (18 percent) tubercular patients received these drugs. As I have discussed, patients' overuse of alcohol was of appreciable concern to practitioners beginning in the mid-Victorian period at RIE owing to increased social scrutiny of the working classes; social concern with such intemperance peaked in 1879 with the passage of the Habitual Drunkards Act (42 and 42 Victoria c. 19), which allowed inebriates to be legally committed to "retreats." As a result, concern with patient immorality — including intemperance with respect to alcohol — became even more prevalent in the late-Victorian period. Opiate use, too, was a focus of moral panic in last quarter of the nineteenth century over fears of its contribution to degeneration among the working classes (Berridge 1978, 109–110). The precipitous decline in prescriptions of alcohol and opiates at the close of the nineteenth century is most likely in response to these moral concerns with the drugs' use.

Table 7.9. P-values for Fisher exact tests of differences in treatments, 1881 – 1905

Treatments	TB-Related vs. Non-TB Pulmonary	TB-Related vs. Other
1. Anodyne	—	—
2. Antacid	0.3156	0.4208
3. Antispasmodic	0.2774	0.0123*
4. Astringent	0.684	0.41
5. Attenuant	—	—
6. Bleeding	—	—
7. Cathartic	0.0362*	0.0820†
8. Corrosive	0.2643	0.1513
9. Demulcent	0.1071	1
10. Diaphoretic	0.8656	0.0882†
11. Diuretic	0.2005	0.1183
12. Emetic	1	1
13. Emollient	—	—
14. Expectorant	0.0663†	0.0374*
15. Mercury	1	1
16. Opium	1	1
17. Refrigerant	—	—
18. Sedative	0.0327*	0.0656†
19. Stimulant	0.3142	0.3689
20. Tonic	0.0058**	0.4978
21. Indeterminate Function	0.7483	0.4034
22. Unclassified Drugs	0.0015[a]**	0.0005[a]***

Note: See appendix L for a complete listing of treatments comprising each category.
[a] P-value obtained through Monte Carlo simulation using 2000 replicates.
† $p < 0.10$
* $p < 0.05$
** $p < 0.01$
*** $p < 0.001$

Table 7.10. Alcohol and opium prescriptions, 1794 – 1905

Time Period	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
1794 – 1820, GRI				
Patients	25	13	13	51
Alcohol	1	4	0	5
Proportion Alcohol	0.04	0.31	0.00	0.10
Opium	14	7	5	26
Proportion Opium	0.56	0.54	0.38	0.51
1821 – 1840, GRI				
Patients	38	25	25	88
Alcohol	2	0	0	2
Proportion Alcohol	0.05	0.00	0.00	0.02
Opium	36	13	7	56
Proportion Opium	0.95	0.52	0.28	0.64
1841 – 1880[a]				
GRI				
Patients	55	24	31	110
Alcohol	15	9	7	31
Proportion Alcohol	0.27	0.38	0.23	0.28
Opium	29	5	2	36
Proportion Opium	0.53	0.21	0.06	0.33
RIE				
Patients	210	95	112	417
Alcohol	92	31	40	163
Proportion Alcohol	0.44	0.33	0.36	0.39
Opium	145	76	57	278
Proportion Opium	0.69	0.80	0.51	0.67
1881 – 1905, GRI				
Patients	124	64	70	268
Alcohol	12	15	8	35
Proportion Alcohol	0.10	0.23	0.11	0.13
Opium	22	11	7	40
Proportion Opium	0.18	0.17	0.10	0.15

[a] Opium prescriptions from 1841 and later include prescriptions of chlorodyne, a laudanum-containing compound drug.

Two new treatments are also notably absent from GRI practice in the late-Victorian period: the antiseptic inhalations suggested by Napier in response to Koch's discovery (response to Carmichael 1882, 67), and the tuberculin treatment developed by Koch himself (reviewed in Gradmann 2001, 19–27). With respect to the former, GRI practitioners employed inhalations only rarely — in eight cases (three percent) overall — and these generally comprised non-antiseptic botanical preparations and amyl nitrate (appendix L). Koch's tuberculin treatment, an injection of prepared tubercle bacillus culture, caused a great deal of excitement upon its announcement in 1890. Koch himself presented on the subject at the British Congress on Tuberculosis, held in London in July 1901 (Koch 1902); tuberculin was one of three major topics — alongside climate and sanatoria — for papers in the medical section (British Congress on Tuberculosis 1902, vol. 3, iii–vii). Participants broadly accepted tuberculin's use in diagnosis at the 1901 congress, and indeed such use continues today.

Tuberculin's therapeutic value was highly controversial (see, for example, discussion following Koch's presentation, British Congress on Tuberculosis 1902, vol. 3, 96–116). However, congress attendees reported that tuberculin treatment had been attempted with varying success in institutions throughout Europe and in the United States (ibid.). GRI was not such a hospital: there was no record in my sample of clinicians employing tuberculin in either the diagnosis or treatment of TB-related disorders. In contrast, Thomas McCall Anderson of the University of Glasgow spoke at length and from first-hand experience on tuberculin at the British Congress on Tuberculosis, referencing his use of the treatment in cases at Western Infirmary (Anderson 1902). This distinction likely reflects Western Infirmary's new preeminence and places it ahead of GRI in the adoption of new therapies.

Turn-of-the-century GRI practitioners instead hewed somewhat more closely to mid-Victorian nutritional therapies; cod liver oil, special diets, and tonics were popular among GRI's clinicians in treating TB-related disorders. However, practitioners reported giving cod liver oil to only 15 percent of tubercular patients in my late-Victorian sample, compared to nearly twice this at the mid-Victorian GRI and more than three times as many at RIE (table 7.11). Practitioners replaced cod liver oil in part by newer remedies purported to have similar effects, including Angier's Emulsion, a petroleum product marketed as a substitute for cod liver oil (Council on Pharmacy and Chemistry 1914, 962). Also becoming popular at the turn of the century were tonics such as Parrish's and Fellow's Syrups containing phosphate compounds, iron, and other ingredients. These were billed as "chemical food" (Reed 1998, 212), fortifying and easily absorbed nutrition for patients with "defective appetite as well as imperfect assimilation [i.e., of nutrition from food] [and] general debility," (Fellows 1882, 10) therefore fulfilling a similar role to that Bennett (1841) described for cod liver oil.

Together, late-Victorian GRI practitioners prescribed cod liver oil, Angier's Emulsion and phosphorous tonics 39 times (31 percent) among TB-related patients, a proportion similar to the frequency with which they prescribed cod liver oil alone in the period 1841 – 1880 (29 percent, table 7.11). The enthusiasm of GRI medical staff for nutritional treatments, then, had yet to be replaced by new treatments at the cutting edge of TB research. Thus, in neither diagnosis nor treatment did Koch's discoveries usher in a sea change for GRI practitioners; at the close of the nineteenth century, older constructs of the disease and its treatment were for the most part maintained.

Table 7.11. Cod liver oil prescriptions, 1841 – 1905

Time Period	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
1841 – 1880				
GRI				
Patients	55	24	31	110
CLO	16	1	1	18
Proportion CLO	0.29	0.04	0.03	0.16
RIE				
Patients	210	95	112	417
CLO	103	5	2	110
Proportion CLO	0.49	0.05	0.02	0.26
1881 – 1905, GRI				
Patients	124	64	70	268
CLO	19	2	0	21
Proportion CLO	0.15	0.03	0.00	0.08

Note: Medical staff did not prescribe cod liver oil in my sample prior to 1841.

The period 1881 through 1905 did see a change in recorded outcomes for patients with tubercular disorders, however. For the first time, GRI medical staff reported more patients with TB-related diseases discharged cured than died. Table 7.12 shows these outcomes from my sample of admissions registers. As in earlier periods, the distribution of outcomes in cases of TB and its analogues differed significantly from those of both non-TB pulmonary disorders and non-TB, non-pulmonary diseases, largely owing to the large proportion of the latter two categories of patients dismissed cured. Unlike earlier periods, in which the number of TB-related patients dismissed cured never ranked above the third most common and always ranked behind the number of these patients dying in hospital (tables 3.5, 4.6, 6.10), in the late-Victorian period only "relieved" was a more frequent state at dismissal than cured.

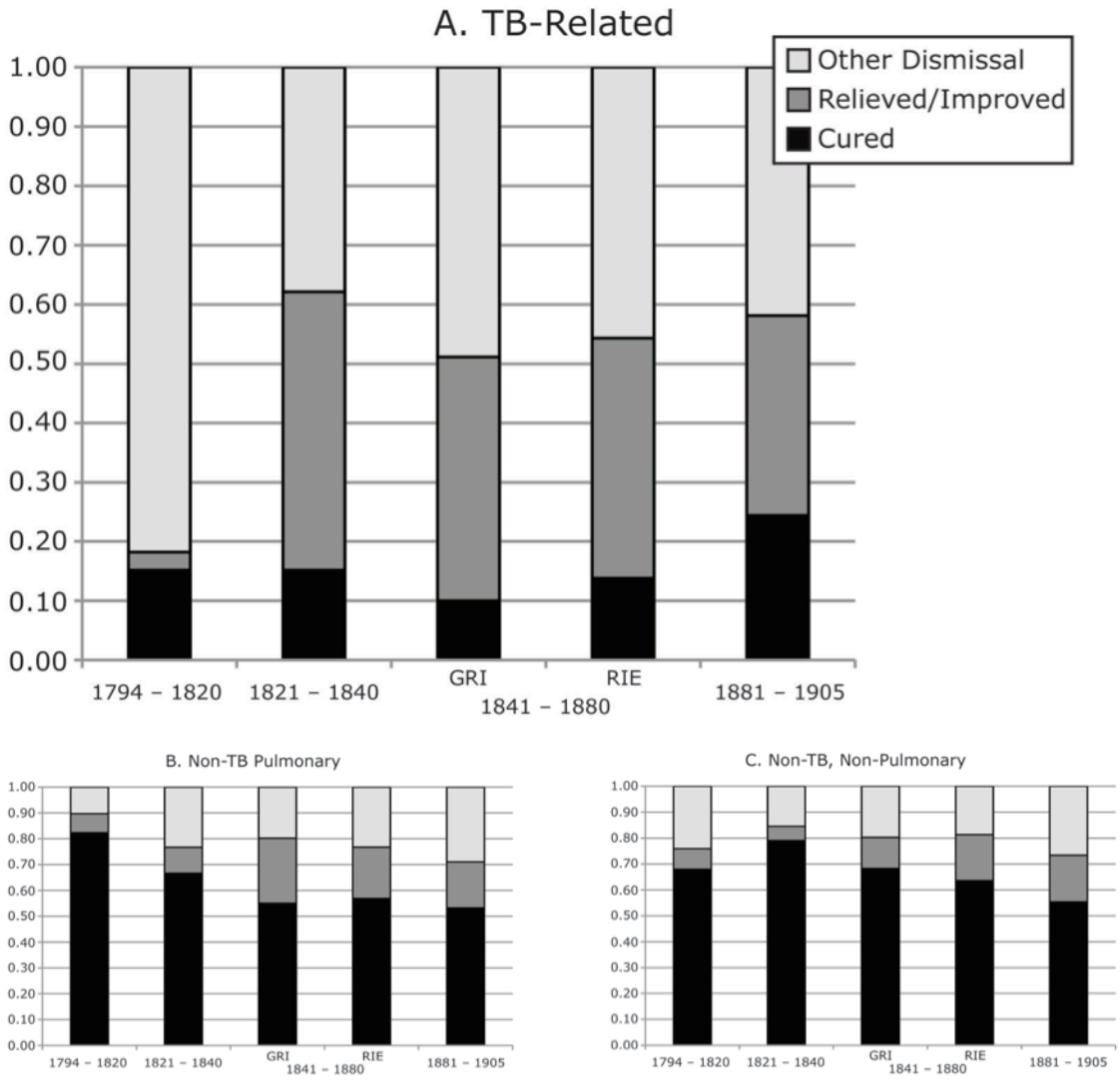
Table 7.12. Outcomes, 1881 – 1905

Outcomes	TB-Related	Non-TB Pulmonary	Other
Advice	0	0	2
By Desire	3	1	19
Cured	392	802	8589
Died	196	242	1465
Improper[a]	0	0	0
Irregular	25	13	188
In Statu Quo[b]	183	42	916
Relieved[c]	541	268	2778
Remitted	24	38	295
Other	238	99	1221
[Blank]	3	1	21
P-Values[d]		0.0005***	0.0005***

[a] This outcome was included by medical staff in 1885 only.
[b] Practitioners used this outcome in 1895 and 1905 only.
[c] The 1895 registers list patients as "improved" instead of "relieved."
[d] P-values were obtained through Monte Carlo simulation using 2000 replicates.
*** $p < 0.001$

This increase in tubercular patients considered by medical staff to be cured at dismissal corresponded to a decline in such patients classified as relieved. After 1820, practitioners classified a similar proportion of patients with TB-related disorders as having benefitted from hospital treatment: that is, the frequency with which tubercular patients were classified as cured, relieved, or improved varied between 51 and 62 percent for every time period from 1821 through 1905 (figure 7.2a) In the late-Victorian period, however, clinicians classified a larger proportion of patients with TB-related disorders who benefitted from hospital treatment as cured rather than relieved. A similar pattern did not obtain for cases of non-TB pulmonary disorders (figure 7.2b) or non-TB, non-pulmonary disorders (figure 7.2c), which practitioners were less likely over time to classify as cured.

Figure 7.2. Proportions of patients dismissed cured, relieved, or neither



Note: For sample sizes, see tables 3.1 (1794 – 1820), 4.1 (1821 – 1840), 6.1 (1841 – 1880), and 7.1 (1881 – 1905).

As a result, the greater proportion of patients with TB-related disorders classified by practitioners as cured at dismissal likely represents a reallocation of patients from "relieved" to "cured." Had the increase in cured TB-related cases been accompanied by an increase in those relieved, it would suggest that an improvement in treatment had caused more patients with tubercular disorders to benefit from hospital treatment. Instead, however, a similar

proportion of such patients benefitted from treatment in the late-Victorian period as in earlier periods. The relatively subtle changes in the treatments prescribed by practitioners across time in TB-related cases further contraindicate an improvement in treatment success. Rather, it is most likely that GRI clinicians in the period 1881 through 1905 began to see patients as cured whom they earlier would have considered only relieved.

Such reclassification followed from practitioners' recognition that tubercular disease processes could resolve. As I have discussed in chapters four and five, clinicians' observations during postmortem dissection of naturally healed tubercular lesions led to increased optimism from the mid-nineteenth century that TB and its analogues could be medically cured. By 1871, John Hughes Bennett was able to state, "there is nothing essentially destructive or necessarily fatal in Phthisis, and … in all stages of the disease it may be checked, and enable the individual affected to live many years subsequently, and die of old age or other disorders. Attention to morbid anatomy in recent times is demonstrating that this occurs far more frequently than was formerly supposed, and is due not only in many cases to the spontaneous efforts of nature, but in not a few to the direct interference of art." (543)

At the same time, clinicians were increasingly aware that many with TB-related disorders experienced periods of remission and disease. Latency of tubercular disease, particularly phthisis, gained support among British practitioners during the late nineteenth century. In response to an 1894 presentation by Edward Squire on heredity and TB-related disorders, for example, president of the Royal Medical and Chirurgical Society Jonathan Hutchinson commented, "The subject [of assessing family history of tubercular disease] was much complicated by the possibility of the latency of the tubercle bacillus, and there was now accumulating a good deal of positive evidence on this point." (Squire 1894, 1422) By

1900, GRI medical staff had incorporated the concept of active vs. inactive tubercular disease into practice. Of James McKenna, treated in that year for pleural effusion but with a history of night sweats and "spinal disease" (HH67/5/3, 229) — often the vertebral caries of skeletal tuberculosis — the clerk noted that in the month McKenna had been treated, "There has been no temperature indicative of any *active* tubercular condition." (231; emphasis added)

During the late-Victorian period, GRI clinicians considered it possible for TB-related disorders to become latent; at the same time, Alexander Napier anticipated that Koch's identification of the tubercle bacillus would inspire new, more effective treatments for tubercular disease (response to Carmichael 1882, 67). Neither of these causes for optimism would have substantially impacted the success with which GRI medical staff treated patients with TB and its analogues; however, together they may have increased clinicians' willingness to consider cured patients whose health and physical signs of disease had sufficiently resolved under hospital care. At the same time, the often-refractory nature of TB-related disorders may have inspired practitioners at the turn of the century to seek explanations for patients' failure to recover in the sufferer him- or herself rather than the quality of medical treatment, a point I shall develop below.

DEGENERATION, IMPROVEMENT, AND TUBERCULAR DISEASE

In the 1870s and 1880s, the middle-class zeal for working-class "improvement" that had characterized earlier decades of the Victorian era began to fade, replaced for some social commentators with a conviction of the working class's biological decline. At the close of the nineteenth century, Britain's global influence was fading, eroded by colonial unrest and approaching eclipse by the growing power of Germany and the United States; combined,

these weakened British optimism regarding their potential for continuous progress and global prominence (described, for example, by Stedman Jones 1971, c. 16). By the 1880s, the coincidence of this pessimism with widespread acceptance of evolutionary ideas (Bowler 2009, 178) led to fears of the British population's sociobiological decline. In *Degeneration: A Chapter in Darwinism*, for example, E. Ray Lankester (1880) capped his discourse on zoological degeneration with a warning to "ourselves, the white races of Europe:"

> the possibility of degeneration seems to be worth some consideration. In accordance with a tacit assumption of universal progress — an unreasoning optimism — we are accustomed to regard ourselves as necessarily progressing, as necessarily having arrived at a higher and more elaborated condition than that which our ancestors reached, and as destined to progress still further. On the other hand, it is well to remember that we are subject to the general laws of evolution, and are as likely to degenerate as to progress …. Possibly we are all drifting, tending to the condition of intellectual Barnacles …. It is possible for us … to reject the good gift of reason with which every child is born, and to degenerate into a contented life of material enjoyment accompanied by ignorance and superstition. (59–61)

Typically of popular perceptions of British decline at the time, the human degeneration Lankester (1880) feared was moral and intellectual as well as physical. Heavily influenced by the Lamarckian concept of the inheritance of acquired characteristics (Bowler 2009, 236–40), British thinking emphasized the role of behavior in populational degeneration. From the point of view of the middle classes, the consequences of vice — sexual or alcoholic intemperance, non-industriousness, insufficient piety — would be propagated and compounded generation after generation (Boddice 2011, 331).

For the late Victorians, social circumstances bore considerable influence upon individuals' development, creating a recursive relationship among environment, behavior, and heredity, and singling out the working classes as most at risk for degeneration. As I discussed in chapter six, J. Milner Fothergill described such a cycle in his 1874 lay guide *The Maintenance of Health*: industrial labor led workers to seek mental stimulation in drink (65), the

cost of which required their children to labor (48). The child labor necessitated by adult intemperance in turn caused physical degeneration. "Sustained toil in an impure atmosphere at an early age, when rapid evolution of the body should go on," Fothergill warned, "exercises a most deleterious influence over the processes of growth, as the stunted and ungainly figures of the manufacturing districts amply demonstrate." (ibid.) The immorality bred by industrial labor resulted in physical degeneration.

Such ideas were persistent. Jack London, in 1903's *The People of the Abyss*, traced degeneration similar to that described by Fothergill to the deprivation, immorality, and crowding of the slum, describing the transformation of bright, beautiful children into "stunted forms, ugly faces, and blunt and stolid minds." (274–75) Referencing evolutionary discourse, he emphasized his point by likening the people of London's East End to atavistic forms, apes and cavemen (284–86). The heritability of acquired characteristics would allow the degenerative processes described by Fothergill (1874) and London (1903) to continue, practically precluding good character (Boddice 2011, 331) and good health among the urban poor and rendering intervention difficult, if not impossible.

Earlier, social reformers had sought to reverse the damage by encouraging the working classes in morality and temperance (Luckin 2006, 247), but by the turn of the century, as I have indicated before, cracks were beginning to show in their veneer of public-mindedness. The self-interest of social reformers did not go unnoticed. Oscar Wilde, in his 1887 short story "Lord Arthur Savile's Crime," lampooned reformers' divergent moral standards for themselves and those they purported to aid: "Thank you so much for the flannel for the Dorcas Society, and also for the gingham," wrote the aristocratic Jane Percy to her aunt, referring to the ladies' charitable association providing clothing to the poor (Wilde 1891, 60). "I quite agree with you that it is nonsense their wanting to wear pretty

things, but everybody is so Radical and irreligious nowadays, that it is difficult to make them see that they should not try and dress like the upper classes. … How true, dear aunt, your idea is, that in their rank of life they should wear what is unbecoming." (60–62)

In actuality as well as fiction, middle-class commentators were often unsympathetic to those they aimed to reform. Thus, Andrew Mearns, in his 1883 "plain recital of plain facts" (2) regarding poverty in London, lamented "the misery inherited [by poor children] from the vice of drunken and dissolute parents," (13) but went on to uncharitably describe these children as "stunted, misshapen and often loathsome objects." (ibid.) Two decades later, such degenerate children were not only abhorrent, but also burdensome. The anonymous author of the *Lancet*'s "Public-Houses and the Spread of Tuberculosis" (1904) reported having seen, through the open door of a pub, "slatternly" female patrons whose toddlers played in the sawdust covering the floor, onto which patrons expectorated posing a risk for TB infection. "That this is not an uncommon occurrence," he wrote:

> those who have occasion to visit poor and squalid neighbourhoods well know and in the light of such facts is it to be wondered that the race is said to be degenerating or that medical science should have such a hard up-hill fight with disease? Granting that these children, probably the offspring of degenerate beings, become infected with the bacillus of tubercle the environment in which they live will necessarily be a potent factor in the development of pulmonary tuberculosis and not only will they themselves suffer but they will also involve the public in great expense for their subsequent treatment and keep. (160)

Such lack of empathy demonstrates that rather than emphasizing the benefits of improvement and philanthropy for the working classes as well, social reform efforts now encouraged middle-class participation by casting the condition of the working classes as a threat. The pub-door peeper above lamented the public expense entailed by the degenerate children of slatternly mothers. The result of Mearns's "sober inquiry" into the living conditions of the poor was a distinctly immoderate warning to his middle-class readers:

"THIS TERRIBLE FLOOD OF SIN AND MISERY IS GAINING UPON US." (1883, 2; emphasis retained)

Amid such fears that the degeneration of the British poor could swamp the nation, tubercular disease — still bearing a strong stamp of essentialist etiology such as hereditary taint — became a touchstone. At the 35th annual meeting of the British Medical Association in 1867, J. Henry Bennet of London prefaced his remarks on the treatment of pulmonary consumption with a lengthy excursus on the disease as evolutionary selective pressure, which indeed leaves the reader wondering that he endorsed treating the disease at all:

> Looking at the subject in a philosophical point of view, tuberculisation was not a scourge or pestilence, but one of the means by which Providence purifies the human race, weeding it of effete, worn-out organisations, unfit to perpetuate it in conditions of health and vigour. Were not such laws in operation, the human race would, in a few generations, become one of dwarfs, pigmies, of misshapen diseased abortions. The attention of thinkers has been much directed to the struggle for life which pertains in wild nature. Effete, worn-out organisations are not allowed to exist. … But man has intellect; can provide for his own old age, and for the sickly existence of his sickly progeny. He has passions, desires shared by the weak as well as the strong, and thus society is full of effete organisations, which propagate the race as well as their stronger brethren. But here the laws of Providence come in to correct our errors. The strong in life, in youth and vitality, give the same principle to their progeny; but the weak, the sickly, the old, cannot give what they have not got. Their children are born with the seeds of disease and death in them, and die of tubercular meningitis, scrofula, and consumption. Thus is the integrity, the sanctity, of the human race preserved. (137)

A similar role for tubercular disease, if in a different tone, was espoused in an anonymous *Lancet* article, "Decline of Phthisis Mortality in England," (1889) addressing said decline reported by the Registrar-General. "The marked and continuous decline in the mortality from phthisis in England and Wales during the past fifty years of civil registration is one of the most hopeful signs of the improving health condition of our population," commented the author, "and affords the strongest evidence against the alleged physical deterioration of the English people." (954) In the latter half of the nineteenth century, then,

tubercular disease and its outcomes were a bellwether for the degeneration or strength of British peoples.

"CONGENIAL SOIL"

The relationship between TB-related disorders and degeneration frequently was expressed in medical literature by means of a "seed and soil" metaphor for the role of Koch's bacteria in tubercular etiology. In this conception, tubercle bacilli could produce disease only when they found a suitable substrate for growth in the tissues of individuals made susceptible by such influences as "heredity, bad food, bad ventilation, overwork of mind or body, unhealthy occupations, [and] damp soil." (Williams 1883, 313) In the decades following Koch's discovery of the tubercle bacillus the metaphor proved popular in the United States (e.g., "Is Consumption Curable?" 1887, 233; N. W. 1886, 87; Thompson 1905, 462) as well as the UK (e.g., Beevor 1899, 1012; Williams 1883, 313; Williams 1882, 618). Critically, the conception of tubercle bacilli as seed requiring suitable soil explained the gap between the number of individuals surely exposed to the bacilli and those developing disease, and aligned the new contagious etiology with older, essentialist constructs of tubercular disorders. By the turn of the century, the Edinburgh-trained Sir Dyce Duckworth of St. Bartholomew's Hospital was able to state the prevalent view, "We may, I believe, regard it as certain that for the production of tuberculosis two factors are necessarily concerned: (*a*) the parasite, and (*b*) the nature and condition of health, or diathetic habit, of the host." (1901, 1253)

The formalization of this interdependence between bacillus and diathesis in tubercular etiology reinforced notions of the poor as inherently and uniquely predisposed to TB-related disorders, a construction common throughout the Western world. Indeed, early-

twentieth-century practitioners in both the UK and United States recited a common refrain: "There are two kinds of consumption — that of the rich and that of the poor. The former is sometimes cured, the latter never." (Burton-Fanning 1909, 4; Latham 1903, 3; Latham 1907, 81; Pratt 1906, 210) This long-recognized vulnerability of the poor to tubercular disease was not blameless, however. "[T]he bacillus requires in every instance a congenial soil to enable it to multiply and to carry on its work," suggested C. Theodore Williams of the Brompton Hospital for Consumption (1883, 313), his choice of words revealing a belief in the patient's contribution to disease. Those developing tubercular disorders — e.g., children raised on pub floors ("Public-houses," 1904) and J. Henry Bennet's (1867) effete, worn-out individuals — went beyond susceptibility, demonstrating a welcoming affinity for the seeds of disease.

The late-Victorian concept of those developing tubercular disease as congenial soil for the bacilli drew upon earlier constructions of these disorders as hereditary. Although tubercle bacilli as the proximate cause of disease was new, comments such as Edward Squire's (1894) that an inherited "tendency to suffer from disease … which the offspring of phthisical parents had in common with the children of weakly parents" (1422) favored disease development was certainly not. Scottish practitioners' concern with patients' family histories was evident in my sample beginning in the mid-Victorian period. As I have discussed, however, late-Victorian GRI practitioners recorded with increased frequency the presence or absence of TB-related disorders in tubercular patients' families (table 7.5), indicating a heightened interest coincident with fears of degeneration and a need to reconcile new concepts of tubercular contagion with older understandings of these diseases.

By implicating the social environment in moral and physical decline, degeneration brought with it an expansion of older constructs of hereditary risk for tubercular disease. This was apparent as early as 1871, when John Hughes Bennett downplayed the importance

of a specifically tubercular diathesis. "Instances are not uncommon in which members of the same family are observed to become affected one after another with Phthisis, on arriving at a certain age. This, however, may depend not so much upon weakness inherited from parents, as it does upon a vicious [i.e., immoral] method of rearing the infants and children of certain families," Bennett explained. "Although … there can be no doubt that weakness in parents is a cause of weakness in the offspring, we are of opinion it is by no means so general or influential a source of Phthisis as is usually supposed." (546) As I quoted above, Squire, too, cited the contribution of "weakly parents" as well as "phthisical parents" to tubercular disease in the offspring (1894, 1422). Indeed, Squire went so far as to state unequivocally, "The family house was certainly of more importance [in TB risk] than the family tree — the surroundings were more dangerous than the pedigree." (1423)

Arguments such as these from Bennett (1871) and Squire (1894) did not entirely reject hereditary risk, however. Just as working-class living conditions were said to induce degeneration, the environments described by Bennett and Squire induced constitutional weakness, a form thereof. As we have seen, such characteristics were widely held to be heritable at the turn of the century; as a result, the change evident in such discourse was a transition from an inherited risk specific for tubercular disease to a heritable constitutional weakness rendering its bearer more congenial to disease.

Late-Victorian GRI clinicians revealed in practice their affinity for the new social and medical emphasis on environment, behavior and heredity. I have discussed the increased frequency with which medical staff recorded patients' family histories; for the first time in this period, clinicians also recorded details about patients' living situations. Some such details referenced earlier-established environmental risks for disease — dampness and whether the windows were kept open or closed, for example. Other information — the number of

rooms, how many lived there, whether they had pulmonary symptoms — related to late-nineteenth-century concern surrounding crowding (and its influence on moral and physical degeneration) and contagion. This signaled an expansion of medical interest in patients' circumstances beyond diet and nutrition in response to social fears of the widespread and long-term repercussions of those circumstances.

In practice, medical staff also demonstrated increased interest in patients' behavior, recording instances of immoral behavior with the greatest frequency of any period in my sample (table 7.13). For the first time, GRI practitioners noted not only how long a patient had been ill, but how long he or she had been away from work because of illness (appendix G), suggesting a new impetus to verify patients' status as "worthy poor." Other reasons for not working were noted, with clear disapprobation when such did not meet practitioners' behavioral expectations: "in the last three years has not done much, as his wife had a business and made sufficient for both," (HH67/12/20, 353) noted the clerk of Francis M., treated for phthisis pulmonalis and alcoholism in 1905. At the same time, medical staff continued to record patient complicity in illness. "Although he knew that his stomach was weak, he never restricted his diet," (ibid.) the clerk entered, again in Francis M.'s case.

Women continued in the late-Victorian period to endure special behavioral scrutiny by clinicians owing to their perceived responsibility for their own health and their families' moral and physical well-being. John T., treated for phthisis in 1893, suggested his disease resulted from exposure to drafts at his office (HH67/7/5, 200). The clerk disagreed, noting, "Patient is married but owing to the intemperance of his wife has been separated from her …. He has been much neglected in consequence, careless housekeeper, badly cooked food, et cetera and this may have to do with his present condition." (ibid.) Perhaps not coincidentally, it was toward a female patient that medical staff revealed a newly callous

attitude, indicating greater emotional removal from the practitioner-patient relationship: thus, when Jane J. was admitted owing to an abdominal stab wound, the clerk jocularly noted her occupation as "horizontal gymnastics." (HH67/22/2, 190) In all, the impression is overwhelmingly of practitioners concerned not only with patients' physical condition, but also with the disease in the social body heralded by their congenial susceptibility to TB and its analogues.

Table 7.13. Histories of immoral behavior

	Diagnosis Groups			
Time Period	TB-Related	Non-TB Pulmonary	Other	TOTAL
1821 – 1840, GRI				
Patients	38	25	25	88
Immorality	2	0	6	8
Proportion Immorality	0.05	0.00	0.24	0.09
1841 – 1880				
GRI				
Patients	55	24	31	110
Immorality	0	2	0	2
Proportion Immorality	0.00	0.08	0.00	0.02
RIE				
Patients	210	95	112	417
Immorality	36	13	13	62
Proportion Immorality	0.17	0.14	0.12	0.15
1881 – 1905, GRI				
Patients	124	64	70	268
Immorality	28	18	17	63
Proportion Immorality	0.23	0.28	0.24	0.24

Note: Medical staff did not record any such behavior in my sample prior to 1821.

CONCLUSIONS

During the late-Victorian period, practitioners reconstructed earlier conceptions of tubercular disease as hereditary, environmental, or nutritional to accommodate the contagious etiology suggested by the findings of Villemin and Koch. Coinciding as these changes did with social concern regarding the degeneration of the British people, clinicians constructed the working classes as pathologically resistant to improvement — congenial soil in which tubercle bacilli would flourish. The advent of Koch's bacilli at GRI did not revolutionize diagnosis, much as it failed to revolutionize disease constructs. Rather, practitioners used bacteriology most often to confirm a diagnosis already established by medical staff using more familiar physical diagnosis techniques. As a result, clinical concepts of tubercular disease remained rooted in pathological anatomy. This placed the tubercle bacillus as incidental to TB-related disorders in practice, echoing the *Lancet*'s early characterization of the tubercle bacillus as a proximate rather than ultimate cause of such disease. Instead, medical staff placed an increased emphasis on patient social conditions and behavior in a manner consistent with broader social concerns regarding moral and physical degeneration, revealing a connection among women and the working classes, tubercular disease and the metaphorical social disease perceived to be weakening the UK. As I will discuss in the concluding chapter, the response to such a nexus was perhaps foreshadowed by none other than Edinburgh's William Cullen, who ca. 1770 wrote, "such is the nature of the animal oeconomy that our fluids are constantly Degenerating, and if they are not thrown out of the Body they would soon be noxious to it." (unpublished MS quoted in Stott 1987, 132)

CHAPTER 8

CONCLUSIONS

Medical responses to TB and related disorders in nineteenth-century Scotland reflected a construct of disease increasingly defined by biological attributes, yet underlain by a social structure assigning the causes of disease disproportionately to the working classes and, to a lesser extent, women. Thus, medical professionals reinforced rather than replaced fears of social pathology with biological disease. The continued dependence of medical practitioners and medical scientists on explanations for tubercular disease based in social distinctions served to reify the latter, naturalizing the greater burden of disease in marginalized groups. The accompanying weight granted to heterodox behavior in explanations for tubercular disease rendered practitioner-patient interactions retrospective rather than prospective, emphasizing etiology instead of treatment — blame instead of cure.

Beginning with the period 1794 through 1820, the diagnosis and treatment of TB and analogous disorders were transitional between the essentialist constructs of the eighteenth century and those rooted in the pathological anatomy of the mid- to late-nineteenth century. Tubercular diseases at the turn of the nineteenth century at GRI were set apart from other diagnoses by a number of clinical indicator categories, indicating a concept of these diseases as specific clinical entities, agreeing with the specificity of their description in eighteenth-century medical treatises. However, there was no statistical distinction between treatments prescribed by medical staff in cases of tubercular and other disorders; this unity of treatments supported contemporaneous explanations of disease as an expression of constitutional disorder, and thus some unity in constructs of disease across diagnosis categories despite diagnostic specificity. At the same time, physicians' external and discursive

mode of patient examination grounded medical practice in the practitioner's unique ability to mine the patient's history for pertinent clues to his or her present condition.

During the years 1821 through 1840, practitioners began to apply this perspicacity internally as well as externally. The introduction of percussion and auscultation into GRI practice created a role for concealed pathology in diagnosis, rooting constructs of TB and its analogues in the pathological anatomy observed in newly accessible corpses for dissection both in the UK and on the continent. Treatment remained non-specific, illustrating the persistence of constitutional explanations for disease across disorders despite the increasingly specific diagnosis made possible by practitioners' physical examination of patients. Practitioners' interactions with patients changed beyond their hands-on mode of examination, becoming marked by clinicians' decreased willingness to record patients' accounts unchallenged. For the first time in my sample, as well, medical staff recorded patients' histories of immoral behavior during examination, signaling the inclusion of heterodoxy among causes of tubercular disorders as well as sexually transmitted infections.

RIE physician John Hughes Bennett was emblematic of the period 1841 – 1880. Bennett advanced cod liver oil as a near-specific treatment for tubercular disease, and his practice and writings highlighted the importance of physical examination and pathological anatomy to medical practice as well as the distinctions practitioners drew between private and charitable medical patients. Bennett's 1841 popularization of cod liver oil introduced a treatment intended to correct the patient's faulty assimilation of nutrition in tubercular disorders, assigning a specific origin to these diseases. Through this etiology, however, practitioners continued to attribute TB and analogous conditions to constitutional factors, retaining an essentialist construct of these diseases despite the increasing anatomical specificity with which they were diagnosed. Bennett was a vociferous champion of both

physical examination and postmortem dissection, encouraging his students to master auscultation and conduct dissections of both private and charitable patients whenever possible. His published discussions of private and charitable patients distinguished the former with anonymity, however, revealing his lesser consideration for the privacy of the latter, parallel to the access to the bodies of the poor granted by the Anatomy Act.

A comparison of practitioners' records at GRI and RIE for the years 1841 through 1880 reveals an increased emphasis by medical staff on patient behavior and family history. Particularly at RIE, instances of immorality figured frequently in practitioners' notes of patient history, indicating a growing role for such behavior in clinicians' explanations for disease across diagnosis categories. Medical staff further displayed for the first time an interest in hereditary contributions to disease, particularly tubercular disorders, recording patients' family histories of such ailments alongside the patient's own history. Through such actions, clinicians revealed a continued focus on constitutional causes for tubercular disease — immorality, heredity, nutrition — alongside their by now habitual use of percussion and auscultation to identify the presence of tubercles and their sequelae.

Practitioners' diagnosis and treatment of tubercular disorders at GRI between 1881 and 1905 demonstrate growing evidence for TB contagion from the 1860s, culminating during this period in Robert Koch's 1882 discovery of the tubercle bacillus. However, support among practitioners for a contagious etiology in the UK wavered through the end of the nineteenth century. In the intervening years, medical staff at GRI increasingly employed bacteriological techniques in cases of TB-related disorders, perfunctorily confirming the absence of tubercle bacilli in non-TB pulmonary cases while repeatedly seeking the bacteria in the sputum of tubercular patients. Bacteriology was thus used to confirm practitioners' diagnoses achieved through examinations emphasizing patient history and pathological

anatomy. The long-held importance of constitutional etiologies for tubercular disease combined during this period with a new medical and social emphasis on the physical and moral degeneration of the British population through inherited weakness caused by immorality and unhealthy environments. This conflation led to the construction of TB sufferers as "congenial soil" for bacilli, providing through their own moral and physical shortcomings an environment welcoming to the seeds of disease.

Through the course of the nineteenth century, then, medical practitioners constructed tubercular disease as a circumscribed biological entity. Beginning with externally visible characteristics, such as those of the pulse, and growing to include physical examination to identify internal pathology from external signs, clinicians' concepts of TB and related disorders at no time lacked specificity. From the earliest sampled cases at the turn of the nineteenth century, a suite of clinical indicators distinguished tubercular disorders from other classes of disease. Rather, from the acceptance of auscultation and percussion by GRI and RIE practitioners, diagnosis during the nineteenth century relied on practitioners' assessment of pathological anatomy in addition to signs and symptoms gathered through traditional external examination. Over the course of the long nineteenth century, symptom categories tended to accrue that significantly differentiated TB and its analogues, indicating practitioners continually refined the clinical constructs of these diseases over time and in response to new techniques: bacteriology, urinalysis, thermometry, and reflex testing in addition to auscultation and percussion.

Clinical constructs of tubercular disease gained this biological specificity and emphasis on pathological anatomy without shedding older concepts of these diseases as essential to the sufferer, however. Throughout the period from 1794 through 1905, practitioners continued to record indicators of patients' constitutional, hereditary, and

behavioral risks of disease across disorders; indeed, clinicians recorded instances of the latter two categories more frequently in the mid- to late-nineteenth century than earlier, when essentialist concepts of disease were supposed to have dominated. The introduction of physical examination and new technologies and techniques during the nineteenth century elaborated upon rather than replaced earlier practitioners' constructs of tubercular disease through the addition of pathological anatomy. Even with the discoveries by Villemin, Koch, and others in the lattermost third of the century, Scottish clinicians demonstrated less a germ revolution than a germ adjustment in their thinking about TB and related disorders.

It is clear that, in Scottish practice, established clinical structures in which tubercular diseases originated with constitutional disorder retained their explanatory power through the many nineteenth-century advancements in medicine. British practitioners' hesitancy to credit Koch's bacillus with the ultimate causation of TB and its analogues resulted in part from the obscure relationship they observed between exposure to tubercular individuals and development of disease: heterogenity of risk for developing active disease meant that some exposed individuals never exhibited TB-related disease, while bacterial latency could interpose years between exposure and disease. British clinicians were aware of tubercular latency — in pathological if not bacteriological terms — from at least the latter half of the nineteenth century, however, and continued to debate the role of the tubercle bacillus in tubercular disease. The complex course followed from exposure to disease does not adequately explain practitioners' continued resort to constitutional explanations for these disorders; we must look elsewhere.

The nineteenth century represented an inflection point in practitioner-patient relationships in Western medicine, in addition to class and gender relations in the UK and beyond. Foucault (1963) identified the power/knowledge structures inherent in the suite of

medical developments that characterized the nineteenth century. Physical examination, anatomical and bacteriological constructs of disease, and hospital treatment combined to situate the practitioner as uniquely capable of accessing the "truth" of the patient's condition, and thus uniquely positioned to dictate what the patient must undertake to get well.

This new authority of middle-class male medical practitioners was characteristic of the broader changes that underscored the social distance between middle-class men and women, the working classes, and — although I have not emphasized it here — the aristocracy. The nineteenth century was marked by the ongoing self-definition of the middle classes in contrast to those above and below them, both professionally and behaviorally, through piety, self-restraint, and distinct social roles for men and women — the latter characterized by withdrawal to the home (e.g., Davidoff and Hall 1987). The crystallization of class and gender roles extended to the naturalization of perceived differences between middle-class men and others. Through construction of themselves as naturally superior to women, children, "savages" — including the working classes — and animals, middle-class men valorized their own social role while casting all others as unstable elements to be subordinated (cf. Boddice 2011, 325).

The powers of self-restraint lauded by the Victorian middle classes were diametrically opposed to the cultural and medical constructs of tubercular disorders as diseases of ill-restraint and excess. As the disease of the romantic poets, TB had long borne cultural weight as a disease of passion and unrestrained emotion. Dubos and Dubos (1952), Sontag (1978), Lawlor (2006), Byrne (2011), and many others have described TB's use as a signifier of such traits in literature and literary biography. An example of the latter is the life of John Keats, whose consumption is commonly said to have originated with the emotional upset

occasioned by a negative review of his work, a story originally circulated by his friend Percy Bysshe Shelley (Lawlor 2006, 137). This construct of tubercular disease was not only cultural, however.

Ill-restrained behavior was included among the causes of TB and its analogues in published literature from at least as early as the eighteenth century, but gained new significance in practice during the Victorian era. GRI practitioners did not record immorality in my sample of patient records prior to 1821, and only rarely prior to 1841. Despite the long cultural and medical history of associations between ill-controlled behavior and emotions and tubercular disease, in Scottish practice these were absent prior to the Victorian era, with the exception of a few instances dating to between 1821 and 1837. In medical practice, then, heterodox behavior was newly emphasized as a cause of disease by clinicians beginning in the mid-nineteenth century.

Despite expectations grounded in the increasingly scientific posture of medical practitioners, then, patients' immoral behavior as a cause of disease became more rather than less important to the clinical construct of tubercular disorders over the course of the nineteenth century. Corresponding to Victorian social models of the working classes and women as having little capacity for self-restraint, this equated the social pathology feared to result from poorly controlling such unstable elements with the physical pathology of disease. TB and its analogues in the working classes thus became the expression of social ills, a visible reminder of these groups' failure to embrace orthodox behavior. As a result, their succumbing to tubercular disease could be seen as the natural consequence of disobeying the "natural" order — adherence to middle-class values and subordination to middle-class men's reason and fitness to lead.

Overall in my research, while I noted a number of class-based constructs of TB-related disorders expressed by medical staff, I identified less evidence of gender influence. Notably absent, for example, are extensive examples of pathological femininity such as that recognized by Barnes (1995) for France and Byrne (2011) for the UK. This likely results from the social conflation of both the working classes and women as other with respect to middle-class men. In addition, as I discussed with respect to the work of Cullen in chapter three (unpublished MSS ca. 1770: cited in Stott 1987, 137–38), there is textual support for a medical construct of gender in which men and women of the working classes were perceived as more similar to each other than were men and women of the middle classes. Either or both of these would have reduced the effect of gender in my sample.

I have discussed the heterodox behavior of the working classes as an increasing focus for medical staff at GRI and RIE during the Victorian era, indicating that tubercular disease came to be seen as the natural consequence of immorality, a violation of the perceived natural order. Here, then, as in Douglas (1966), disease is the consequence of violating a naturalized boundary. Particularly at the close of the nineteenth century, the increased risk for tubercular disease among the working classes would seem to have been constructed by medical practitioners as the natural consequence of their otherness. At the turn of the century, degeneration theory rendered the distinction between the working classes and middle classes biological as well as social; the behavior and environment of the urban working classes contributed to their heritable degeneration and weakness, which in turn led to their decreased resistance to tubercular disease. In the parlance of the time, however, they did not become a susceptible or vulnerable population, but rather congenial soil, the willing source of disease in the social body.

Indeed, to the present day, clinicians have predicated TB diagnosis and treatment on pathological anatomy, an approach that in effect retains the nineteenth-century construct limiting disease to congenial others. Diagnosis of TB presently requires evidence of pathological anatomy or clinical indicators of active disease in addition to the presence of *M. tuberculosis* in a clinical specimen (e.g., Centers for Disease Control 2011, appendix A). These requirements limit the disease to those who have failed to maintain latency, while the United States Centers for Disease Control currently recommends against treating those carrying the "seed" (ibid., appendix C) — likely most of those exhibiting a positive tuberculin skin test (National Health and Nutrition Examination Survey 2011, s. 2.1).

Such restrictions of TB to cases exhibiting active pathology rather than latent bacterial infection by definition locates the disease within the marginalized: failure to maintain latency is itself othering, indicative of pathological immunosuppression in the present-day construct, while "normal" individuals contain the bacteria. Individuals with active disease remain degenerate, as they were at the close of the nineteenth century, having lost the normal ability to limit *M. tuberculosis* infection. Then, individuals with tubercular disease were the site of pathology, both biological and social, threatening the naturalized social order — by analogy to William Cullen, constantly degenerating and necessitating exclusion before they destroyed the social body. Today, little has changed; risk factors for TB, including HIV/AIDS, substance abuse, poverty, and even malnutrition, continue to bear connotations of immorality and blame.

When we forget that the construct of tubercular disease as a biological entity — as a natural phenomenon — was grounded in nineteenth-century social structures and social fears regarding the ungovernability of women and particularly the working classes, we ignore the ways in which this prejudices diagnosis and treatment today. Eliding these social

foundations made TB's distribution within and among human groups appear natural, the result of unhealthy behavior among unhealthy people, excusing inaction; what is natural is difficult to alter. But TB is a metaphor, we are reminded today, again and again (Sontag 1978); TB is a social disease (Dubos and Dubos 1952). Only by facing it as such can we strip the disease of its historical weight and understand how best to effect change.

WORKS CITED

UNPUBLISHED PRIMARY SOURCES

LOTHIAN HEALTH SERVICE ARCHIVE

Royal Infirmary of Edinburgh

Series LHB1/1, Minutes of the Managers' Meetings

Series LHB1/6, Records of the Managers' Visits

Series LHB1/126, Admission and Discharge Registers

Series LHB1/129, Ward Journals

NATIONAL HEALTH SERVICE GREATER GLASGOW AND CLYDE BOARD ARCHIVE

Glasgow Royal Infirmary

Series HB14/1, Minutes of the Managers' Meetings

Series HB14/2, Annual Reports

Series HB14/5, Ward Journals

Series HB14/6, Correspondence

Series HH67/1 through -/32, Ward Journals

Series HH67/56, Admission and Discharge Registers

PRIVATE COLLECTION

Bennett, J. Hughes to Dr. Reynolds, 23 December 1870, private collection.

PUBLISHED PRIMARY SOURCES

Anonymous. 1822. "1. *De l'auscultation médiate; ou traité du diagnostic des maladies des poumons et du coeur, fondé principalement sur ce nouveau moyen d'exploration*. Par R. T. H. Laennec, D.M.P. 2. *A Treatise on the Diseases of the Chest, in Which They are Described According to Their Anatomical Characters, and Their Diagnosis Established on a New Principle, by Means of Acoustic Instruments*. With Plates. Translated from the French of R. T. H. Laennec, M.D. with a preface and notes, by John Forbes, M.D." *Edinburgh Medical and Surgical Journal* 18: 447–74.

———. 1825. "Review From the West: *An Introduction to the Use of the Stethoscope, With its Application to the Diagnosis in Diseases of the Thoracic Viscera, Including the Pathology of These Various Affections*. By William Stokes, M.D. MacLachlan & Co., Edinburgh; Baldwin & Co., London; and Hodges & Co., Dublin. 8vo. 1825. Pp. 224." *Lancet* 5: 471–75.

———. 1855. "Brompton Hospital. Physical Signs of Phthisis. Analysis of Eighty Cases." *Lancet* 66: 387–89.

———. 1857. "Where to Draw the Line." *Lancet* 70: 585–86.

———. 1867. "Cause and Nature of Tuberculosis, and its Transmission by Inoculation. By Dr Villemin, Professeur Agrégé Au Val-de-grace." *Edinburgh Medical Journal* 12: 756–65.

———. 1867. "What is Tubercle?" *Medical Times and Gazette* 1: 414–15.

———. 1875. "Obituary: John Hughes Bennett, M.D., F.R.S.E." *British Medical Journal* 2: 473–78.

———. 1882. "*Davos Platz, and the Effects of High Altitude on Phthisis*. By Alfred Wise, M.D. London: J. & A. Churchill: 1881." *Edinburgh Medical Journal* 27: 640.

———. 1882. "A New Disease." *Edinburgh Medical Journal* 28: 573.

———. 1882. "A New Disease." *Medical Record* 22: 532.

———. 1882. "New Parasites." *Edinburgh Medical Journal* 28: 285–86.

———. 1882. "New Parasites." *Medical News* 41: 27.

———. 1882. "Recent Researches on Bacteria: I. Koch's Methods of Investigating Pathogenic Bacteria." *British Medical Journal* 1: 624–25.

———. 1882. "Recent Researches on Bacteria: II. Koch's Investigations on Tuberculosis." *British Medical Journal* 1: 706–07.

———. 1882. "Saturday, April 22, 1882." *Lancet* 119: 654–57.

———. 1882. "*Scrofula and its Gland Diseases*. By Frederick Treves. London: Smith, Elder, & Co.: 1881." *Edinburgh Medical Journal* 28: 324–25.

———. 1885. "Treatment of Nephritis." *Maryland Medical Journal* 12: 247.

———. 1887. "Is Consumption Curable?" *Science* 10: 38–41.

———. 1887. "McKenzie's Eye-lotion." *Chemist and Druggist* 31: 559.

———. 1889. "Decline of Phthisis Mortality in England." *Lancet* 133: 954.

———. 1893. "Tuberculosis and its Prevention." *Lancet* 142: 1019.

———. 1904. "Public-houses and the Spread of Tuberculosis." *Lancet* 164: 160.

Allchin, William Henry. 1887. *Scheme for Case Reporting*. London: Adlard and Son.

Alcott, William A. 1857. *The Laws of Health*. Boston: John P. Jewett and Company.

Allbutt, Clifford. 1882. "Commentary on 'The Contagion of Phthisis'." *British Medical Journal* 2: 620–21.

Anderson, T. McCall. 1902. "Tuberculin – Discussion." In *Transactions of the British Congress on Tuberculosis for the Prevention of Consumption*. Vol. 3, 100–104. London: William Clowes and Sons, Limited.

Auenbrugger, Leopold. 1761. *Inventum novum ex percussione thoracis humani ut signo abstrusos interni pectoris morbos detegendi*. Vienna: Joannis Thomae Trattner.

Auenbrugger, Leopold, Jean-Nicolas Corvisart, and John Forbes. 1824. "On Percussion of the Chest: Being a Translation of Auenbrugger's Original Treatise, Entitled 'Inventum novum ex percussione thoracis humani, ut signo, abstrusos interni pectoris morbos detegendi;' and of a Selection of the More Important Commentaries of Corvisart on That Work." In *Original Cases With Dissections and Observations Illustrating the use of the Stethoscope and Percussion in the Diagnosis of Diseases of the Chest; Also Commentaries on the Same Subjects Selected and Translated From Auenbrugger, Corvisart, Laennec and Others*, edited and translated by John Forbes, 1–64. London: T. and G. Underwood.

Bardswell, Noel Dean, and John Ellis Chapman. 1908. *Diets in Tuberculosis: Principles and Economics*. London: Oxford University Press.

Bartholow, Roberts. 1879. *A Practical Treatise on Materia Medica and Therapeutics*. New York: D. Appleton and Company.

Beale, Lionel S. 1864. *Urine, Urinary Deposits, and Calculi; and on the Treatment of Urinary Diseases*. London: John Churchill & Sons.

Beddoes, Thomas. 1795a. "Observations on the Character and Writings of John Brown, MD." In *The Elements of Medicine*, edited by John Brown, xxxv–cii. London: J. Johnson.

Beddoes, Thomas. 1795b. "Of the Brunonian Doctrine." In *The Elements of Medicine*, edited by John Brown, cxxvi–clxviii. London: J. Johnson.

Beevor, Hugh. 1899. "An Oration on the Declension of Phthisis (Pulmonary Tuberculosis)." *Lancet* 153: 1005–20.

Bennet, J. Henry. 1867. "The Treatment of Pulmonary Consumption." *British Medical Journal* 2: 137.

Bennett, John Hughes. 1841. *Treatise on the Oleum Jecoris Aselli, or Cod Liver Oil, as a Therapeutic Agent in Certain Forms of Gout, Rheumatism, and Scrofula: With Cases*. London: S Highley.

Bennett, John Hughes. 1850. *Lectures on Clinical Medicine*. Edinburgh: Sutherland and Knox.

Bennett, John Hughes. 1851. *The Mesmeric Mania of 1851, With a Physiological Explanation of the Phenomena Produced: A Lecture*. Edinburgh: Sutherland and Knox.

Bennett, John Hughes. 1853. *The Pathology and Treatment of Pulmonary Tuberculosis; and on the Local Medication of Pharyngeal and Laryngeal Diseases Frequently Mistaken for, or Associated With, Phthisis*. Edinburgh: Sutherland and Knox.

Bennett, John Hughes. 1855. *The Present State of the Theory and Practice of Medicine: an Introductory Lecture to the Class of the Institutes of Medicine in the University of Edinburgh Delivered November 6, 1855*. Edinburgh: Sutherland and Knox.

Bennett, John Hughes. 1856. *Practical Remarks on Some Points in the Physical Diagnosis of Phthisis Pulmonalis*. Edinburgh: Sutherland and Knox.

Bennett, John Hughes. 1858. *Clinical Lectures on the Principles and Practice of Medicine*. New York: Samuel S. & William Wood.

Bennett, John Hughes. 1859. *The Pathology and Treatment of Pulmonary Consumption; and the Local Medication of Pharyngeal, Laryngeal, Bronchial, and Nasal Diseases Mistaken for, or Associated With, Phthisis*. Edinburgh: Adam and Charles Black.

Bennett, John Hughes. 1860. *Universities and Corporation Under the New Medical Act: an Address to the Graduates in Medicine at the Conferring of Degrees in the University of Edinburgh, by the Vice-Chancellor and Senatus. August 1, 1860*. Edinburgh: Adam and Charles Black.

Bennett, John Hughes. 1868. *On the Atmospheric Germ Theory and Origin of Infusoria: a Lecture Delivered to the Royal College of Surgeons of Edinburgh, 17th January 1868*. Edinburgh: Adam and Charles Black.

Bennett, John Hughes. 1871. "Phthisis Pulmonalis." In *A System of Medicine*, edited by J. Russell Reynolds, 537–90. London: MacMillan and Co.

Black, D. Campbell. 1872. "Special Hospitals." *Lancet* 100: 60–61.

Bowley, Arthur L. 1900. *Wages in the United Kingdom in the Nineteenth Century*. Cambridge: Cambridge University Press.

British Congress on Tuberculosis. 1902. *Transactions of the British Congress on Tuberculosis for the Prevention of Consumption*. London: William Clowes and Sons, Limited.

Brown, John. 1795. *The Elements of Medicine*. London: J. Johnson.

Brown, Lawrason. 1928. *Rules for Recovery From Pulmonary Tuberculosis: A Layman's Handbook for Treatment*. Philadelphia: Lea and Febiger.

Buchan, William. 1790. *Domestic Medicine: Or, a Treatise on the Prevention and Cure of Diseases by Regimen and Simple Medicines*. 11th Ed. London: A. Strahan.

Buchan, William. 1796. *Observations Concerning the Prevention and Cure of the Venereal Disease*. London: Chapman.

Bunyan, John. 1680. *The Life and Death of Mr Badman; Presented to the World in a Familiar Dialogue Between Mr Wiseman and Mr Attentive*. Rothwell: Nathaniel Ponder. Reprint. 1808. London: W. Nicholson for W. Baynes.

Burton-Fanning, F. W. 1909. *The Open-Air Treatment of Pulmonary Tuberculosis*. London: Cassell and Company, Limited.

Butler, George F. 1908. *A Text-Book of Materia Medica, Pharmacology and Therapeutics*. Philadelphia: W. B. Saunders Company.

Butler, Josephine E. 1879. *Social Purity*. [Online]: Victorian Women Writers Project, www.dlib.indiana.edu/collections/vwwp/.

Cadge, W. 1875. "The Case of the Late Professor Hughes Bennett." *British Medical Journal* 2: 453–54.

Carmichael, N. 1882. "Glasgow Southern Medical Society: Session 1881 – 82, Meeting XV – 1st June, 1882." *Glasgow Medical Journal* 28: 65–69.

Carter, Thomas. 1828a. "Query: Glasgow Infirmary?" *Lancet* 11: 60.

Carter, Thomas. 1828b. "Glasgow Infirmary." *Lancet* 11: 218–20.

Chadwick, Edwin. 1842. *Report to Her Majesty's Principal Secretary of State for the Home Department, From the Poor Law Commissioners, on an Inquiry Into the Sanitary Condition of the Labouring Population of Great Britain*. London: W. Clowes and Sons.

Coats, Joseph, W. T. Gairdner, Hugh Thomson, John Lindsay Steven, Charles Workman, and J. Walker Downie. 1892. "Tuberculosis." *Lancet* 139: 218–19.

Coley, J. M. 1858. "Abuses at the London Hospitals (Letter From Dr. J. M. Coley)." *Lancet* 71: 47.

Corvisart, Jean-Nicolas. 1808. *Nouvelle méthode pour reconnaître les maladies internes de la poitrine par la percussion de cette cavité*. Paris: E. Crossman.

Council on Pharmacy and Chemistry. 1914. "Angier's Emulsion." *JAMA* 63: 962–64.

Craig, William. 1882. "The Treatment of the Night-Sweats of Phthisis by Coto." *Edinburgh Medical Journal* 27: 750.

Cullen, William. 1778. *First Lines in the Practice of Physic*. Edinburgh: W. Creech.

Cullen, William. 1784. *First Lines in the Practice of Physic*. 4th Ed. Edinburgh: C. Elliot.

Cullen, William. 1789. *A Treatise of the Materia Medica*. Edinburgh: Charles Elliot.

Cullen, William. 1797. *Clinical Lectures Delivered in the Years 1765 and 1766*. London: Messrs. Lee and Hurst.

Dale, William. 1881. "Phthisis and Infection." *Lancet* 118: 1108.

Dickens, Charles. 1859. *A Tale of Two Cities*. London: Chapman & Hall.

Dobell, Horace. 1865. "On the Assimilation of Fat in Consumption: A Third Report of Cases Treated With Pancreatic Emulsion at the Royal Infirmary for Diseases of the Chest, City-Road." *Lancet* 86: 561–62.

Duckworth, Dyce. 1901. "An Address on the Personal Factor in Tuberculosis." *Lancet* 158: 1250–53.

Duncan, Andrew. 1830. *The Edinburgh New Dispensatory*. Edinburgh: Bell & Bradfute.

Evershed. 1889. "North London Consumption Hospital: A Case of Phthisis in Which the Fatal Result was due to Pulmonary Embolism; Remarks." *Lancet* 134: 851–52.

Fellows, James I. 1882. *A Few Remarks Upon Fellow's Hypophosphites of Quinine, Strychnine, Iron, Lime, Potassa and Manganese*. London: Jas. I. Fellows.

Felter, Harvey Wickes, and John Uri Lloyd. 1909. *King's American Dispensatory*. Cincinnati OH: The Ohio Valley Company.

Fothergill, J. Milner. 1874. *The Maintenance of Health: a Medical Work for Lay Readers*. London: Smith, Elder, & Co.

Fowler, J. K. 1894. "The Influence of Heredity in Phthisis." *Lancet* 144: 1514.

Fox, Wilson. 1868. *On the Artificial Production of Tubercle in the Lower Animals*. London: MacMillan and Co.

Gavin, Hector. 1847. *Unhealthiness of London, and the Necessity of Remedial Measures*. London: John Churchill. Reprint, in *The Rise of Urban Britain: A Collection of Thirty-Five Important Titles Documenting this Major Transformation and the Responses to It*, edited by L. H. Lees and A. Lees. 1985. New York: Garland Publishing, Inc.

Gavin, Hector. 1850. *The Habitations of the Industrial Classes: Their Influence on the Physical and on the Social and Moral Condition of These Classes*. London: Society for Improving the Condition of the Labouring Classes. Reprint, in *The Rise of Urban Britain: A Collection of Thirty-Five Important Titles Documenting this Major Transformation and the Responses to It*, edited by L. H. Lees and A. Lees. 1985. New York: Garland Publishing, Inc.

Gowers, William R. 1901. "Tuberculosis and Heredity." *Lancet* 158: 1007.

Hambledon, G.W. 1890. "The Suppression of Consumption." *Science* 15: 253–58.

Heath, Christopher. 1863. "Westminster Hospital: Introductory Lecture." *Lancet* 82: 401–02.

Hope, James. 1825. "Dissertatio medica inauguralis de aortae aneurismate." MD Diss., University of Edinburgh.

J. A. A. 1882. "A Precursory Sign of Pulmonary Phthisis." *Glasgow Medical Journal* 27: 315.

Jones, Sydney. 1863. "St Thomas's Hospital: Introductory Lecture." *Lancet* 82: 399–401.

Koch, R. 1902. "Opening Address." In *Transactions of the British Congress on Tuberculosis for the Prevention of Consumption*. Vol. 3, 93–96. London: William Clowes and Sons, Limited.

Koch, Robert. 1882a. "Die Aetiologie der Tuberculose." *Berliner Klinische Wochenschrift* 19: 221–30.

Koch, Robert. 1882b. *The Aetiology of Tuberculosis. Berliner Klinische Wochenschrift* 19: 221–30. Reprint, translated by M. Pinner and B. Pinner. 1932. New York: National Tuberculosis Association.

Laënnec, R. T. H. 1819. *De l'auscultation médiate; ou traité du diagnostic des maladies des poumons et du coeur, fondé principalement sur ce nouveau moyen d'exploration*. Paris: J.-A. Brosson et J.-S. Chaudé.

Laënnec, R. T. H. 1821. *A Treatise on the Diseases of the Chest, in Which They are Described According to Their Anatomical Characters, and Their Diagnosis Established on a New Principle by Means of Acoustick Instruments*. Translated by John Forbes. London: Underwood.

Laënnec, R. T. H. 1834. *A Treatise on the Diseases of the Chest and on Mediate Auscultation: Translated From the Latest French Edition, With Copious Notes and a Sketch of the Author's Life*. Translated by John Forbes. London: Henry Renshaw.

Laënnec, R. T. H., and Mériadec Laënnec. 1831. *Traité de l'auscultation médiate et des maladies des poumons et du coeur*. Paris: J.-S. Chaudé, Libraire-Éditeur.

Lankester, E. Ray. 1880. *Degeneration: A Chapter in Darwinism*. London: Macmillan and Co.

Latham, Arthur. 1903. "Essay on the Erection of a Sanatorium for the Treatment of Tuberculosis in England." *Lancet* 1: 3–42.

Latham, Arthur. 1907. *The Diagnosis and Modern Treatment of Pulmonary Consumption*. New York: William Wood & Company.

Locock, Charles. 1821. "Dissertatio medica inauguralis de cordis papitatione." MD Diss., University of Edinburgh.

London, Jack. 1903. *The People of the Abyss*. Reprint. 1904. New York: Macmillan Company.

Lynch, Samuel. 1795. "Table of Excitement and Excitability." In *The Elements of Medicine*, edited by John Brown, n.p. London: J. Johnson.

Main, John S. 1882. "Remarks on the Premonitory Stage of Phthisis and its Importance in Relation to Treatment." *Glasgow Medical Journal* 27: 184–87.

Malthus, T. R. 1798. *An Essay on the Principle of Population*. London: J. Johnson. 4th Ed., 1807. London: T. Bensley for J. Johnson.

Marten, Benjamin. 1720. *A New Theory of Consumptions: More Especially of a Phthisis, or Consumption of the Lungs*. London: R. Knaplock.

McCormac, Henry. 1861. "On the True Nature and Absolute Preventability of Tubercular Consumption." *Dublin Quarterly Journal of Medical Science* 32: 254–65.

Mearns, Andrew, and William C. Preston. 1883. *The Bitter Cry of Outcast London: an Inquiry Into the Condition of the Abject Poor*. London: James Clarke & Co.

Milton, John. 1674. *Paradise Lost*. London: S. Simmons. Reprint, edited by Thomas H. Luxon. 2008. Dartmouth NH: The Milton Reading Room. http://www.dartmouth.edu/~milton

Moore, John. 1782. *An Essay on the Causes, Nature, and Cure, of Consumptions, in a Letter to a Friend*. Boston: Robert Hodge for William Green.

Morton, Richard. 1720. *Phthisiologia: Or, a Treatise of Consumptions. Wherein the Difference, Nature, Causes, Signs, and Cure of All Sorts of Consumptions are Explained*. London: Printed for W. and J. Innys.

N. W. 1886. "The Causation of Pulmonary Consumption." *Science* 7: 86–88.

Napier, Alexander. 1882a. "Experiments on the Infectiousness of the Blood and Urine of Tuberculous Animals." *Glasgow Medical Journal* 27: 68–69.

Napier, Alexander. 1882b. "Methods for Examining the Sputum for Tubercle-bacilli." *Glasgow Medical Journal* 28: 221–23.

Parkes, Edmund A. 1860. *The Composition of the Urine, in Health and Disease, and Under the Action of Remedies*. London: John Churchill.

Parliament of the United Kingdom of Great Britain and Ireland. 1832. *The Statutes of the United Kingdom of Great Britain and Ireland, 2 & 3 William IV*. London: His Majesty's Printers.

Parliament of the United Kingdom of Great Britain and Ireland. 1834. *A Collection of the Public General Statutes Passed in the Fourth and Fifth Year of the Reign of His Majesty King William the Fourth, 1834*. London: George Eyre and Andrew Spottiswoode.

Parliament of the United Kingdom of Great Britain and Ireland. 1845. *The Statutes of the United Kingdom of Great Britain and Ireland, 8 & 9 Victoriae*. London: Her Majesty's Printers.

Parliament of the United Kingdom of Great Britain and Ireland. 1879. *The Public General Statutes Passed in the Forty-Second and Forty-Third Years of the Reign of Her Majesty Queen Victoria, 1879*. London: The Queen's Printing Office.

Pharmaceutical Society of Great Britain. 1907. *The British Pharmaceutical Codex*. London: Pharmaceutical Society of Great Britain.

Potts, John. 1930. *Getting Well and Staying Well: A Book for Tuberculous Patients, Public Health Nurses, and Doctors*. St. Louis: The C. V. Mosby Company.

Pratt, Joseph H. 1906. "The 'Home Sanatorium' Treatment of Consumption." *Boston Medical and Surgical Journal* 154: 210–16.

Registrar-General. 1841. *Appendix to the Third Annual Report of the Registrar-General of Births, Deaths, and Marriages, in England*. London: Her Majesty's Stationery Office.

Registrar-General. 1856. *First Annual Report of the Registrar-General of Births, Deaths, and Marriages, in Scotland*. Edinburgh: Her Majesty's Stationery Office.

Registrar-General. 1857. *Eighteenth Annual Report of the Registrar-General of Births, Deaths, and Marriages in England*. London: Her Majesty's Stationery Office.

Registrar-General. 1867. *Twenty-Eighth Annual Report of the Registrar-General of Births, Deaths, and Marriages in England*. London: Her Majesty's Stationery Office.

Registrar-General. 1868. *Eleventh Detailed Annual Report of the Registrar-General of Births, Deaths, and Marriages in Scotland*. Edinburgh: Her Majesty's Stationery Office.

Registrar-General. 1877. *Thirty-Eighth Annual Report of the Registrar-General of Births, Deaths, and Marriages in England (Abstracts of 1875)*. London: Her Majesty's Stationery Office.

Registrar-General. 1880. *Twenty-First Detailed Annual Report of the Registrar-General of Births, Deaths, and Marriages in Scotland (Abstracts of 1875)*. Edinburgh: Her Majesty's Stationery Office.

Registrar-General. 1886. *Forty-Ninth Annual Report of the Registrar-General of Births, Deaths, and Marriages in England (1885)*. London: Her Majesty's Stationery Office.

Registrar-General. 1888. *Thirty-First Detailed Annual Report of the Registrar-General of Births, Deaths, and Marriages in Scotland (Abstracts of 1885)*. Edinburgh: Her Majesty's Stationery Office.

Registrar-General. 1896. *Fortieth Detailed Annual Report of the Registrar-General of Births, Deaths, and Marriages in Scotland (Abstracts of 1894)*. Edinburgh: Her Majesty's Stationery Office.

Registrar-General. 1897. *Fifty-Eighth Annual Report of the Registrar-General of Births, Deaths, and Marriages in England (1895)*. London: Her Majesty's Stationery Office.

Registrar-General. 1906. *Forty-Ninth Annual Detailed Report of the Registrar-General of Births, Deaths, and Marriages in Scotland (Abstracts of 1903)*. Glasgow: His Majesty's Stationery Office.

Registrar-General. 1907. *Sixty-Eighth Annual Report of the Registrar-General of Births, Deaths and Marriages in England and Wales (1905)*. London: His Majesty's Stationery Office.

Reynolds, J. Russell, ed. 1871. *A System of Medicine*. New York: MacMillan and Co.

Roberts, William. 1862. "Lectures on Certain Points in the Clinical Examination of Urine." *Lancet* 79: 480–82.

Royal Medical and Chirurgical Society. 1861. "Commentary on 'On the True Nature and Absolute Preventability of Tubercular Consumption' By Henry McCormac, M.D." *Lancet* 77: 434.

Sayre, Francis Bower. 1790. *An Inaugural Dissertation on the Causes Which Produce a Predisposition to Phthisis Pulmonalis, and the Method of Obviating Them*. Trenton, NJ: Isaac Collins.

Shepherd, R. J. 1888. "Phthisis a Disease of the Night." *Lancet* 131: 1000–1001.

Squire, Edward. 1894. "The Influence of Heredity in Phthisis." *Lancet* 144: 1422–23.

Steven, John Lindsay. 1882. "On the Pathological Anatomy of Tuberculosis of the Fallopian Tube." *Glasgow Medical Journal* 27: 411–18.

Stevenson, T. H. C. 1928. "The Vital Statistics of Wealth and Poverty." *Journal of the Royal Statistical Society* 91: 207–30.

Stokes, William. 1825. *An Introduction to the Use of the Stethoscope With its Application to the Diagnosis in Diseases of the Thoracic Viscera*. Edinburgh: MacLachlan & Co.

Thompson, W. Gilman. 1905. *Practical Dietetics: With Special Reference to Diet in Disease*. New York: D. Appleton and Company.

Thomson, Charles W. 1912. "Medical Science in Scotland." *The Caledonian* 12: 59–64.

Turner, A. Logan. 1937. *Story of a Great Hospital: The Royal Infirmary of Edinburgh 1729 – 1929*. Edinburgh: Oliver and Boyd, Ltd. Reprint. 1979. Edinburgh: The Mercat Press.

Villemin, J.-A. 1868. *Études sur la tuberculose: Preuves rationnelles et expérimentales de sa spécificité et de son inoculabilité*. Paris: J.-B. Baillière et Fils.

Wells, Edward F. 1893. "Calcium Sulphide in the Treatment of Pulmonary Tuberculosis." *Journal of the American Medical Association* 20: 439–40.

Wilde, Oscar. 1891. *Lord Arthur Savile's Crime and Other Stories*. London: James R. Osgood, McIlvaine and Co.

Williams, C. Theodore. 1882. "The Contagion of Phthisis." *British Medical Journal* 2: 618–21.

Williams, C. Theodore. 1883. "Remarks on the Relation of the Tubercle Bacillus to Phthisis." *Lancet* 121: 312–13.

Williams, W. Roger. 1901. "'Tuberculosis and Heredity'." *Lancet* 158: 1080.

Wilmer, Harry A. 1943. *Huber the Tuber: A Story of Tuberculosis*. New York: National Tuberculosis Association.

Young, John M. 1882. "On Agaricus in the Treatment of Night Sweating in Phthisis." *Glasgow Medical Journal* 27: 176–79.

PUBLISHED CONTEMPORARY SOURCES

Acevedo-Garcia, Dolores. 2000. "Residential Segregation and the Epidemiology of Infectious Diseases." *Social Science and Medicine* 51: 1143–61.

Atkinson, Dwight. 1992. "The Evolution of Medical Research Writing From 1735 to 1985: the Case of the *Edinburgh Medical Journal*." *Applied Linguistics* 13: 337–74.

Aufderheide, Arthur C., and Conrado Rodriguez-Martín. 1998. *The Cambridge Encyclopedia of Human Paleopathology*. Cambridge: Cambridge University Press.

Barker, Kristin. 2002. "Self-help Literature and the Making of an Illness Identity: the Case of Fibromyalgia Syndrome (FMS)." *Social Problems* 49: 279–300.

Barnes, David S. 1995. *The Making of a Social Disease: Tuberculosis in Nineteenth-Century France*. Berkeley: University of California Press.

Berridge, Virginia. 1978. "Opium Eating and the Working Class in the Nineteenth Century: The Public and Official Reaction." *British Journal of Addiction* 73: 107–12.

Boddice, Rob. 2011. "The Manly Mind? Revisiting the Victorian 'Sex in Brain' Debate." *Gender and History* 23: 321–40.

Bonner, Thomas Neville. 1995. *Becoming a Physician: Medical Education in Britain, France, Germany, and the United States, 1750 – 1945*. New York: Oxford University Press.

Bowler, Peter J. 2009. *Evolution: The History of an Idea*. Berkeley CA: University of California Press.

Broom, Dorothy H., and Roslyn V. Woodward. 1996. "Medicalisation Reconsidered: Toward a Collaborative Approach to Care." *Sociology of Health and Illness* 18: 357–78.

Brosch, R., S. V. Gordon, M. Marmiesse, P. Brodin, C. Buchrieser, K. Eiglmeier, T. Garnier, C. Gutierrez, G. Hewinson, K. Kremer, L. M. Parsons, A. S. Pym, S. Samper, D. van Soolingen, and S. T. Cole. 2002. "A New Evolutionary Scenario for the *Mycobacterium tuberculosis* Complex." *Proceedings of the National Academy of Sciences USA* 99: 3684–89.

Brown, Michael. 2010. "'Like a Devoted Army': Medicine, Heroic Masculinity, and the Military Paradigm in Victorian Britain." *Journal of British Studies* 49: 592–622.

Bryder, Linda. 1988. *Below the Magic Mountain: A Social History of Tuberculosis in Twentieth-Century Britain*. Oxford: Clarendon Press.

Burrell, Sean, and Geoffrey V. Gill. 2005. "The Liverpool Cholera Epidemic of 1832 and Anatomical Dissection — Medical Mistrust and Civil Unrest." *Journal of the History of Medicine and Allied Sciences* 60: 478–98.

Buscemi, Nicole Desiree. 2009. "Diagnosing Narratives: Illness, the Case History, and Victorian Fiction." PhD Diss., University of Iowa.

Byrne, Katherine. 2011. *Tuberculosis and the Victorian Literary Imagination*. Cambridge UK: Cambridge University Press.

Centers for Disease Control. 1992. "Prevention and Control of Tuberculosis in U.S. Communities With At-Risk Minority Populations. Recommendations of the Advisory Council for the Elimination of Tuberculosis." *Morbidity and Mortality Weekly Report* 41: 9 pp.

Centers for Disease Control. 2010. *Reported Tuberculosis in the United States, 2009*. Atlanta, GA: U.S. Department of Health and Human Services.

Centers for Disease Control. 2011. *Reported Tuberculosis in the United States, 2011*. Atlanta, GA: U.S. Department of Health and Human Services.

Clarke, Adele E., Janet K. Shim, Laura Mamo, Jennifer Ruth Fosket, and Jennifer R. Fishman. 2003. "Biomedicalization: Technoscientific Transformations of Health, Illness, and U.S. Biomedicine." *American Sociological Review* 68: 161–94.

Comaroff, Jean. 1982. "Medicine: Symbol and Ideology." In *The Problem of Medical Knowledge: Examining the Social Construction of Medicine*, edited by Peter Wright and Andrew Treacher, 49–68. Edinburgh UK: Edinburgh University Press.

Comeau, Tammy Duerden. 2007. "Gender Ideology and Disease Theory: Classifying Cancer in Nineteenth Century Britain." *Journal of Historical Sociology* 20: 158–81.

Conrad, Peter. 1994. "Wellness as Virtue: Morality and the Pursuit of Health." *Culture, Medicine and Psychiatry* 18: 385–401.

Crawford, Robert. 1994. "The Boundaries of the Self and the Unhealthy Other: Reflections on Health, Culture and AIDS." *Social Science and Medicine* 38: 1347–65.

Crofton, John. 1959. "Chemotherapy of Pulmonary Tuberculosis." *British Medical Journal* 1: 1610–14.

Das, Veena. 2001. "Stigma, Contagion, Defect: Issues in the Anthropology of Public Health" (Podium Presentation). *Stigma and Global Health: Developing a Research Agenda.* http://www.stigmaconference.nih.gov/DasPaper.htm

Das, Veena. 2006. "Poverty, Marginality, and Illness." *American Ethnologist* 33: 27–32.

Davidoff, Leonore, and Catherine Hall. 1987. *Family Fortunes: Men and Women of the English Middle Class 1780 – 1850*. London: Hutchinson.

Dievler, Anne, and Gregory Pappas. 1999. "Implications of Social Class and Race for Urban Public Health Policy Making: A Case Study of HIV/AIDS and TB Policy in Washington, DC." *Social Science and Medicine* 48: 1095–102.

Dolin, Paul John, Mario C. Raviglione, and Arata Kochi. 1993. *A Review of Current Epidemiological Data and Estimation of Future Tuberculosis Incidence and Mortality*. Geneva: World Health Organization.

Douglas, Mary. 1966. *Purity and Danger: An Analysis of Concepts of Pollution and Taboo*. London: Routledge & Kegan Paul.

Dubos, René, and Jean Dubos. 1952. *The White Plague: Tuberculosis, Man, and Society*. Boston: Little, Brown and Company. Reprint. 1996. New Brunswick, NJ: Rutgers University Press.

EMC+ Medicine Guides. 2011. *Buttercup Syrup*. Datapharm. http://services.medicines.org.uk/external/assets/printable/b/u/buttercup_syrup/buttercup_syrup.pdf

Farmer, Paul. 1999. *Infections and Inequalities: The Modern Plagues*. Berkeley: University of California Press.

Farmer, Paul. 2004. "An Anthropology of Structural Violence." *Current Anthropology* 45: 305–25.

Fido, Martin. 1988. *Bodysnatchers: A History of the Resurrectionists 1742 – 1832*. London: Weidenfeld and Nicolson.

Fletcher, Anthony. 1995. *Gender, Sex and Subordination in England 1500 – 1800*. New Haven CT: Yale University Press.

Formicola, Vincenzo, Quinzio Milanesi, and Caterina Scarsini. 1987. "Evidence of Spinal Tuberculosis at the Beginning of the Fourth Millennium BC From Arene Candide." *American Journal of Physical Anthropology* 72: 1–6.

Foucault, Michel. 1963. *The Birth of the Clinic: An Archaeology of Medical Perception.* Reprint, translated by A. M. Sheridan Smith. 1994. New York: Vintage Books.

Gadow, Sally. 1980. "Body and Self: A Dialectic." *Journal of Medicine and Philosophy* 5: 172–85.

Gallagher, Catherine. 1986. "The Body Versus the Social Body in the Works of Thomas Malthus and Henry Mayhew." *Representations* 14: 83–106.

Gandy, Matthew, and Alimuddin Zumla. 2002a. "The Resurgence of Disease: Social and Historical Perspectives on the 'New' Tuberculosis." *Social Science & Medicine* 55: 385–96.

Gandy, Matthew, and Alimuddin Zumla. 2002b. "Theorizing Tuberculosis: A Reply to Porter and Ogden." *Social Science & Medicine* 55: 399–401.

Garro, Linda C. 1992. "Chronic Illness and the Construction of Narratives." In *Pain as Human Experience: An Anthropological Perspective*, edited by Mary-Jo DelVecchio Good, Paul E. Brodwin, Byron J. Good, and Arthur Kleinman, 100–37. Berkeley CA: University of California Press.

Gilman, Sander L. 1985. *Difference and Pathology: Stereotypes of Sexuality, Race, and Madness.* Ithaca, NY: Cornell University Press.

Gilman, Sander L. 1995a. *Franz Kafka, the Jewish Patient.* New York: Routledge.

Gilman, Sander L. 1995b. *Health and Illness: Images of Difference.* London: Reaktion Books.

Gradmann, Christoph. 2001. "Robert Koch and the Pressures of Scientific Research: Tuberculosis and Tuberculin." *Medical History* 45: 1–32.

Grange, John M., and Patrick J. Bishop. 1982. "'Über Tuberkulose': A Tribute to Robert Koch's Discovery of the Tubercle Bacillus, 1882." *Tubercle* 63: 3–17.

Gregg, Jessica L. 2011. "An Unanticipated Source of Hope: Stigma and Cervical Cancer in Brazil." *Medical Anthropology Quarterly* 25: 70–84.

Gutierrez, M. Cristina, Sylvain Brisse, Roland Brosch, Michel Fabre, Bahia Omaïs, Magali Marmiesse, Philip Supply, and Veronique Vincent. 2005. "Ancient Origin and Gene Mosaicism of the Progenitor of *Mycobacterium tuberculosis*." *PLoS Pathogens* 1: e5.

Haley, Bruce. 1978. *The Healthy Body and Victorian Culture.* Cambridge MA: Harvard University Press.

Hamilton, David. 1981. *The Healers: A History of Medicine in Scotland.* Edinburgh: Canongate. Reprint. 1999. Gretna LA: Pelican Publishing.

Jarcho, Saul. 1961. "Auenbrugger, Laennec, and John Keats: Some Notes on the Early History of Percussion and Auscultation." *Medical History* 5: 167–72.

Jenkinson, Jacqueline. 2012. "A 'Crutch to Assist in Gaining an Honest Living': Dispensary Shopkeeping by Scottish General Practitioners and the Responses of the British Medical Elite, ca. 1852 – 1911." *Bulletin of the History of Medicine* 86: 1–36.

Jordanova, Ludmilla. 1996. "Medical Men 1780 – 1820." In *Portraiture: Facing the Subject*, edited by Joanna Woodall, 101–15. Manchester UK: Manchester University Press.

Joshi, Priti. 2004. "Edwin Chadwick's Self-Fashioning: Professionalism, Masculinity, and the Victorian Poor." *Victorian Literature and Culture* 32: 353–70.

Keers, R. Y. 1978. *Pulmonary Tuberculosis: A Journey Down the Centuries*. London: Baillière Tindall.

Kennedy, Meegan. 2010. *Revising the Clinic: Vision and Representation in Victorian Medical Narrative and the Novel*. Columbus OH: The Ohio State University Press.

Kleinman, Arthur, Leon Eisenberg, and Byron Good. 1978. "Culture, Illness, and Care: Clinical Lessons From Anthropologic and Cross-Cultural Research." *Annals of Internal Medicine* 88: 251–58.

Krieger, Nancy, and Sofia Gruskin. 2001. "Frameworks Matter: Ecosocial and Health and Human Rights Perspectives on Disparities in Women's Health – The Case of Tuberculosis." *Journal of the American Medical Women's Association* 56: 137–42.

Lawlor, Clark. 2006. *Consumption and Literature: The Making of the Romantic Disease*. Basingstoke, Hampshire: Palgrave MacMillan.

Lawlor, Clark, and Akihito Suzuki. 2000. "The Disease of the Self: Representing Consumption, 1700-1830." *Bulletin of the History of Medicine* 74: 458–94.

Lawrence, Christopher. 1985. "Incommunicable Knowledge: Science, Technology and the Clinical Art in Britain 1850 – 1914." *Journal of Contemporary History* 20: 503–20.

Lawrence, Christopher. 1988. "Cullen, Brown and the Poverty of Essentialism." In *Brunonianism in Britain and Europe*, edited by W. F. Bynum and Roy Porter, 1–21. London: Wellcome Institute for the History of Medicine.

Lawrence, Christopher. 1998. "Medical Minds, Surgical Bodies: Corporeality and the Doctors." In *Science Incarnate: Historical Embodiments of Natural Knowledge*, edited by Christopher Lawrence and Steven Shapin, 156–201. Chicago IL: University of Chicago Press.

Lawrence, Christopher. 2006. "The Shaping of Things to Come: Scottish Medical Education 1700-1939." *Medical Education* 40: 212–18.

Lawrence, Jody, Robin A. Kearns, Julie Park, Linda Bryder, and Heather Worth. 2008. "Discourses of Disease: Representations of Tuberculosis Within New Zealand Newspapers 2002 – 2004." *Social Science & Medicine* 66: 727–39.

Lawrence, Susan C. 1993. "Educating the Senses: Students, Teachers and Medical Rhetoric in Eighteenth-century London." In *Medicine and the Five Senses*, edited by W.F. Bynum and Roy Porter, 154–78. Cambridge: Cambridge University Press.

Long, Nguyen Hoang, Eva Johansson, Vinod K. Diwan, and Anna Winkvist. 1999. "Different Tuberculosis in Men and Women: Beliefs From Focus Groups in Vietnam." *Social Science and Medicine* 49: 815–22.

Lönnroth, Knut, Brian G. Williams, Peter Cegielski, and Christopher Dye. 2010. "A Consistent Log-linear Relationship Between Tuberculosis Incidence and Body Mass Index." *International Journal of Epidemiology* 39: 149–55.

Lorber, Judith. 1967. "Deviance as Performance: The Case of Illness." *Social Problems* 14: 302–10.

Luckin, Bill. 2006. "Revisiting the Idea of Degeneration in Urban Britain, 1830-1900." *Urban History* 33: 234–52.

Marshall, Tim. 1995. *Murdering to Dissect: Grave-robbing, Frankenstein and the Anatomy Literature*. Manchester, UK: Manchester University Press.

McFarland, C. T., Y. Y. Fan, R. S. Chapkin, B. R. Weeks, and D. N. McMurray. 2008. "Dietary Polyunsaturated Fatty Acids Modulate Resistance to *Mycobacterium tuberculosis* in Guinea Pigs." *Journal of Nutrition* 138: 2123–28.

McMichael, Anthony J. 1995. "The Health of Persons, Populations, and Planets: Epidemiology Comes Full Circle." *Epidemiology* 6: 633–36.

Menegoni, Lorenza. 1996. "Conceptions of Tuberculosis and Therapeutic Choices in Highland Chaipas, Mexico." *Medical Anthropology Quarterly* 10: 381–401.

Michailakis, Dimitris, and Werner Schirmer. 2010. "Agents of Their Health? How the Swedish Welfare State Introduces Expectations of Individual Responsibility." *Sociology of Health & Illness* 32: 930–47.

Miravitlles, Marc, Frank Kruesmann, Daniel Haverstock, Renee Perroncel, Shurjeel H. Choudhri, and Pierre Arvis. 2012. "Sputum Colour and Bacteria in Chronic Bronchitis Exacerbations: A Pooled Analysis." *European Respiratory Journal* 39: 1354–60.

Mortimer, Ian. 2009. *The Dying and the Doctors: The Medical Revolution in Seventeenth-Century England*. Woodbridge UK: Boydell & Brewer.

Morton, R. S. 1967. "The Sibbens of Scotland." *Medical History* 11: 374–80.

Muenning, Peter. 2008. "The Body Politic: The Relationship Between Stigma and Obesity-Associated Disease." *BMC Public Health* 8: 128 (10 pp.).

Myers, J. Arthur. 1974. "Development of Knowledge of Unity of Tuberculosis and of the Portals of Entry of Tubercle Bacilli." *Journal of the History of Medicine and Allied Sciences* 29: 213–28.

National Health and Nutrition Examination Survey. 2011. *Tuberculosis Skin Test Procedures Manual*. Atlanta GA: Centers for Disease Control.

Nenadic, Stana. 2010. "Writing Medical Lives, Creating Posthumous Reputations: Dr Matthew Baillie and his Family in the Nineteenth Century." *Social History of Medicine* 23: 509–27.

Nichter, Mark. 1994. "Illness Semantics and International Health: The Weak Lungs/TB Complex in the Philippines." *Social Science and Medicine* 38: 649–63.

Nicolson, Malcolm. 1993. "The Introduction of Percussion and Stethoscopy to Early Nineteenth-Century Edinburgh." In *Medicine and the Five Senses*, edited by W. F. Bynum and Roy Porter, 134–53. Cambridge: Cambridge University Press.

O'Neal, John C. 1998. "Auenbrugger, Corvisart, and the Perception of Disease." *Eighteenth-Century Studies* 31: 473–89.

Office of Dietary Supplements. 2011. "Dietary Supplement Fact Sheet: Vitamin D." http://ods.od.nih.gov/factsheets/VitaminD-HealthProfessional/

Office of Dietary Supplements. 2012. "Dietary Supplement Fact Sheet: Vitamin A." http://ods.od.nih.gov/factsheets/VitaminA-HealthProfessional/

Parker, Richard, and Peter Aggleton. 2003. "HIV and AIDS-Related Stigma and Discrimination: A Conceptual Framework and Implications for Action." *Social Science & Medicine* 57: 13–24.

Parsons, Talcott. 1951. *The Social System*. Glencoe IL: The Free Press.

Paton, Nicholas I., Yueh-Khim Chua, Arul Earnest, and Cynthia B. E. Chee. 2004. "Randomized Controlled Trial of Nutritional Supplementation in Patients With Newly Diagnosed Tuberculosis and Wasting." *American Journal of Clinical Nutrition* 80: 460–65.

Pender, Stephen. 2006. "Examples and Experience: On the Uncertainty of Medicine." *British Journal for the History of Science* 39: 1–28.

Pinell, Patrice. 2001. "Degeneration Theory and Heredity Patterns Between 1850 and 1900." In *Heredity and Infection: The History of Disease Transmission*, edited by Jean-Paul Gaudillière and Ilana Löwy, London: Routledge.

Poovey, Mary. 1986. "'Scenes of an Indelicate Character': The Medical 'Treatment' of Victorian Women." *Representations* 14: 137–68.

Porter, Dorothy, and Roy Porter. 1989. *Patient's Progress: Doctors and Doctoring in Eighteenth-century England*. Stanford CA: Stanford University Press.

Porter, John D. H., and Jessica A. Ogden. 2002. "Commentary on 'The Resurgence of Disease: Social and Historical Perspectives on the 'New' Tuberculosis'." *Social Science & Medicine* 55: 397–98.

Porter, Roy. 1985. "Making Faces: Physiognomy and Fashion in Eighteenth-Century England." *Etudes Anglaises* 38: 385–96.

Porter, Roy. 1993. "The Rise of Physical Examination." In *Medicine and the Five Senses*, edited by W.F. Bynum and Roy Porter, 179–97. Cambridge: Cambridge University Press.

Promtussananon, Supa, and Karl Peltzer. 2005. "Perceptions of Tuberculosis: Attributions of Cause, Suggested Means of Risk Reduction, and Preferred Treatment in the Limpopo Province, South Africa." *Journal of Health, Population, and Nutrition* 23: 74–81.

R Development Core Team. 2012. *R: A Language and Environment for Statistical Computing*. Vienna, Austria: R Foundation for Statistical Computing.

Reed, Caroline M. 1998. "Parrish's Chemical Food Never Contained Arsenic." *BMJ* 317: 212.

Richardson, Ruth. 1988. *Death, Dissection and the Destitute*. London: Phoenix Press.

Rolleston, H. D., and Alexander G. Bearn. 2004. "Allbutt, Sir (Thomas) Clifford (1836 – 1925)." In *Oxford Dictionary of National Biography*, 3 pp. Oxford University Press. http://www.oxforddnb.com/view/printable/30382

Rosenberg, Charles E. 1997. "The Bitter Fruit: Heredity, Disease, and Social Thought." In *No Other Gods: on Science and American Social Thought*, 25–53. Baltimore MD: The Johns Hopkins University Press.

Rosenman, Kenneth D., and Nancy Hall. 1998. "Occupational Risk Factors for Developing Tuberculosis." *American Journal of Industrial Medicine* 30: 148–54.

Rosner, Lisa. 2010. *The Anatomy Murders: Being the True and Spectacular History of Edinburgh's Notorious Burke and Hare and the Man of Science Who Abetted Them in the Commission of Their Most Heinous Crimes*. Philadelphia PA: University of Pennsylvania Press.

Rubel, Arthur J., and Linda C. Garro. 1992. "Social and Cultural Factors in the Successful Control of Tuberculosis." *Public Health Reports* 107: 626–36.

Sakula, Alex. 1982. "Robert Koch: Centenary of the Discovery of the Tubercle Bacillus, 1882." *Thorax* 37: 246–51.

Scambler, Graham. 2009. "Health-Related Stigma." *Sociology of Health & Illness* 31: 441–55.

Sharma, Sujata, and Mark A. Yoder. 2011. "New Weapons in the War on Tuberculosis." *American Journal of Therapeutics* 18: e101–12.

Showalter, Elaine. 1980. "Victorian Women and Insanity." *Victorian Studies* 23: 157–81.

Smith, F. B. 1979. *The People's Health 1830 – 1910*. New York: Holmes & Meier Publishers, Inc.

Smith, F. B. 1988. *The Retreat of Tuberculosis 1850 – 1950*. London: Croom Helm.

Sontag, Susan. 1978. *Illness as Metaphor*. New York: Farrar, Straus, and Giroux.

Spence, D. P. S., J. Hotchkiss, C. S. D. Williams, and P. D. O. Davies. 1993. "Tuberculosis and Poverty." *BMJ* 307: 759–61.

Stedman Jones, Gareth. 1971. *Outcast London: A Study in the Relationship Between Classes in Victorian Society*. Oxford: Clarendon Press.

Stockley, Robert A., Christine O'Brien, Anita Pye, and Susan L. Hill. 2000. "Relationship of Sputum Color to Nature and Outpatient Management of Acute Exacerbations of COPD." *Chest* 117: 1638–45.

Stott, Rosalie. 1987. "Health and Virtue: Or, How to Keep Out of Harm's Way. Lectures on Pathology and Therapeutics By William Cullen *c.* 1770." *Medical History* 31: 123–42.

Sundareshan, Vidya, and Martin E. Evans. 2010. "Tuberculosis." In *Management of Antimicrobials in Infectious Disease: Impact of Antibiotic Resistance*, edited by Arch G. Mainous and Claire Pomeroy, 61–78. New York: Humana Press.

Taussig, Michael T. 1980. "Reification and the Consciousness of the Patient." *Social Science & Medicine* 14B: 3–13.

Tesh, Sylvia Noble. 1988. *Hidden Arguments: Political Ideology and Disease Prevention Policy*. New Brunswick, NJ: Rutgers University Press.

Thompson, E. P. 1963. *The Making of the English Working Class*. London: Victor Gollancz Ltd. Reprint. 1966. New York, NY: Vintage Books.

United States Department of Agriculture. 2011. "National Nutrient Database for Standard Reference." http://ndb.nal.usda.gov/ndb/foods/list

Wall, Rosemary. 2011. "Using Bacteriology in Elite Hospital Practice: London and Cambridge, 1880 – 1920." *Social History of Medicine* 24: 776–95.

Wallis, Patrick. 2012. "Exotic Drugs and English Medicine: England's Drug Trade, *c.* 1550 – *c.* 1800." *Social History of Medicine* 25: 20–46.

Walpola, H. C., V. Siskind, A. M. Patel, A. Konstantios, and P. Derhy. 2003. "Tuberculosis-Related Deaths in Queensland, Australia, 1989 – 1998: Characteristics and Risk Factors." *International Journal of Tuberculosis and Lung Disease* 7: 742–50.

Wirth, Thierry, Falk Hildebrand, Caroline Allix-Béguec, Florian Wölbeling, Tanja Kubica, Kristin Kremer, Dick van Soolingen, Sabine Rüsch-Gerdes, Camille Locht, Sylvain Brisse, Axel Meyer, Philip Supply, and Stefan Niemann. 2008. "Origin, Spread and Demography of the *Mycobacterium tuberculosis* Complex." *PLoS Pathogens* 4: e1000160 (10 pp.).

Wolfe, Nathan D., Claire Panosian Dunavan, and Jared Diamond. 2007. "Origins of Major Human Infectious Diseases." *Nature* 447: 279–83.

Worboys, Michael. 2000. *Spreading Germs: Disease Theories and Medical Practice in Britain, 1865 – 1900*. Cambridge: Cambridge University Press.

Worboys, Michael. 2001. "From Heredity to Infection: Tuberculosis, 1870 – 1890." In *Heredity and Infection: The History of Disease Transmission*, edited by Jean-Paul Gaudillière and Ilana Löwy, 81–100. London: Routledge.

World Health Organization. 2010. *Fact Sheet No. 104: Tuberculosis*. Geneva: WHO/OMS.

World Health Organization. 2013. *Fact Sheet No. 104: Tuberculosis*. Geneva: WHO/OMS.

APPENDIX A

DOCUMENTATION OF IRB EXEMPTION

ASU ARIZONA STATE UNIVERSITY
RESEARCH AND ECONOMIC AFFAIRS

Office of Research Integrity and Assurance

To:	Jane Buikstra ANTH
From:	Mark Roosa, Chair Soc Beh IRB
Date:	12/16/2008
Committee Action:	**Exemption Granted**
IRB Action Date:	12/16/2008
IRB Protocol #:	0812003544
Study Title:	Medical Constructs of the Consumptive in Nineteenth-Century Scotland

The above-referenced protocol is considered exempt after review by the Institutional Review Board pursuant to Federal regulations, 45 CFR Part 46.101(b)(4) .

This part of the federal regulations requires that the information be recorded by investigators in such a manner that subjects cannot be identified, directly or through identifiers linked to the subjects. It is necessary that the information obtained not be such that if disclosed outside the research, it could reasonably place the subjects at risk of criminal or civil liability, or be damaging to the subjects' financial standing, employability, or reputation.

You should retain a copy of this letter for your records.

APPENDIX B

FREQUENCIES OF DIAGNOSES CLASSIFIED AS TB-RELATED

	GRI		RIE	
Diagnosis	Admissions	Case Notes	Admissions	Case Notes
Cachexia	2	1	—	—
Consumption	2	1	2	—
Haemoptysis	46	8	51	8
Hydrocephalus	9	1	12	—
Lupus	58	4	70	9
Phthisis	1361	169	1322	259
[Unspecified]	*982*	*126*	*543*	*192*
Abdominal	*3*	—	*1*	—
Fibroid	*10*	—	*3*	—
Glandular	—	*1*	—	—
Incipient	*6*	*10*	*91*	*17*
Laryngeal	*8*	—	*7*	*1*
Lesion	*1*	*1*	—	—
Meningeal	—	—	—	*1*
Miner's/Stone Mason's	—	—	*2*	*2*
Pulmonary	*351*	*30*	*673*	*46*
Skeletal	—	—	*2*	—
Urogenital	—	*1*	—	—
Pott's Disease of Spine	56	9	10	1
Angular Curvature	*21*	—	—	—
Pott's Curvature/Disease	*33*	*8*	*10*	—
Spinal Caries	*2*	*1*	—	*1*
Psoas Abscess	38	2	22	—
Scrofulous/Strumous	227	9	248	5
[Unspecified]	*16*	—	*11*	*1*
Abdominal	*1*	—	—	—
Dermal	*12*	—	*3*	—
General	—	—	*1*	—
Glandular	*26*	—	*17*	*2*
Hernia	*1*	—	—	—
Lesion	*52*	*3*	*43*	*1*
Ocular	*8*	*1*	*53*	—
Skeletal	*93*	*2*	*112*	*1*
Laryngeal	*1*	—	—	—
Tumor	*5*	—	—	—
Urogenital	*12*	*3*	*8*	—

Appendix B (*continued*)

	GRI		RIE	
Diagnosis	Admissions	Case Notes	Admissions	Case Notes
Sub-tubercular nephritis	2	—	—	—
Tabes mesenterica	25	1	7	4
Tuberculosis/Tubercular	759	47	113	18
[Unspecified]	*25*	*6*	*19*	*7*
Abdominal	*58*	*14*	*18*	*5*
Cerebral	*2*	—	—	—
Dermal	*2*	—	*9*	—
Disseminated/Miliary	*10*	—	*2*	—
Glandular	*157*	*4*	*1*	*1*
Incipient	—	—	*1*	—
Laryngeal	*9*	—	*4*	*1*
Lesion	*77*	*1*	*2*	*1*
Meningeal	*22*	*3*	*13*	*1*
Muscular	*1*	—	—	—
Ocular	*1*	—	—	—
Phthisis	*1*	*5*	*7*	—
Pulmonary	*25*	*6*	*27*	*1*
Skeletal	*324*	*5*	*3*	—
Syphilitic	*1*	—	*1*	*1*
Urogenital	*44*	*3*	*6*	—
TOTAL	2585	252	1857	304

APPENDIX C

SIGNS AND SYMPTOMS RECORDED IN SAMPLED CASES

FOR THE ESSENTIALIST PERIOD, 1794 – 1820

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
1. APPEARANCE AND COMPORTMENT	44	18	13	75
Anxiety	*1*	*2*	*0*	*3*
Chest, Narrow	*0*	*1*	*0*	*1*
Debility	*17*	*4*	*5*	*26*
Dizziness	*0*	*1*	*3*	*4*
Emaciation[1]	*13*	*2*	*2*	*17*
Eyes, Yellow	*0*	*0*	*1*	*1*
Languor[2]	*2*	*1*	*0*	*3*
Pallor	*1*	*0*	*2*	*3*
Position	—	—	—	—
Back[3]	*1*	*0*	*0*	*1*
Head Raised	*1*	*1*	*0*	*2*
Right Side	*2*	*2*	*0*	*4*
Unaffected Side	*4*	*4*	*0*	*8*
Other[4]	*2*	*0*	*0*	*2*
2. CIRCULATORY	30	20	9	59
Heart, Beat Anomalies[5]	*2*	*5*	*0*	*7*
Heart, Pain	*0*	*1*	*0*	*1*
Pulse	—	—	—	—
75 – 84	*1*	*1*	*4*	*6*
85 – 94	*1*	*1*	*2*	*4*
95 – 104	*8*	*3*	*2*	*13*
105 – 114	*8*	*3*	*1*	*12*
115 and Above	*6*	*3*	*0*	*9*
Feeble/Weak	*2*	*0*	*0*	*2*
Hard/Sharp	*2*	*3*	*0*	*5*
3. DERMATOLOGICAL	25	9	30	64
Encrustation/Discharge	*1*	*0*	*6*	*7*
Flushing/Redness	*3*	*0*	*3*	*6*
Node[6]	*0*	*0*	*3*	*3*
Node, Pain/Inflammation	*0*	*0*	*4*	*4*
Perspiration	—	—	—	—
Frequent/Profuse	*4*	*1*	*1*	*6*
Night	*6*	*1*	*0*	*7*
Other[7]	*2*	*0*	*2*	*4*
Rash	*2*	*0*	*3*	*5*

Appendix C (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
3. DERMATOLOGICAL, CONT.				
Skin	—	—	—	—
Cool	*0*	*1*	*0*	*1*
Dry/Harsh	*2*	*2*	*1*	*5*
Warm/Hot	*3*	*3*	*2*	*8*
Ulcer, Neck/Chest	*2*	*0*	*0*	*2*
Ulcer, Oral/Facial	*0*	*1*	*5*	*6*
4. DIGESTIVE	49	13	20	82
Acidity[8]	*4*	*0*	*1*	*5*
Anorexia	*15*	*7*	*6*	*28*
Borborygmus[9]	*1*	*2*	*1*	*4*
Bowels	—	—	—	—
Constipation	*7*	*4*	*3*	*14*
Diarrhea	*7*	*0*	*1*	*8*
Gripes/Tormina	*2*	*0*	*0*	*2*
Stool Anomalies[10]	*1*	*0*	*2*	*3*
Flatulence	*4*	*0*	*1*	*5*
Nausea/Retching/Vomiting	*6*	*0*	*4*	*10*
Other Stomach[11]	*2*	*0*	*1*	*3*
5. GENERAL	38	20	9	67
Ear Anomalies[12]	*1*	*1*	*0*	*2*
Sleep, Disturbed	*7*	*6*	*3*	*16*
Temperature	—	—	—	—
Alternations[13]	*1*	*0*	*0*	*1*
Fever/Heat	*4*	*0*	*3*	*7*
Shivering	*6*	*2*	*1*	*9*
Thirst, Increased	*16*	*6*	*2*	*24*
Thirst, Reduced	*0*	*2*	*0*	*2*
Tumor, All Types	*3*	*3*	*0*	*6*
6. GENITOURINARY	5	4	7	16
Amenorrhea	*2*	*0*	*3*	*5*
Urine	—	—	—	—
Frequent/Increased	*0*	*1*	*1*	*2*
High Colored	*1*	*1*	*1*	*3*
Incontinence	*1*	*0*	*1*	*2*
Scanty	*1*	*2*	*1*	*4*

Appendix C (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
7. HISTORY	47	21	22	90
Duration	—	—	—	—
< 1 Month	*2*	*6*	*3*	*1*
1 – 4 Months[14]	*10*	*3*	*2*	*4*
5 – 8 Months	*3*	*0*	*1*	*4*
≥ 9 Months	*7*	*0*	*3*	*10*
Onset, Sudden	*0*	*1*	*0*	*1*
Previous	—	—	—	—
Expectoration, Blood	*1*	*0*	*0*	*1*
Exposure to Elements	*8*	*1*	*1*	*10*
Fatigue/Hardship	*2*	*3*	*1*	*6*
Injury	*2*	*1*	*2*	*5*
OB/GYN[15]	*0*	*1*	*1*	*2*
Non-STI Disorder[16]	*8*	*4*	*1*	*13*
STI[17]	*1*	*0*	*4*	*5*
Other[18]	*1*	*0*	*3*	*4*
Seasonal Disorder, Fall/Winter	*2*	*1*	*0*	*3*
8. MOUTH AND THROAT	21	12	16	49
Dental/Alveolar Disease[19]	*1*	*0*	*1*	*2*
Difficulty Speaking/Hoarseness	*1*	*1*	*0*	*2*
Difficulty Swallowing[20]	*1*	*0*	*2*	*3*
Edema/Swelling	*0*	*0*	*2*	*2*
Inflamed/Livid	*2*	*0*	*2*	*4*
Pain/Soreness/Cynanche	*1*	*1*	*3*	*5*
Tongue	—	—	—	—
Dry/Parched	*2*	*1*	*1*	*4*
Foul/Furred[21]	*6*	*2*	*3*	*11*
Red	*1*	*1*	*0*	*2*
White	*5*	*6*	*2*	*13*
Uvula, Short	*1*	*0*	*0*	*1*
9. PAIN	57	34	25	116
Acute/Severe/Violent	*2*	*1*	*1*	*4*
Dull/General/Unspecified[22]	*6*	*2*	*1*	*9*
Face/Head	*3*	*4*	*5*	*12*
Increased by	—	—	—	—
Cough/Inspiration	*13*	*5*	*0*	*18*
Exertion/Motion	*1*	*1*	*1*	*3*
Pressure	*3*	*2*	*2*	*7*
Other[23]	*1*	*1*	*2*	*4*

Appendix C (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
9. PAIN, CONT.				
Limbs, Lower	*2*	*1*	*2*	*5*
Trunk, Back/Shoulders	*4*	*5*	*4*	*13*
Trunk, Chest/Thorax	*14*	*10*	*1*	*25*
Trunk, Other[24]	*8*	*2*	*6*	*16*
10. RESPIRATORY	128	54	7	189
Congestion, Chest	*1*	*1*	*0*	*2*
Cough	—	—	—	—
Distressing[25]	*12*	*10*	*0*	*22*
Disturbs Sleep	*6*	*0*	*0*	*6*
Evening/Night	*4*	*3*	*1*	*8*
Little[26]	*1*	*3*	*1*	*5*
Longstanding	*1*	*1*	*0*	*2*
Morning	*1*	*1*	*0*	*2*
Other[27]	*1*	*0*	*1*	*2*
Unspecified	*14*	*3*	*2*	*19*
Dyspnea, All Types	*20*	*9*	*0*	*29*
Expectoration	—	—	—	—
Abundant[28]	*11*	*3*	*0*	*14*
Bloody	*6*	*1*	*1*	*8*
Dense[29]	*8*	*5*	*0*	*13*
Easy/Frothy	*1*	*0*	*1*	*2*
Ill-Tasting[30]	*4*	*1*	*0*	*5*
Mucus	*3*	*1*	*0*	*4*
Opaque/White	*1*	*4*	*0*	*5*
Pus[31]	*4*	*0*	*0*	*4*
Scanty	*2*	*1*	*0*	*3*
Viscid	*7*	*2*	*0*	*9*
Yellow	*7*	*0*	*0*	*7*
Other	*4*	*0*	*0*	*4*
Hemoptysis, All Types	*8*	*0*	*0*	*8*
Respiration Anomalies[32]	*1*	*5*	*0*	*6*

Appendix C (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
11. MISCELLANEOUS	10	3	19	32
Abdomen, Uneasiness[33]	*1*	*0*	*0*	*1*
Abdomen, Edema[34]	*2*	*1*	*0*	*3*
Bone and Joint	—	—	—	—
Hip Joint[35]	*0*	*0*	*6*	*6*
Pain, Knees	*0*	*0*	*1*	*1*
Pain, Sacrum	*1*	*0*	*0*	*1*
Rheumatism[36]	*1*	*0*	*4*	*5*
Edema, Limbs, Lower	*4*	*2*	*1*	*7*
Edema, Other	*1*	*0*	*7*	*8*
TOTAL	454	208	177	839

[1] Also includes weight loss more generally, which accounts for only one count in each diagnosis group.

[2] Also includes the related symptoms drowsiness and dullness.

[3] This and the subsequent four "position" categories describe the position in which the patient reported feeling most comfortable.

[4] Includes those patients who reported they were unable to lie on their unaffected side (i.e., the side free of chest pain).

[5] Includes irregular, jarring or obscure heartbeat, irregular pulse, and palpitations.

[6] "Node" most often refers to the external appearance of treponemal periostitis.

[7] Includes cold, sour-smelling and unspecified perspirations.

[8] Includes acid stomach, acid eructations (belching), and pyrosis (heartburn).

[9] Includes abdominal fluctuation and sensation of motion in the abdomen, as well as borborygmus (abdominal gurgling due to fluid and/or gas).

[10] Includes stool described as bloody, feculent, or slimy.

[11] Includes abdominal distension, weak stomach, and uneasy stomach.

[12] Includes tinnitus auriam and an unspecified ear affection.

[13] The patient reported alternating sensations of heat and cold.

[14] Includes one non-TB pulmonary case with a duration of "several" months.

[15] Includes cases in which the onset of disease was linked to menstruation or childbirth.

[16] Includes histories of cold, diarrhea, dysentery, fever, tumors, or ulcer (i.e., of the skin).

[17] Includes explicit reference to prior episodes of syphilis, as well as reference to a history of chancres, the salient feature of primary syphilis.

[18] Includes histories of cold bathing after perspiration, exposure to contagion, or occupational exposure to lead.

[19] Includes maxillary suppuration and loose teeth with pus at the roots, both likely resulting from treponemal infection.

[20] Also includes globus hystericus, the sensation of having something caught in one's throat even when no obstruction is located.

[21] Also includes loaded and streaked tongues.

[22] Also includes "muscular" pain of unspecified anatomical position.

[23] Includes pain increased after eating, after resting, at night, and upon stooping.

[24] Includes pain, soreness or stitch in the abdomen, breasts, side, or torso.

[25] Also includes coughs described as frequent, hard, much, or severe.

[26] Also includes coughs described as short, slight, or tickling.

[27] Includes a dry cough and a cough increased upon taking liquids.

[28] Also includes expectoration described as copious or frequent.

[29] Also includes expectoration described as difficult, thick, or tough.

[30] Also includes expectoration described as bitter or salty.

[31] Also includes expectoration described as purulent or puriform.

[32] Includes difficult, impaired, or noisy respiration.

[33] Includes a feeling of uneasiness referred to the hypochondrium, or the region of the stomach and liver.

[34] Also includes abdominal swelling or tumefaction.

[35] Includes flattening, flaccidity, lowering, and pain or soreness of the hip joint.

[36] Also includes joint stiffness.

APPENDIX D

SIGNS AND SYMPTOMS RECORDED IN SAMPLED CASES

FOR THE PATHOLOGICAL PERIOD I, 1821 – 1840

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
1. APPEARANCE AND COMPORTMENT	59	19	13	91
Anxiety	*5*	*2*	*1*	*8*
Constitution, Delicate/Weak	*5*	*1*	*0*	*6*
Countenance, Collapsed/Sunk	*2*	*0*	*0*	*2*
Countenance, Other[1]	*1*	*1*	*0*	*2*
Debility[2]	*17*	*4*	*4*	*25*
Dizziness[3]	*0*	*2*	*4*	*6*
Emaciation[4]	*19*	*0*	*2*	*21*
Eyes, Abnormal Appearance[5]	*1*	*1*	*0*	*2*
Languor	*1*	*0*	*0*	*1*
Pallor	*2*	*0*	*1*	*3*
Position	—	—	—	—
Back[6]	*1*	*3*	*0*	*4*
Difficulty, Back	*2*	*1*	*0*	*3*
Difficulty, Left	*0*	*1*	*0*	*1*
Head Raised[7]	*1*	*0*	*0*	*1*
Right Side	*1*	*1*	*0*	*2*
Unaffected Side	*0*	*1*	*1*	*2*
Other[8]	*0*	*1*	*0*	*1*
Temperament, Bilious	*1*	*0*	*0*	*1*
2. CIRCULATORY	60	52	31	143
Auscultation	—	—	—	—
Heart, Action Decreased[9]	*0*	*2*	*0*	*2*
Heart, Action Increased[10]	*4*	*0*	*0*	*4*
Heart, 1st Sound Anomalies[11]	*1*	*2*	*2*	*5*
Heart, 2nd Sound Anomalies[12]	*1*	*2*	*0*	*3*
Heart, Beat Anomalies[13]	*6*	*6*	*4*	*16*
Heart, Oppression/Weakness[14]	*1*	*2*	*0*	*3*
Pulse	—	—	—	—
≤ 74	*2*	*5*	*5*	*12*
75 – 84	*6*	*8*	*3*	*17*
85 – 94	*2*	*1*	*2*	*5*
95 – 104	*7*	*5*	*4*	*16*
105 – 114	*5*	*0*	*0*	*5*
≥ 115	*11*	*2*	*3*	*16*
Bounding[15]	*1*	*1*	*1*	*3*
Feeble/Weak[16]	*11*	*8*	*3*	*22*
Hard/Sharp[17]	*2*	*8*	*4*	*14*

Appendix D (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
3. DERMATOLOGICAL	46	20	26	92
Encrustation/Discharge[18]	*3*	*0*	*3*	*6*
Flushing/Redness[19]	*8*	*6*	*0*	*14*
Perspiration	—	—	—	—
Frequent/Profuse	*7*	*0*	*0*	*7*
Night	*10*	*3*	*0*	*13*
Other[20]	*5*	*1*	*0*	*6*
Rash	*0*	*1*	*11*	*12*
Skin	—	—	—	—
Cool	*1*	*1*	*0*	*2*
Dry/Harsh[21]	*4*	*1*	*1*	*6*
Warm/Hot	*2*	*2*	*4*	*8*
Other[22]	*0*	*3*	*1*	*4*
Ulcer	—	—	—	—
Genital[23]	*0*	*0*	*3*	*3*
Limbs[24]	*2*	*0*	*1*	*3*
Neck/Chest	*1*	*0*	*0*	*1*
Oral/Facial[25]	*1*	*2*	*2*	*5*
Unspecified[26]	*2*	*0*	*0*	*2*
4. DIGESTIVE	46	26	36	108
Abdominal Fluctuation	*1*	*2*	*3*	*6*
Acidity[27]	*2*	*1*	*2*	*5*
Anorexia[28]	*11*	*6*	*3*	*20*
Bowels	—	—	—	—
Constipation[29]	*3*	*8*	*12*	*23*
Diarrhea[30]	*9*	*1*	*1*	*11*
Gripes/Tenesmus	*1*	*2*	*0*	*3*
Stool Anomalies[31]	*5*	*4*	*0*	*9*
Flatulence	*2*	*0*	*6*	*8*
Nausea/Retching/Vomiting	*10*	*0*	*8*	*18*
Other Digestive[32]	*2*	*1*	*2*	*5*
5. GENERAL	41	16	17	74
Complaint Alleviated by Heat	*0*	*0*	*2*	*2*
General Health Impaired[33]	*5*	*0*	*2*	*7*
Sleep, Disturbed[34]	*13*	*6*	*5*	*24*

Appendix D (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
5. GENERAL, CONT.				
Temperature	—	—	—	—
Alternations[35]	*3*	*1*	*0*	*4*
Fever/Heat	*2*	*1*	*0*	*3*
Shivering[36]	*8*	*5*	*3*	*16*
Thirst, Increased	*10*	*3*	*5*	*18*
6. GENITOURINARY	12	5	14	31
Amenorrhea	*4*	*1*	*2*	*7*
Urine	—	—	—	—
Difficult/Obstructed	*0*	*0*	*3*	*3*
High Colored	*2*	*2*	*2*	*6*
Muddy/Sediment	*2*	*2*	*3*	*7*
Scanty	*4*	*0*	*1*	*5*
Specific Gravity Recorded	*0*	*0*	*1*	*1*
Vagina, Discharge	*0*	*0*	*2*	*2*
7. HISTORY	114	74	70	258
Duration	—	—	—	—
< 1 Month	*4*	*11*	*6*	*21*
1 – 4 Months	*16*	*6*	*9*	*31*
5 – 8 Months	*2*	*0*	*2*	*4*
≥ 9 Months	*9*	*4*	*5*	*18*
Precipitating Factors	—	—	—	—
Disorder, Pulmonary	*26*	*25*	*2*	*53*
Disorder, STI	*3*	*0*	*10*	*13*
Disorder, Other	*19*	*11*	*18*	*48*
Expectoration, Blood	*5*	*3*	*0*	*8*
Exposure to Elements	*12*	*3*	*5*	*20*
Fatigue[37]	*3*	*0*	*2*	*5*
Immorality[38]	*2*	*0*	*6*	*8*
Injury	*2*	*0*	*0*	*2*
OB/GYN[39]	*0*	*1*	*2*	*3*
Other[40]	*0*	*0*	*2*	*2*
Seasonal Disorder, Fall/Winter	*1*	*0*	*0*	*1*
Symptoms	—	—	—	—
Pain, Trunk	*3*	*4*	*0*	*7*
Perspiration, Night	*2*	*1*	*0*	*3*
Shivering	*3*	*4*	*1*	*8*
Other	*2*	*1*	*0*	*3*

Appendix D (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
8. MOUTH AND THROAT	15	32	28	75
Dental/Alveolar Disease[41]	*0*	*0*	*2*	*2*
Difficulty Speaking/Hoarseness	*1*	*2*	*1*	*4*
Difficulty Swallowing[42]	*0*	*1*	*5*	*6*
Edema/Swelling	*0*	*1*	*1*	*2*
Inflamed/Livid	*0*	*3*	*1*	*4*
Pain/Soreness[43]	*2*	*3*	*4*	*9*
Tongue	—	—	—	—
Dry/Parched[44]	*0*	*2*	*1*	*3*
Foul/Furred[45]	*0*	*3*	*3*	*6*
Red[46]	*3*	*5*	*0*	*8*
White[47]	*9*	*11*	*9*	*29*
Other[48]	*0*	*1*	*1*	*2*
9. PAIN	64	71	51	186
Acute/Severe/Violent[49]	*7*	*8*	*7*	*22*
Dull/General/Unspecified[50]	*3*	*2*	*5*	*10*
Head	*6*	*3*	*2*	*11*
Increased by	—	—	—	—
Cough/Inspiration[51]	*13*	*18*	*0*	*31*
Exertion/Motion	*0*	*0*	*3*	*3*
Pressure	*4*	*6*	*6*	*16*
Other[52]	*0*	*0*	*8*	*8*
Limbs, Lower[53]	*1*	*1*	*3*	*4*
Trunk, Back/Shoulders	*4*	*2*	*6*	*12*
Trunk, Chest/Thorax	*16*	*22*	*1*	*39*
Trunk, Other	*9*	*8*	*7*	*24*
Other[54]	*1*	*1*	*3*	*5*
10. NEUROLOGICAL	7	6	15	28
Confusion/Delirium	*2*	*3*	*2*	*7*
Muscular Weakness	*1*	*1*	*1*	*3*
Vision Anomalies[55]	*3*	*2*	*6*	*11*
Other[56]	*1*	*0*	*6*	*7*
11. RESPIRATORY, GENERAL	193	112	12	317
Cough	—	—	—	—
Distressing[57]	*28*	*19*	*1*	*48*
Disturbs Sleep	*2*	*0*	*0*	*2*
Evening/Night	*5*	*5*	*0*	*10*
Little[58]	*4*	*3*	*2*	*9*

Appendix D (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
11. RESPIRATORY, GENERAL, CONT.				
Cough, Cont.				
Longstanding	*0*	*1*	*0*	*1*
Morning	*3*	*0*	*0*	*3*
Other[59]	*8*	*5*	*0*	*13*
Unspecified	*14*	*2*	*2*	*18*
Dyspnea, All Types[60]	*13*	*12*	*1*	*26*
Expectoration	—	—	—	—
Abundant[61]	*15*	*4*	*0*	*19*
Bloody[62]	*9*	*5*	*0*	*14*
Dense[63]	*8*	*6*	*0*	*14*
Easy/Frothy[64]	*6*	*6*	*0*	*12*
Mucus	*18*	*13*	*1*	*32*
Opaque/White	*3*	*0*	*0*	*3*
Pus[65]	*17*	*3*	*1*	*21*
Scanty	*2*	*3*	*0*	*5*
Viscid	*3*	*7*	*0*	*10*
Yellow/Greenish	*12*	*0*	*0*	*12*
Other	*2*	*2*	*0*	*4*
Hemoptysis, All Types	*7*	*1*	*0*	*8*
Respiration Anomalies[66]	*14*	*15*	*4*	*33*
12. RESPIRATORY, AUSCULTATION AND PERCUSSION	168	69	17	254
Auscultation	—	—	—	—
Amphoric/Cavernous	*7*	*0*	*0*	*7*
Bronchial	*16*	*5*	*1*	*22*
Bronchophony	*10*	*1*	*0*	*11*
Cough, Tracheal	*0*	*1*	*0*	*1*
Crepitus[67]	—	—	—	—
Emphysematous	*0*	*2*	*0*	*2*
Pleuritic	*0*	*1*	*0*	*1*
Pneumonic	*9*	*0*	*0*	*9*
Tubercular	*8*	*0*	*0*	*8*
Subcrepitus/Other[68]	*15*	*13*	*1*	*29*

Appendix D (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
12. RESPIRATORY, AUSCULTATION AND PERCUSSION, CONT.				
Auscultation, Cont.				
Frottement (Friction)	*1*	*5*	*0*	*6*
Gurgling/Gargouillement	*6*	*0*	*1*	*7*
Mucus	*5*	*4*	*1*	*10*
Pectoriloquy	*14*	*0*	*1*	*15*
Puerile	*3*	*3*	*0*	*6*
Rales, Unspecified	*1*	*0*	*0*	*1*
Respiration, Faint/Obscure	*4*	*1*	*3*	*8*
Respiration, Loud/Raucous	*4*	*0*	*0*	*4*
Sibilus (Whistling)	*14*	*10*	*2*	*26*
Sonorous	*7*	*6*	*2*	*15*
Vocal Resonance	*14*	*3*	*2*	*19*
Percussion	—	—	—	—
Dull/Diminished Resonance	*28*	*12*	*3*	*43*
Hollow/Tympanitic	*2*	*2*	*0*	*4*
13. RESPIRATORY, CHEST FORM/SENSATION	29	7	0	36
Form	—	—	—	—
Arched	*3*	*0*	*0*	*3*
Contracted[69]	*6*	*1*	*0*	*7*
Expanded/Broad	*2*	*0*	*0*	*2*
Measurement, Sides Unequal	*3*	*2*	*0*	*5*
Sensation	—	—	—	—
Constriction[70]	*7*	*4*	*0*	*11*
Fullness/Stuffing	*3*	*0*	*0*	*3*
Gurgling, Inspiration[71]	*2*	*0*	*0*	*2*
Suffocation[72]	*3*	*0*	*0*	*3*
14. MISCELLANEOUS	11	22	24	57
Abdomen	—	—	—	—
Liver Anomalies[73]	*0*	*2*	*2*	*4*
Edema[74]	*2*	*5*	*8*	*15*
Sensation, Heat	*0*	*0*	*1*	*1*

Appendix D (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
14. MISCELLANEOUS, CONT.				
Bone and Joint, Pain[75]	*1*	*1*	*5*	*7*
Bone and Joint, Rheumatism[76]	*0*	*1*	*0*	*1*
Edema, Limbs, Lower	*8*	*7*	*4*	*19*
Edema, Other	*0*	*6*	*4*	*10*
TOTAL	865	531	354	1750

[1] Includes one patient whose countenance was described as "somewhat wild and maniacal" and one "sharp."

[2] Also includes exhaustion, fatigue, feeble, oppression/febrile oppression, strength loss, and weakness.

[3] Includes faintishness, giddiness and vertigo.

[4] Includes cheek bony or prominent, thin or wasted appearance, and weight loss.

[5] Includes visible blood vessels and yellow conjunctivae.

[6] This and the subsequent six "position" categories describe the position in which the patient reported feeling most comfortable.

[7] This individual was recorded as compelled to sit.

[8] Includes those patients who reported they were unable to lie on their unaffected side (i.e., the side free of chest pain).

[9] Includes decreased action or sound of heart.

[10] Includes increased action or sound of heart.

[11] Includes bellows murmur and its French term soufflet in the first or systolic heart sound.

[12] Includes dullness or prolongation in the second heart sound.

[13] Includes feeble, rapid, tumultuous or violent action of the heart, considerable impulse of the heart, and palpitations.

[14] Includes precordial weakness.

[15] Also includes pulses described as jerking or quick.

[16] Also includes pulses described as intermitting, small, soft, and thready.

[17] Also includes pulses described as firm, full, incompressible, and strong.

[18] Includes exudation, purulent/puriform discharge, and crusting.

[19] Includes hectic flush and floridity.

[20] Includes perspiration described as cold, occurring at intervals or in the morning, and preceded by heat, as well as unspecified perspiration.

[21] Also includes scaly skin.

[22] Includes moist, yellow/yellowish, and petechial skin.

[23] Also includes anogenital excoriations.

[24] Also includes scabs on the limbs.

[25] Also includes pustules, sloughy patches, and vesicles.

[26] Also includes unspecified scabs.

[27] Includes acid stomach, acid eructation, and heartburn.

[28] Includes appetites described as bad, defective, gone, impaired, lost, or poor.

[29] Also includes costive or slow bowels.

[30] Also includes irritated or loose bowels.

[31] Includes stool described as bloody, light colored, dark, grey, thin, watery, or scanty.

[32] Includes deranged digestion, dyspeptic ailments, hemorrhoids, or irritable stomach.

[33] Also includes general derangement of constitution.

[34] Also includes restlessness.

[35] Includes patients reporting alternations of hot and cold sensations.

[36] Also includes chills and rigors.

[37] Also includes overexertion and overheating.

[38] Includes addiction to/use of spirits, denial of primary syphilis, impure connection, and intemperance.

[39] Includes surgical removal of the placenta.

[40] Includes high excitation of mind and long confinement to house.

[41] Includes foul teeth and sore gums.

[42] Also includes dysphagia, feeling of choking, and feeling of obstruction to swallowing.

[43] Includes pain in the larynx and on deglutition.

[44] Also includes fissured tongues.

[45] Also includes loaded tongues.

[46] Also includes florid or brownish tongues.

[47] Also includes grey or pale tongues.

[48] Includes vesicles on tonsils and salivation.

[49] Also includes pain described as excessive, heavy, or sharp.

[50] Also includes pain described as constant, fixed, or gnawing.

[51] Includes pain impeding cough or respiration.

[52] Includes pain increased after eating and at certain times of day.

[53] Includes pain at the greater trochanter, groin, hip, loins, sciatic nerve, and sciatic notch.

[54] Includes pains obtuse, occasional, relieved by heat, slight, and wandering.

[55] Includes epiphora, light intolerance, pupils contracted/uneven, spectral illusions, and vision dim or impaired.

[56] Includes halting gait, impeded locomotion, incoherent speech, numbness, and tremor.

[57] Also includes coughs described as constant, frequent, hard, harsh, much, painful, severe, troublesome, urgent, or violent.

[58] Also includes coughs described as occasional, short, slight, or tickling.

[59] Includes coughs aggravated by pressure or respiration, croupy, dry, increased/increasing, paroxysms, stridulous, and tracheal.

[60] Also includes orthopnea.

[61] Also includes expectoration described as amounting to one quart in twelve hours, considerable, copious, frequent, increased, and profuse.

[62] Also includes expectoration described as brown tinged, rusty, or tinged (i.e., with blood).

[63] Also includes expectoration described as difficult, tenacious, thick, or tough.

[64] Also includes expectoration described as thin or watery.

[65] Also includes expectoration described as purulent or puriform.

[66] Includes accelerated, difficult, hurried, impaired, labored/laborious, noisy, oppressed, or rapid respiration.

[67] Crepitus refers to a crackling noise produced in the lung.

[68] Includes dryish, edematous, large, moist, slight, universal, and watery crepitus or subcrepitus.

[69] Also includes chests described as narrow or small.

[70] Also includes sensations of compression, tightness, or weight.

[71] Also includes a sensation of croaking on inspiration.

[72] Also includes a sensation of oppression.

[73] Includes liver enlargement, extended dullness on percussion, and unevenness.

[74] Also includes abdominal appearance of fullness, distension, enlargement, hardness, swelling or tumefaction, as well as tympanitic abdomen.

[75] Includes greater trochanter, hip, large joints, sciatic notch, and tibiae/tibial spines.

[76] Also includes joint stiffness.

APPENDIX E

SIGNS AND SYMPTOMS RECORDED IN SAMPLED CASES

FOR THE PATHOLOGICAL PERIOD II, 1841 – 1880,

AT GLASGOW ROYAL INFIRMARY

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
1. APPEARANCE AND COMPORTMENT	85	14	19	118
Anemic	*4*	*0*	*3*	*7*
Anxiety[1]	*1*	*0*	*0*	*1*
Complexion, Waxy	*0*	*1*	*1*	*2*
Complexion, Sallow/Yellow	*2*	*0*	*1*	*3*
Constitution, Phthisical	*1*	*0*	*1*	*2*
Countenance, Collapsed/Sunk[2]	*1*	*1*	*0*	*2*
Countenance, Tranquil	*1*	*0*	*0*	*1*
Debility[3]	*34*	*6*	*4*	*44*
Dizziness	*0*	*2*	*0*	*2*
Emaciation[4]	*34*	*2*	*4*	*40*
Eyes, Abnormal Appearance[5]	*1*	*0*	*3*	*4*
Languor[6]	*1*	*1*	*1*	*3*
Pallor[7]	*5*	*0*	*1*	*6*
Position, Head Raised[8]	*0*	*1*	*0*	*1*
2. CIRCULATORY	105	45	36	186
Auscultation	—	—	—	—
Heart, Action Decreased[9]	*6*	*4*	*3*	*13*
Heart, Action Increased[10]	*8*	*6*	*5*	*19*
Heart, 1st Sound Anomalies[11]	*3*	*2*	*4*	*9*
Heart, 2nd Sound Anomalies[12]	*1*	*3*	*4*	*8*
Heart	—	—	—	—
Beat Anomalies[13]	*2*	*1*	*2*	*5*
Oppression/Weakness[14]	*0*	*0*	*2*	*2*
Palpitation[15]	*2*	*4*	*3*	*9*
Percussion, Anomalies[16]	*3*	*1*	*1*	*5*
Pulse	—	—	—	—
≤ 74	*4*	*3*	*2*	*9*
75 – 84	*1*	*5*	*3*	*9*
85 – 94	*4*	*1*	*4*	*9*
95 – 104	*11*	*5*	*6*	*22*
105 – 114	*5*	*2*	*1*	*8*
≥ 115	*18*	*2*	*1*	*21*
Feeble/Weak[17]	*35*	*5*	*6*	*46*
Hard/Sharp[18]	*2*	*1*	*1*	*4*

Appendix E (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
3. DERMATOLOGICAL	53	16	23	92
Encrustation/Discharge[19]	*2*	*0*	*0*	*2*
Flushing/Redness	*2*	*6*	*4*	*12*
Perspiration	—	—	—	—
Frequent/Profuse[20]	*7*	*0*	*1*	*8*
Night	*22*	*4*	*1*	*27*
Other[21]	*4*	*2*	*5*	*11*
Rash	*0*	*1*	*3*	*4*
Skin	—	—	—	—
Cool[22]	*2*	*1*	*1*	*4*
Dry/Harsh	*3*	*1*	*2*	*6*
Warm/Hot[23]	*5*	*1*	*4*	*10*
Other[24]	*0*	*0*	*2*	*2*
Ulcer, Oral/Facial[25]	*6*	*0*	*0*	*6*
4. DIGESTIVE	72	34	39	145
Abdominal Fluctuation	*0*	*0*	*2*	*2*
Acidity[26]	*0*	*1*	*1*	*2*
Alkalinity[27]	*0*	*1*	*0*	*1*
Anorexia[28]	*31*	*11*	*14*	*56*
Appetite, Increased	*1*	*0*	*0*	*1*
Bowels, Constipation[29]	*14*	*13*	*13*	*40*
Bowels, Diarrhea[30]	*12*	*0*	*1*	*13*
Flatulence	*0*	*2*	*2*	*4*
Nausea/Retching/Vomiting[31]	*8*	*2*	*2*	*12*
Other Digestive[32]	*6*	*4*	*4*	*14*
5. GENERAL	20	7	10	37
General Health Impaired[33]	*5*	*0*	*2*	*7*
Hectic[34]	*5*	*0*	*0*	*5*
Sleep, Disturbed[35]	*2*	*3*	*5*	*10*
Temperature	—	—	—	—
Alternations	*1*	*0*	*0*	*1*
Fever/Heat[36]	*2*	*2*	*3*	*7*
Shivering	*1*	*0*	*0*	*1*
Thermometer	*1*	*1*	*0*	*2*
Thirst, Increased	*3*	*1*	*0*	*4*

Appendix E (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
6. GENITOURINARY	26	8	41	75
Amenorrhea[37]	*15*	*0*	*4*	*19*
Hysterical	*1*	*0*	*1*	*2*
Urinalysis	—	—	—	
Specific Gravity Reported[38]	*1*	*0*	*5*	*6*
Acid	*0*	*0*	*4*	*4*
Alkaline	*0*	*0*	*1*	*1*
Albuminous	*0*	*1*	*2*	*3*
Sugar Present	*1*	*0*	*0*	*1*
Urination, Incontinence	*0*	*2*	*1*	*3*
Urination, Frequent Urge	*0*	*0*	*1*	*1*
Urine	—	—	—	—
Difficult/Obstructed[39]	*1*	*2*	*2*	*5*
High Colored[40]	*2*	*1*	*2*	*5*
Muddy/Sediment[41]	*3*	*1*	*1*	*5*
Scanty[42]	*1*	*1*	*2*	*4*
Vagina, Discharge[43]	*0*	*0*	*4*	*4*
Vagina, Physical Exam	*1*	*0*	*6*	*7*
Other Genitourinary[44]	*1*	*1*	*5*	*7*
7. HISTORY, MEDICAL	108	43	60	211
Other Disorders	—	—	—	—
Cold	*10*	*4*	*4*	*18*
OB/GYN[45]	*5*	*3*	*5*	*13*
Pulmonary[46]	*3*	*2*	*2*	*7*
Strumous/Neck Swellings[47]	*2*	*0*	*1*	*3*
Venereal	*1*	*2*	*2*	*5*
Other	*3*	*6*	*9*	*18*
Seasonal Disorder, Fall/Winter	*1*	*5*	*3*	*9*
Symptoms	—	—	—	—
Debility[48]	*6*	*1*	*0*	*7*
Digestive[49]	*16*	*3*	*3*	*22*
Edema	*0*	*1*	*7*	*8*
Pain/Uneasiness, Trunk	*6*	*0*	*1*	*7*
Pain/Uneasiness, Other	*4*	*2*	*3*	*9*
Perspiration, Night	*2*	*0*	*0*	*2*
Pulmonary, Cough	*18*	*2*	*2*	*22*
Pulmonary, Dyspnea	*5*	*0*	*1*	*6*
Pulmonary, Expectoration	*13*	*5*	*4*	*22*
Pulmonary, Hemoptysis	*7*	*0*	*1*	*8*

Appendix E (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
7. HISTORY, MEDICAL, CONT.				
Symptoms, Cont.				
Shivering[50]	*2*	*6*	*3*	*11*
Weight Loss	*1*	*0*	*0*	*1*
Other[51]	*3*	*1*	*9*	*13*
8. HISTORY, PERSONAL	86	32	45	163
Duration	—	—	—	—
< 1 Month	*2*	*9*	*4*	*15*
1 – 4 Months	*17*	*8*	*8*	*33*
5 – 8 Months	*9*	*1*	*4*	*14*
≥ 9 Months	*13*	*3*	*8*	*24*
Months, Unspecified	*5*	*0*	*0*	*5*
Family History	—	—	—	—
Death, Childhood/Infancy	*5*	*2*	*2*	*9*
Death, Pulmonary	*6*	*0*	*1*	*7*
TB-Related	*7*	*1*	*2*	*10*
TB-Related Unknown[52]	*3*	*0*	*0*	*3*
Other	*10*	*1*	*7*	*18*
Habits	—	—	—	—
Abstaining[53]	*2*	*0*	*0*	*2*
Poor Diet[54]	*2*	*0*	*2*	*4*
Poorhouse	*0*	*2*	*0*	*2*
Precipitating Factors	—	—	—	—
Exposure to Elements[55]	*4*	*3*	*2*	*9*
Fatigue[56]	*1*	*0*	*2*	*3*
Immorality[57]	*0*	*2*	*0*	*2*
Injury	*0*	*0*	*1*	*1*
Other[58]	*0*	*0*	*2*	*2*
9. MOUTH AND THROAT	40	20	23	83
Difficulty Speaking/Hoarseness[59]	*2*	*0*	*0*	*2*
Difficulty Swallowing[60]	*1*	*1*	*3*	*5*
Gums, Red Line	*5*	*1*	*1*	*7*
Inflamed/Livid[61]	*0*	*2*	*0*	*2*
Pain/Soreness[62]	*2*	*0*	*2*	*4*

Appendix E (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
9. MOUTH AND THROAT, CONT.				
Tongue	—	—	—	—
Dry/Fissured	*6*	*0*	*1*	*7*
Foul/Furred[63]	*16*	*11*	*9*	*36*
Red[64]	*5*	*1*	*1*	*7*
White[65]	*2*	*2*	*4*	*8*
Other Oral[66]	*1*	*2*	*2*	*5*
10. PAIN	51	31	48	130
Acute/Severe/Violent[67]	*0*	*5*	*5*	*10*
Dull/General/Unspecified[68]	*4*	*2*	*7*	*13*
Head[69]	*1*	*1*	*6*	*8*
Increased by	—	—	—	—
Cough/Inspiration[70]	*2*	*2*	*0*	*4*
Exertion/Motion	*1*	*0*	*1*	*2*
Pressure	*5*	*1*	*1*	*7*
Other[71]	*2*	*0*	*5*	*7*
Limbs[72]	*3*	*0*	*5*	*8*
Trunk	—	—	—	—
Back/Shoulders	*7*	*0*	*4*	*11*
Chest/Thorax[73]	*15*	*14*	*3*	*32*
Other[74]	*10*	*5*	*10*	*25*
Other Pain[75]	*1*	*1*	*1*	*3*
11. NEUROLOGICAL	5	0	7	12
Confusion/Delirium[76]	*1*	*0*	*0*	*1*
Eye/Vision Anomalies[77]	*0*	*0*	*2*	*2*
Muscular Weakness[78]	*3*	*0*	*1*	*4*
Other Neurological[79]	*1*	*0*	*4*	*5*
12. RESPIRATORY, GENERAL	143	76	10	229
Cough	—	—	—	—
Distressing[80]	*29*	*13*	*1*	*43*
Disturbs Sleep	*1*	*0*	*0*	*1*
Little[81]	*2*	*1*	*1*	*4*
Longstanding	*1*	*0*	*0*	*1*
Morning	*0*	*1*	*0*	*1*
Unspecified	*14*	*8*	*1*	*23*
Other[82]	*1*	*1*	*0*	*2*

Appendix E (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
12. RESPIRATORY, GENERAL, CONT.				
Dyspnea, All Types	*14*	*12*	*4*	*30*
Expectoration	—	—	—	—
Abundant[83]	*11*	*2*	*0*	*13*
Bloody[84]	*6*	*6*	*0*	*12*
Dense[85]	*4*	*3*	*0*	*7*
Easy/Frothy[86]	*8*	*8*	*1*	*17*
Mucus	*3*	*4*	*1*	*8*
Opaque/White	*0*	*1*	*0*	*1*
Pus[87]	*15*	*0*	*0*	*15*
Scanty[88]	*3*	*0*	*0*	*3*
Viscid	*4*	*2*	*0*	*6*
Yellow/Greenish	*3*	*0*	*0*	*3*
Other	*6*	*5*	*0*	*11*
Hemoptysis, All Types	*3*	*0*	*0*	*3*
Phthisis Pulmonalis[89]	*5*	*0*	*0*	*5*
Respiration	—	—	—	—
Abdominal	*1*	*1*	*0*	*2*
Impaired[90]	*2*	*9*	*1*	*12*
Rapid	*2*	*3*	*0*	*5*
Rate, ≤ 29	*1*	*0*	*0*	*1*
Rate, 30 – 39	*2*	*1*	*0*	*3*
Rate, ≥ 40	*2*	*1*	*0*	*3*
13. RESPIRATORY, AUSCULTATION AND PERCUSSION	182	46	8	236
Auscultation	—	—	—	—
Amphoric/Cavernous	*2*	*0*	*0*	*2*
Bronchial/Puerile[91]	*13*	*2*	*1*	*16*
Bronchophony	*8*	*2*	*0*	*10*
Crepitus	—	—	—	—
Bronchitic	*3*	*2*	*1*	*6*
Pneumonic	*0*	*1*	*0*	*1*
Tubercular	*13*	*0*	*0*	*13*
Unspecified	*11*	*5*	*0*	*16*
Subcrepitus/Other[92]	*14*	*7*	*2*	*23*

Appendix E (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
13. RESPIRATORY, AUSCULTATION AND PERCUSSION, CONT.				
Auscultation, Cont.				
Frottement[93]	*2*	*1*	*0*	*3*
Gurgling/Gargouillement[94]	*5*	*0*	*0*	*5*
Metallic Tinkle	*0*	*1*	*0*	*1*
Mucus	*6*	*1*	*0*	*7*
Pectoriloquy	*8*	*0*	*0*	*8*
Respiration	—	—	—	—
Blowing	*3*	*0*	*0*	*3*
Faint/Obscure[95]	*6*	*3*	*0*	*9*
Loud/Raucous[96]	*11*	*9*	*2*	*22*
Prolonged	*3*	*1*	*0*	*4*
Tubular	*11*	*0*	*0*	*11*
Other[97]	*1*	*2*	*0*	*3*
Sibilus (Whistling)	*6*	*8*	*2*	*16*
Sonorous	*1*	*4*	*0*	*5*
Vocal Resonance[98]	*12*	*3*	*0*	*15*
Palpation, Vocal Fremitus[99]	*3*	*1*	*0*	*4*
Percussion	—	—	—	—
Cracked Pot	*3*	*0*	*0*	*3*
Dull/Diminished Resonance[100]	*35*	*12*	*2*	*49*
Hollow/Tympanitic[101]	*2*	*4*	*0*	*6*
14. RESPIRATORY, CHEST FORM/SENSATION	22	6	2	30
Expansion, Imperfect	*2*	*0*	*1*	*3*
Form	—	—	—	—
Contracted	*1*	*0*	*0*	*1*
Expanded/Broad	*8*	*0*	*0*	*8*
Flattening	*9*	*0*	*1*	*10*
Ill	*1*	*0*	*0*	*1*
Measurement, Sides Unequal	*1*	*0*	*0*	*1*
Sensation, Suffocation[102]	*0*	*6*	*0*	*6*

Appendix E (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
15. MISCELLANEOUS	25	2	39	66
Abdomen	—	—	—	—
Edema[103]	*4*	*1*	*10*	*15*
Liver Anomalies[104]	*2*	*0*	*0*	*2*
Percussion, Dullness[105]	*1*	*1*	*3*	*5*
Tumor	*0*	*0*	*3*	*3*
Bone and Joint	—	—	—	—
Pain	*0*	*0*	*4*	*4*
Rheumatism	*0*	*0*	*2*	*2*
Other[106]	*2*	*0*	*7*	*9*
Fingers, Clubbed/Incurved	*4*	*0*	*1*	*5*
Edema, Limbs, Lower[107]	*3*	*0*	*7*	*10*
Edema, Other[108]	*8*	*0*	*2*	*10*
Percussion, Dullness, Other[109]	*1*	*0*	*0*	*1*
TOTAL	1023	380	410	1650

[1] Includes patients described as having an anxious expression.

[2] Includes thin faces.

[3] Includes exhaustion, fatigue, weakness, loss of strength, and patients unfit for exertion.

[4] Includes cachectic, thin, wasted, and weight loss.

[5] Includes blanched/bloodless/pale conjunctiva as well as discharge from eyelid.

[6] Includes drowsiness, dullness, and heavy head.

[7] Includes patients with blanched or pale lips.

[8] Includes patients with semi-erect posture.

[9] Includes heart action described as feeble, indistinct, or weak.

[10] Includes heart action described as excited, hurried, more distinct than normal, quick, rapid, tumultuous, or violent.

[11] Includes first or systolic sounds described as concealing second sound, diffuse, loud, or accompanied by a murmur.

[12] Includes second sounds described as indistinct, prolonged, roughened, or accompanied by a murmur.

[13] Includes beats described as clicking, displaced apex beat, intermitting, interval between first and second sounds, preternaturally clear.

[14] Includes sensations of deadness or dullness in the cardiac region.

[15] Includes increased pulsation of the neck vessels.

[16] Includes displacement or expansion of the area of cardiac dullness.

[17] Includes pulses described as compressible, intermitting, small, soft, thready, or of uneven strength.

[18] Includes pulses described as firm, full, incompressible, strong, or wiry.

[19] Includes nasal discharge.

[20] Includes perspiration described as abundant, free, or much.

[21] Includes perspiration described as clammy or slight, as well as perspiration of the joints and damp or moist skin.

[22] Includes chilly skin and a cold sensation in the legs.

[23] Includes a burning sensation in the soles of the feet.

[24] Includes skin described as hard or brawny.

[25] Includes abraded surface, fissure, or itching scab on the face or mouth.

[26] Includes acid eructation, acid stomach, heartburn, and pyrosis.

[27] Includes alkaline eructation.

[28] Includes appetites described as bad, gone, impaired, indifferent, lost, or poor.

[29] Includes confined, costive, and irregular bowels.

[30] Includes loose and relaxed bowels.

[31] Also includes sickness.

[32] Includes anal prolapse/stricture, deranged digestion, dyspepsia, gurgling in iliac fossa, indigestion, inguinal hernia, irritable or weak stomach, or sensation of weight in epigastrium.

[33] Includes general health described as falling off, indifferent, lost, shattered, or weak.

[34] Includes hectic flush.

[35] Includes sleep disturbed by pain, much/occasional dreaming, restless, and uneasy.

[36] Includes feverish skin and sensation of heat.

[37] Includes one patient with irregular menses.

[38] All reported urine densities are within normal range.

[39] Includes painful urination and stoppage or suppression of urine.

[40] Includes amber colored urine.

[41] Includes precipitates described as flocculent, phosphatic, and uric acid.

[42] Includes decreased urine.

[43] Includes bleeding, menorrhagia, scanty lochia, and discharge described as chorous, fetid, and purulent.

[44] Includes genital pain/heat, menstrual pain, uterine prolapse, and uterine tumor.

[45] Includes amenorrhea, breastfeeding for two years, caring for sick child, difficult recovery from childbirth, flooding after childbirth, heavy medical washing after childbirth, menopause, menorrhagia, miscarriage with hemorrhage, pregnancy, and stillbirth.

[46] Includes inflammation of the side.

[47] Includes strumous eyes.

[48] Includes delicacy, loss of strength, and weakness.

[49] Includes appetite loss, bad appetite, dyspepsia, nausea, vomit, and stomach described as irritable, sour, or weak.

[50] Includes chills and rigors.

[51] Includes bad/weak health, delirium, dizziness, eczema, epistaxis, hair loss, hemorrhoids, numbness in limbs, palpitation, perspiration, sensation of peas running up veins, and skin vesicles.

[52] Includes patients noted as unaware of phthisis predisposition.

[53] Includes patients and spouses noted to be abstaining.

[54] Includes diets chiefly tea or bread and tea, insufficient diet, and patients reporting they were poorly taken care of.

[55] Includes clothes frequently wet at employment, work carrying wet clothes on back.

[56] Includes work carrying children as well as extensive standing and walking in course of occupation.

[57] Includes irregular habits and taking stimulants.

[58] Includes origin of disease at birth and patients described as never healthy.

[59] Includes loss of voice and husky voice.

[60] Includes dysphagia, globus hystericus, and sensation of choking.

[61] Includes swelling pharynx/throat.

[62] Includes pyloric pain.

[63] Includes coated, loaded, and rough.

[64] Includes brownish, florid, and mottled.

[65] Includes grey and pale.

[66] Includes bad breath, bad taste in mouth, dry lips/mouth, and salivation from medical treatment.

[67] Includes excessive, heavy, lancinating, sharp, shooting, and stabbing.

[68] Includes aching, burning, constant, fixed, and gnawing.

[69] Includes facial pain.

[70] Includes pain hindering respiration.

[71] Includes pain on eating, increased by rest/eased by motion, and increased at night.

[72] Includes groin.

[73] Includes cardialgia.

[74] Includes abdomen, epigastric, gastric, hypogastric, iliac fossa, liver, side, and stomach.

[75] Includes pain all through body, in neck, and impairing locomotion.

[76] Includes patients described as unreasonable.

[77] Includes uneven pupils.

[78] Includes back weakness, locomotion impaired by weakness, and weakened grip.

[79] Includes difficult locomotion, involuntary motion, nervous symptoms, and numbness.

[80] Includes aggravated, annoying, bad, constant, frequent, good deal, hard, harassing, harsh, heavy, increased, much, painful, pretty constant, severe, troublesome, urgent, vehement, and violent.

[81] Includes occasional, short, slight, some, and tickling.

[82] Includes coughs described as dry or increased by severe wetting.

[83] Includes copious, excessive, frequent, and increased.

[84] Includes blood-streaked, blood-tinged, brownish, and rusty.

[85] Includes difficult, tenacious, thick, and tough.

[86] Includes expectoration described as pretty free, thin, or watery.

[87] Includes muco-purulent.

[88] Includes little, occasional, and slight.

[89] Includes patients described as having all ordinary/usual symptoms of, symptoms of, or third stage of phthisis.

[90] Includes difficult, embarrassed by exertion, laborious, noisy, and short.

[91] These describe normal breath sounds.

[92] Includes crepitus/rales described as coarse, cooing, creaking, distinct, dry, fine, little, moist, occasional, small, snoring, wheezing, or whistling.

[93] Includes friction and friction-like.

[94] Includes bubbling.

[95] Includes absent, feeble, and gone.

[96] Includes respiration described as coarse, harsh, noisy, rough, or wheezing.

[97] Includes irregular and asperity of respiration.

[98] Includes increased vocal resonance and aggravated voice sounds.

[99] Includes increased vocal fremitus.

[100] Includes flat, wooden, and varied.

[101] Includes resonant.

[102] Includes sensations of constriction, no air entering lung, oppression, stifling, or weight.

[103] Includes distension, hardness, swelling, tumefaction, and tympanitic abdomen.

[104] Includes enlargement.

[105] Includes dullness anomalies at the hypochondrium or liver.

[106] Includes knock-knees, joint weakness, Pott's curvature of spine, prominence on back, rachitic curves, swelling, and wrist redness.

[107] Includes fluid knee.

[108] Includes edema or swelling after eating and of the eyelid/face/glands, mass in groin, strumous glands, and strumous testicle.

[109] Includes dullness at an internal mass.

APPENDIX F

SIGNS AND SYMPTOMS RECORDED IN SAMPLED CASES FOR THE PATHOLOGICAL PERIOD II, 1841 – 1880, AT THE ROYAL INFIRMARY OF EDINBURGH

Signs and Symptoms	Diagnosis Groups			TOTAL
	TB-Related	Non-TB Pulmonary	Other	
1. APPEARANCE AND COMPORTMENT	432	136	175	743
Anemic	*8*	*3*	*1*	*12*
Anxiety[1]	*17*	*13*	*16*	*46*
Appearance, Healthy[2]	*18*	*8*	*8*	*34*
Appearance, Unhealthy[3]	*2*	*1*	*4*	*7*
Constitution	—	—	—	—
Delicate/Weak[4]	*6*	*1*	*6*	*13*
Phthisical	*3*	*0*	*1*	*4*
Other[5]	*8*	*1*	*3*	*12*
Complexion	—	—	—	—
Dark[6]	*28*	*6*	*4*	*38*
Light[7]	*10*	*0*	*9*	*19*
Sallow/Yellow[8]	*6*	*1*	*12*	*19*
Waxy	*1*	*0*	*1*	*2*
Countenance	—	—	—	—
Collapsed/Sunk[9]	*3*	*1*	*4*	*8*
Suffering[10]	*5*	*3*	*4*	*12*
Tranquil[11]	*5*	*2*	*3*	*9*
Debility[12]	*107*	*26*	*26*	*159*
Dizziness[13]	*14*	*8*	*9*	*31*
Emaciation[14]	*110*	*20*	*21*	*151*
Eyes, Abnormal Appearance[15]	*14*	*7*	*10*	*31*
Languor[16]	*7*	*1*	*8*	*16*
Pallor[17]	*36*	*11*	*12*	*59*
Position	—	—	—	—
Back[18]	*3*	*6*	*1*	*10*
Difficulty, Back	*0*	*0*	*1*	*1*
Difficulty, Left Side	*2*	*0*	*0*	*2*
Difficulty, Right Side	*0*	*1*	*0*	*1*
Head Raised[19]	*4*	*1*	*1*	*6*
Left Side	*2*	*1*	*1*	*4*
Right Side	*3*	*0*	*1*	*4*
Unaffected Side	*3*	*5*	*3*	*11*
Other Appearance[20]	*7*	*9*	*5*	*22*

Appendix F (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
2. CIRCULATORY	461	223	254	938
Auscultation	—	—	—	—
Heart, Action Decreased[21]	*23*	*17*	*6*	*46*
Heart, Action Increased[22]	*12*	*1*	*3*	*16*
Heart, 1st Sound Anomalies[23]	*16*	*9*	*20*	*45*
Heart, 2nd Sound Anomalies[24]	*3*	*3*	*4*	*10*
Heart/Blood Vessels, Other[25]	*3*	*3*	*10*	*16*
Beat/Impulse	—	—	—	—
Impulse Decreased[26]	*22*	*10*	*12*	*44*
Impulse Increased[27]	*5*	*4*	*11*	*20*
Other Beat Anomalies[28]	*13*	*8*	*13*	*34*
Heart, Palpitation[29]	*22*	*16*	*17*	*55*
Percussion, Anomalies[30]	*6*	*5*	*7*	*18*
Pulse	—	—	—	—
≤ 74[31]	*38*	*17*	*16*	*71*
75 – 84	*34*	*27*	*24*	*85*
85 – 94	*16*	*9*	*14*	*39*
95 – 104[32]	*31*	*17*	*16*	*64*
105 – 114	*15*	*4*	*3*	*22*
≥ 115	*28*	*6*	*7*	*41*
Bounding[33]	*10*	*7*	*10*	*27*
Feeble/Weak[34]	*135*	*49*	*63*	*247*
Hard/Sharp[35]	*21*	*7*	*15*	*43*
Other Pulse Anomalies[36]	*5*	*4*	*9*	*18*
Other Circulatory[37]	*3*	*0*	*3*	*6*
3. DERMATOLOGICAL	323	105	196	624
Encrustation/Discharge[38]	*12*	*1*	*8*	*21*
Flushing/Redness[39]	*21*	*17*	*21*	*59*
Perspiration	—	—	—	—
Frequent/Profuse[40]	*58*	*16*	*12*	*86*
Night	*69*	*9*	*7*	*85*
Other[41]	*42*	*17*	*15*	*74*
Rash[42]	*26*	*7*	*74*	*107*
Node/Mass[43]	*3*	*3*	*11*	*17*
Skin	—	—	—	—
Cool[44]	*4*	*2*	*1*	*7*
Dry/Harsh[45]	*26*	*14*	*21*	*61*
Warm/Hot[46]	*27*	*17*	*21*	*65*
Other[47]	*16*	*2*	*4*	*22*

Appendix F (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
3. DERMATOLOGIAL, CONT.				
Ulcer	—	—	—	—
Genital	*1*	*0*	*0*	*1*
Limbs	*0*	*0*	*1*	*1*
Neck/Chest[48]	*1*	*0*	*0*	*1*
Oral/Facial[49]	*8*	*0*	*0*	*8*
Lupus/Scrofulous	*6*	*0*	*0*	*6*
Unspecified/Other	*3*	*0*	*0*	*3*
4. DIGESTIVE	380	120	234	734
Abdominal Fluctuation	*0*	*0*	*3*	*3*
Acidity[50]	*6*	*1*	*7*	*14*
Anorexia[51]	*135*	*49*	*59*	*243*
Appetite, Increased[52]	*4*	*1*	*3*	*8*
Bowels	—	—	—	—
Constipation[53]	*59*	*26*	*27*	*41*
Diarrhea[54]	*36*	*7*	*14*	*57*
Gripes/Tenesmus[55]	*9*	*1*	*9*	*19*
Stool Anomalies[56]	*14*	*0*	*27*	*41*
Eructation, Non-Acid	*2*	*0*	*1*	*3*
Flatulence	*2*	*2*	*4*	*8*
Nausea/Retching/Vomiting[57]	*86*	*28*	*44*	*158*
Other Digestive[58]	*27*	*5*	*23*	*55*
5. GENERAL	188	86	98	372
General Health Impaired[59]	*4*	*0*	*0*	*4*
Sleep, Disturbed[60]	*58*	*21*	*30*	*109*
Hectic[61]	*6*	*0*	*0*	*6*
Temperature	—	—	—	—
Alternations	*6*	*3*	*6*	*15*
Fever/Heat[62]	*15*	*5*	*8*	*28*
Shivering[63]	*41*	*22*	*16*	*79*
Thermometer[64]	*8*	*5*	*3*	*16*
Thirst, Increased	*50*	*30*	*35*	*115*

Appendix F (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
6. GENITOURINARY	472	231	317	1020
Amenorrhea[65]	*38*	*8*	*13*	*59*
Hysterical	*0*	*1*	*0*	*1*
Urinalysis	—	—	—	—
Acid	*40*	*19*	*21*	*80*
Albuminous	*7*	*4*	*6*	*17*
Alkaline	*4*	*0*	*1*	*5*
Chemical Reaction[66]	*15*	*16*	*13*	*44*
Chlorides, Abundant	*26*	*9*	*7*	*42*
Components, Diminished[67]	*5*	*9*	*3*	*17*
Components, Other[68]	*38*	*29*	*26*	*93*
Deposit, Biological[69]	*38*	*9*	*33*	*80*
Deposit, Other[70]	*34*	*25*	*30*	*89*
Specific Gravity, Normal	*104*	*49*	*56*	*209*
Specific Gravity, Abnormal[71]	*1*	*1*	*1*	*3*
Sugar Present	*0*	*0*	*2*	*2*
Urine Color	—	—	—	—
Dark[72]	*33*	*15*	*23*	*71*
Light[73]	*33*	*6*	*15*	*54*
Other[74]	*4*	*5*	*4*	*13*
Urine, Difficult/Obstructed[75]	*10*	*9*	*18*	*37*
Urine, Quantity, Decreased[76]	*11*	*7*	*18*	*36*
Urine, Quantity, Increased[77]	*6*	*1*	*4*	*11*
Urination, Frequent Urge[78]	*2*	*3*	*4*	*9*
Urination, Incontinence	*1*	*0*	*3*	*4*
Vagina, Physical Exam	*0*	*0*	*2*	*2*
Vagina, Discharge[79]	*16*	*4*	*4*	*24*
Other Genitourinary[80]	*6*	*2*	*10*	*18*
7. HISTORY, MEDICAL	457	203	200	860
Other Disorders	—	—	—	—
Cold	*55*	*20*	*10*	*85*
OB/GYN[81]	*25*	*7*	*15*	*47*
Pulmonary[82]	*35*	*18*	*8*	*61*
Strumous/Neck Swellings[83]	*7*	*0*	*1*	*8*
Venereal[84]	*26*	*4*	*10*	*40*
Other[85]	*65*	*35*	*58*	*158*
Seasonal Disorder, Fall/Winter[86]	*9*	*8*	*0*	*17*

Appendix F (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
7. HISTORY, MEDICAL, CONT.				
Symptoms	—	—	—	—
Debility[87]	*12*	*6*	*5*	*23*
Digestive[88]	*33*	*17*	*18*	*68*
Edema	*6*	*7*	*6*	*19*
Pulmonary, Cough	*21*	*9*	*6*	*36*
Pulmonary, Dyspnea	*6*	*2*	*1*	*9*
Pulmonary, Expectoration	*8*	*4*	*2*	*14*
Pulmonary, Hemoptysis[89]	*34*	*10*	*2*	*46*
Pain/Uneasiness, Trunk	*15*	*9*	*6*	*30*
Pain/Uneasiness, Other	*10*	*9*	*11*	*30*
Perspiration, Night	*9*	*2*	*0*	*11*
Shivering[90]	*20*	*16*	*17*	*53*
Weight Loss	*3*	*0*	*0*	*3*
Other[91]	*33*	*16*	*17*	*66*
Treatment, Cod Liver Oil	*17*	*2*	*0*	*19*
Treatment, Other[92]	*8*	*2*	*7*	*17*
8. HISTORY, PERSONAL	506	199	210	915
Disease Duration	—	—	—	—
< 1 Month	*12*	*39*	*43*	*94*
1 – 4 Months	*42*	*18*	*19*	*79*
5 – 8 Months	*31*	*6*	*12*	*49*
≥ 9 Months	*89*	*19*	*20*	*128*
Months, Unspecified	*1*	*0*	*0*	*1*
Family History	—	—	—	—
Death, Childhood/Infancy	*19*	*5*	*1*	*25*
Death, Pulmonary	*12*	*4*	*0*	*16*
TB-Related[93]	*23*	*3*	*2*	*28*
TB-Related Unknown[94]	*16*	*6*	*6*	*28*
Other[95]	*27*	*7*	*13*	*47*
Habits, Diet, Poor[96]	*57*	*10*	*15*	*82*
Habits, Diet, Other[97]	*11*	*0*	*2*	*13*
Habits, Temperate[98]	*7*	*8*	*8*	*23*
Precipitating Factors	—	—	—	—
Exposure to Elements[99]	*86*	*47*	*34*	*167*
Fatigue[100]	*21*	*6*	*9*	*36*
Immorality[101]	*36*	*13*	*13*	*62*
Injury	*11*	*5*	*8*	*24*
Other[102]	*5*	*3*	*5*	*13*

Appendix F (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
9. MOUTH AND THROAT	369	177	211	757
Dental/Alveolar Disease[103]	*6*	*6*	*14*	*26*
Difficulty Speaking/Hoarseness[104]	*30*	*9*	*7*	*46*
Difficulty Swallowing[105]	*12*	*1*	*12*	*25*
Gums, Red Line	*2*	*0*	*0*	*2*
Livid/Livid[106]	*18*	*4*	*11*	*33*
Edema/Enlargement[107]	*11*	*5*	*10*	*26*
Pain/Soreness	*13*	*2*	*10*	*25*
Tongue	—	—	—	—
Dry/Fissured[108]	*23*	*16*	*15*	*54*
Foul/Furred[109]	*100*	*61*	*48*	*209*
Red[110]	*29*	*20*	*20*	*69*
White[111]	*67*	*26*	*29*	*122*
Other Tongue[112]	*12*	*6*	*8*	*26*
Other Oral[113]	*46*	*21*	*27*	*94*
10. PAIN	425	250	291	966
Acute/Severe/Violent[114]	*29*	*20*	*35*	*84*
Dull/General/Unspecified[115]	*25*	*14*	*16*	*55*
Head[116]	*61*	*34*	*43*	*138*
Increased by	—	—	—	—
Cough/Inspiration[117]	*31*	*29*	*10*	*70*
Exertion/Motion[118]	*8*	*2*	*7*	*17*
Pressure[119]	*32*	*15*	*29*	*76*
Other[120]	*21*	*2*	*10*	*33*
Limbs[121]	*14*	*9*	*20*	*43*
Trunk, Back/Shoulders[122]	*32*	*17*	*30*	*79*
Trunk, Chest/Thorax[123]	*79*	*56*	*27*	*162*
Trunk, Other[124]	*69*	*40*	*45*	*154*
Other Pain[125]	*24*	*12*	*19*	*55*
11. NEUROLOGICAL	90	37	67	194
Confusion/Delirium[126]	*7*	*6*	*7*	*20*
Ear/Hearing Anomalies[127]	*23*	*11*	*18*	*52*
Eye/Vision Anomalies[128]	*32*	*8*	*20*	*60*
Muscular Weakness[129]	*13*	*7*	*9*	*29*
Other Neurological[130]	*15*	*5*	*13*	*33*

Appendix F (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
12. RESPIRATORY, GENERAL	913	408	152	1473
Cough	—	—	—	—
Distressing[131]	*153*	*59*	*12*	*224*
Disturbs Sleep	*17*	*4*	*2*	*23*
Little[132]	*26*	*16*	*31*	*73*
Longstanding	*24*	*11*	*2*	*37*
Morning	*19*	*1*	*3*	*23*
Night	*29*	*10*	*2*	*41*
Unspecified	*47*	*18*	*2*	*67*
Other[133]	*15*	*13*	*6*	*34*
Dyspnea, All Types	*65*	*57*	*20*	*142*
Expectoration	—	—	—	—
Abundant[134]	*70*	*29*	*6*	*105*
Bloody[135]	*29*	*22*	*3*	*54*
Dense[136]	*36*	*24*	*3*	*63*
Easy/Frothy[137]	*51*	*35*	*9*	*95*
Mucus[138]	*24*	*20*	*9*	*53*
Opaque/White	*9*	*3*	*0*	*12*
Pus[139]	*87*	*23*	*5*	*115*
Scanty[140]	*18*	*13*	*11*	*42*
Viscid	*9*	*8*	*3*	*20*
Yellow/Greenish[141]	*29*	*11*	*3*	*43*
Other[142]	*47*	*13*	*4*	*64*
Hemoptysis, All Types	*38*	*2*	*3*	*43*
Phthisis Pulmonalis	*1*	*0*	*0*	*1*
Respiration	—	—	—	—
Abdominal[143]	*6*	*4*	*0*	*10*
Impaired[144]	*12*	*16*	*3*	*31*
Rapid[145]	*15*	*8*	*5*	*28*
Other[146]	*3*	*2*	*0*	*5*
Rate, ≤ 29	*13*	*10*	*1*	*24*
Rate, 30 – 39	*12*	*5*	*2*	*19*
Rate, ≥ 40	*9*	*4*	*2*	*15*

Appendix F (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
13. RESPIRATORY, AUSCULTATION AND PERCUSSION	1131	261	100	1492
Auscultation	—	—	—	—
Amphoric/Cavernous[147]	*29*	*0*	*0*	*29*
Bronchial/Puerile[148]	*35*	*11*	*1*	*47*
Bronchophony/Aegophony	*16*	*4*	*0*	*20*
Cough[149]	*3*	*3*	*0*	*6*
Crepitus	—	—	—	—
Coarse/Loud[150]	*30*	*8*	*0*	*38*
Cooing/Musical	*5*	*3*	*0*	*8*
Slight/Small[151]	*14*	*8*	*8*	*30*
Unspecified	*55*	*20*	*0*	*75*
Subcrepitus/Other[152]	*19*	*6*	*0*	*25*
Friction/Frottement	*21*	*15*	*1*	*37*
Gurgling/Gargouillement[153]	*38*	*2*	*0*	*40*
Metallic Tinkle[154]	*2*	*0*	*0*	*2*
Mucus	*70*	*25*	*5*	*100*
Pectoriloquy	*29*	*3*	*0*	*32*
Rales, Unspecified	*3*	*8*	*0*	*11*
Respiration	—	—	—	—
Blowing[155]	*18*	*3*	*0*	*21*
Faint/Obscure[156]	*45*	*23*	*8*	*76*
Loud/Raucous[157]	*122*	*39*	*19*	*180*
Prolonged	*105*	*26*	*12*	*143*
Tubular	*25*	*13*	*0*	*38*
Other[158]	*15*	*4*	*3*	*22*
Sibilus	*45*	*48*	*22*	*115*
Sonorous	*22*	*30*	*5*	*57*
Vocal Resonance, Increased[159]	*119*	*27*	*5*	*151*
Vocal Resonance, Other[160]	*6*	*5*	*0*	*11*
Palpation, Vocal Fremitus	—	—	—	—
Decreased[161]	*4*	*5*	*0*	*9*
Increased[162]	*13*	*5*	*0*	*18*
Percussion	—	—	—	—
Cracked Pot[163]	*30*	*3*	*1*	*34*
Dull/Diminished Resonance[164]	*174*	*47*	*19*	*240*
Hollow/Tympanitic[165]	*19*	*12*	*0*	*31*

Appendix F (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
14. RESPIRATORY, CHEST FORM/SENSATION	120	44	10	174
Expansion, Imperfect[166]	*46*	*16*	*1*	*63*
Form	—	—	—	—
Arched[167]	*5*	*10*	*3*	*18*
Contracted[168]	*2*	*1*	*1*	*4*
Expanded/Broad[169]	*3*	*3*	*2*	*8*
Flattening[170]	*51*	*5*	*0*	*56*
Other[171]	*2*	*1*	*2*	*5*
Measurement, Sides Unequal	*5*	*0*	*0*	*5*
Sensation	—	—	—	—
Constriction/Tightness	*3*	*3*	*0*	*6*
Fullness/Stuffing	*1*	*1*	*0*	*2*
Gurgling on Inspiration[172]	*0*	*1*	*0*	*1*
Suffocation[173]	*2*	*3*	*1*	*6*
15. MISCELLANEOUS	82	41	131	254
Abdomen	—	—	—	—
Edema[174]	*15*	*10*	*25*	*50*
Liver Anomalies[175]	*1*	*0*	*3*	*4*
Percussion, Dullness[176]	*12*	*4*	*8*	*24*
Tumor	*2*	*1*	*12*	*15*
Bone and Joint	—	—	—	—
Pain	*6*	*1*	*16*	*23*
Rheumatism	*0*	*1*	*2*	*3*
Other[177]	*16*	*1*	*6*	*23*
Fingers/Nails, Anomalies[178]	*5*	*0*	*3*	*8*
Edema, Limbs, Lower	*14*	*12*	*26*	*52*
Edema, Other[179]	*11*	*11*	*30*	*52*
TOTAL	6349	2521	2646	10601

[1] Includes dread, and patients described as agitated, distressed, excited, fretful, impatient, irritable, nervous, peevish, and sharp.

[2] Includes patients described as fairly/well developed/nourished, healthy, muscular, obese/plethoric, of full habit, robust, strong, or well/strongly built/made.

[3] Includes patients described has having general malaise or uneasiness, and sickly.

[4] Includes patients delicate looking, not robust, or of delicate complexion.

[5] Includes biliousness, as well as bilious, leuco-phlegmatic, lymphatic, neurovascular, sanguine, and vascular diathesis/temperament.

[6] Includes black (hair), bronzed, dusky, and olive.

[7] Includes clear, fair, red (hair), and transparent.

[8] Includes icteric tint and jaundiced.

[9] Includes cadaverous face, haggard countenance, sunken eyes, and thin face.

[10] Includes patients described as crying on interrogation, depressed, desponding, querulous, and worn.

[11] Includes calm, free from pain, not nervous, quiet, and soft face.

[12] Includes patients described as exhausted, fatigued, feeble, prostrated, weak, or worn out, as well as having lost power/strength or describing a sensation of heaviness.

[13] Includes faintishness, giddiness, light-headedness, and vertigo.

[14] Includes disappearance of subcutaneous fat and flesh/weight loss, as well as patients described as cachectic, imperfectly/poorly nourished, of spare build/habit, or thin.

[15] Includes bluish/pale conjunctivae, brilliant/flashing eyes, congested/edematous/closed eyelids, congested/suffused eyes, dark circles under eyes, discharge of mucus/water, large eyes, increased pigment in eyelids, long eyelashes, and yellow/bile-tinged conjunctivae.

[16] Includes drowsiness, dullness, eyes closed, heavy head/eyes, inclined to dose, and sleepiness.

[17] Includes patients described as pale-lipped, pasty, and white-faced.

[18] Includes patients unable to sit in bed.

[19] Includes patients unable to lie due to difficulty breathing/expectorating, as well as semirecumbant and sitting.

[20] Includes appearance intelligent, interesting, vacant, or young for age; asymmetrical face; intelligent but not forthcoming; limited intelligence; loquacious; stunted/short stature; tattooed; and very hairy thorax and abdomen.

[21] Includes heart sounds described as diffused, distant, faint, feeble, inaudible, indistinct, intermittent, masked (i.e., by respiratory sounds), muffled, obscured, wanting tone, or weak.

[22] Includes heart sounds described as abnormally (i.e., more broadly than usual) transmitted, frequent, hurried, loud, palpitating, rapid, or tumultuous.

[23] Includes first or systolic sounds described as harsh, loud, prolonged, rough, rasping, sharp, short, weak, or accompanied by a murmur or bruit.

[24] Includes second sounds described as accentuated, flapping, prolonged, weak, or accompanied by a murmur.

[25] Includes venous murmur at jugular vein, both sounds harsh, sounds unequal, and murmurs accompanying both sounds, between sounds, described as anemic, or unspecified as to which sound they accompany.

[26] Includes apex beat, impulse, or action of heart described as diffused, feeble, imperceptible, intermittent, interrupted, slow, or weak.

[27] Includes apex beat, impulse, or action of heart described as abrupt, excited, heaving, increased, intensified, jerking, jogging, quick, rapid, strong, tumultuous, or visible.

[28] Includes apex beat, impulse, or action of heart described as displaced or irregular.

[29] Includes increased pulsation of the neck vessels.

[30] Includes patients for whom the extent of cardiac dullness was reported as expanded or with measurements, as well as reported precordial dullness.

[31] Includes one TB-related patient for whom the pulse was recorded as less than 80 per minute.

[32] Includes two TB-related patients for whom the pulse was recorded as greater than 100 per minute.

[33] Includes pulses described as frequent, jerking, quick, or rapid.

[34] Includes pulses described as compressible, intermitting, small, soft, or thready.

[35] Includes pulses described as firm, forcible, full, incompressible, strong, visible, or wiry.

[36] Includes pulses described as double, excitable, irregular, slow, unequal between sides, and wavy or undulating.

[37] Includes congested/prominent/varicose veins, thrilling of heart impulse in precordial region, and twisted radial arteries.

[38] Includes discharge from the breast, ear, mouth, nose (coryza, epistaxis), palate, prepuce, or urethra.

[39] Includes congestion, earthy discoloration, ecchymosis, floridity, inflammation, injection, and ruddiness.

[40] Includes perspiration described as considerable, constant, copious, covering, good/great deal, often, severe, and affecting the whole body.

[41] Includes unspecified perspiration, as well as that accompanying cough, exertion, shivering, sleep, or waking; described as acid, clammy, cold, gentle, hot, not much, occasional, occurring in the morning, of a sour/rheumatic odor, rare, slight; or occurring on the palms, feet, or forehead; and skin described as damp, moist, or boggy.

[42] Includes acne, blisters, claret-colored/dark pigmentous/roseolous spots, eruption, itching, knots, lentigo, papules, pustules, scabs, scales, and typhus mottling.

[43] Includes boil, knotty mass, tumor, vascular nevi, and wart.

[44] Includes chilly skin.

[45] Includes desquamated and rough skin.

[46] Includes pungent skin.

[47] Includes abnormal odors, bruise, dirty, face full/pitted, flabby, flea bites, hemorrhage, indurated, lice, little hair (scalp), palm mottled white, raised brown line, scarring, sensitive to touch/cold, and tender.

[48] Includes sternal abscess.

[49] Includes destroyed nasal alae and perforated septum nasi.

[50] Includes acid eructation, acid stomach, heartburn, and pyrosis.

[51] Includes all descriptions of reduced appetite.

[52] Includes cravings for specific food items (i.e., raw meat and salt, both TB-related cases) and ravenous.

[53] Includes bowels described as bound, confined, costive, irregular, not moved in two or more days, or torpid.

[54] Includes bowels described as loose, open multiple times daily, or relaxed, as well as semifluid or thin stool.

[55] Includes bowel pain, tormina, and a sensation of bearing down in abdomen.

[56] Includes passing tapeworm segments.

[57] Includes retching, sickness, uneasy stomach, and vomiting.

[58] Includes abdomen lax/soft/flaccid/doughy/corrugated, abdominal cramping/pain, auscultatory findings of abdominal gurgling, borborygni, bowel prolapse, colon enlarged, disagreeable sensations (i.e., oppression, pain, tightness, tumidity, weight) after eating, dyspepsia, fistula in ano, hiccough, indigestion, irritable/weak stomach, piles, sensation of heat/distension in bowels, sensation of weight/ball/biting in abdomen, and stools passed involuntarily

[59] Includes general health described as declined or reduced, as well as feeling out of tone.

[60] Includes dreaming, frequent starting on falling asleep, restlessness, sleep prevented by discomfort, and wandering at night.

[61] Includes hectic flush.

[62] Includes febrile symptoms, feverish, heat, and sensation of heat.

[63] Includes sensations of cold or chilliness.

[64] All recorded thermometer readings were above normal, ranging from 99°F to 107°F.

[65] Includes irregular menses, late period, menopausal, and seven-week menstrual cycle.

[66] Includes all cases in which a reaction (e.g., appearance of a coagulum or precipitate) occurred following exposure of the urine to heat or nitrogen solutions such as nitric acid.

[67] Includes chlorides, phosphates, and oxalates.

[68] Includes the presence of bile, cholesterine, indican, lithate, oxalates, phosphates, urates, or uric acid.

[69] Includes deposits of blood/white/rheumatic corpuscles, epithelium, granular matter, moving bodies, mucus, polymorphic cells, pus, tube casts, or vibriones, as well as bloody or the appearance of blood in urine.

[70] Includes urine described as muddy, opalescent, opaque, ropy, or smoky, as well as deposits described by their color or as flakes, fleecy, or flocculent.

[71] All abnormal densities are higher than the normal range.

[72] Includes urine described as amber, brown, dark, deep, gamboge, high-colored, or sherry, as well as dark, deep, or marked shades of amber or straw and urine that stains bedclothes.

[73] Includes urine described as almond, clear, light, pale, whey-like, and light or pale shades of amber, straw, or yellow.

[74] Includes urine described as black, olive, orange, pink, porter, or red.

[75] Includes burning, dysuria, painful, slow/not full stream, stricture, suppressed, and unable to completely empty bladder.

[76] Includes diminished, infrequent, reduced, scanty, scarce, and small quantity.

[77] Includes abundant, considerable, copious, and unusual quantity.

[78] Includes irritable bladder.

[79] Includes leucorrhea, light-colored menses, menorrhagia, or scanty menses, as well as discharge described as bloody, white, or yellow.

[80] Includes fetid urine, genital heat/pain, menstrual pain, penile/testicular anomalies and disease, sensation of menstruation coming on, and thick urine.

[81] Includes amenorrhea, breast abscess while nursing, chloroform during labor, child born with cord around neck, childbirth by instruments, considerable lochia after childbirth, early loss of milk, hysteric fits, irregular menses, lactation, leucorrhea, long confinement, menorrhagia, menstruation, miscarriage, origin of disease coincident with menses due, pregnant, previous medical treatment for uterine disease, recent childbirth, severe flooding after delivery, sitting up nights during daughter's confinement, stillbirth, unadvisedly sat up during confinement, uterine prolapse, and whooping cough while pregnant (also counted under history, pulmonary disorders).

[82] Includes inflammation of the side.

[83] Includes lump on sternum, lupus, skin tubercles, and ulcerated gland.

[84] Includes bubo, chancre, disease of testicle, mercury treatment, phymosis, swelling testicle, and urinary stricture.

[85] Includes low spirits resulting from reading a treatise entitled "Errors of Youth."

[86] Includes ailment (TB-related) always worse in winter.

[87] Includes always weakly, bad constitution, delicacy, easily affected by vicissitudes of weather, feeling dull/oppressed/unwell, malaise, never robust/very healthy, and weakness.

[88] Includes appetite poor/lost, biliousness, bowel complaints, flatulence, gastric fever, nausea, passage of worms, sickness, and vomiting.

[89] Includes bloody expectoration.

[90] Includes chills and rigors.

[91] Includes abnormal discharge, alternations of temperature, cramps, deafness, delirium, disturbed sleep, dizziness, dullness on percussion, dumb/loss of voice/aphonia, eruption, epistaxis, feverish, headache, irregular heartbeat, muscle weakness, palpitation, perspiration, thirst, tumor, ulcers, unspecified pectoral symptoms, urine abnormalities, and wheezing.

[92] Includes advice to change climate/go to country, blisters, cough mixture, croton oil, digitalis, liniment, opiate, potassium iodide, prescribed diet, and taraxacum.

[93] Includes hydrocephalus, mason's chest disease, and miner's phthisis.

[94] Includes records noting deaths from causes unknown, history unimportant, no family history, no history of constitutional affection, no history of phthisis, and unaware of any chest complaints.

[95] Includes accident, apoplexy, brain fever, cancer, childbirth, cholera, death from a decline, diabetes, dropsy, epilepsy, fever, general weakness, heart disease, ill with pain in head and stomach, in asylum, jaundice, rheumatism, rubeola, typhus, and unhealthy/not strong.

[96] Includes bad circumstances, chiefly farinaceous/tea and bread/vegetable, deficient, insufficient, little butcher meat, little food, living in lodgings, long intervals between meals, not plentiful, not nourishing, poorly kept, privation, scanty, sudden poverty and destitution, and want of proper nourishment.

[97] Includes cannot take broth or vegetables, cannot take/dislike of fat, dislike of animal food, exceedingly fond of broth, good deal of salt butter, high intake of salted meats, and prefers acid things.

[98] Includes moderately steady, never addicted to venereal pleasures, never hard drinker, never drunk spirits, not habitual drinker, not indulge in intoxicating drinks, regular, sober, and tolerably temperate.

[99] Includes exposure to wet/cold, changes of temperature (e.g., going out into cold after being heated at work), damp housing/work conditions, deficient clothing, sea bathing, sea voyage, sun on back while working, and washing (counted as both exposure and fatigue).

[100] Includes hard/heavy work, increased/new labor, long work hours, overexertion, perspiration while working, walking journey, and washing (counted as both exposure and fatigue).

[101] Includes alcohol use by self or spouse, carelessness, child/intercourse out of wedlock, dancing freely after alcohol, delirium tremens, dissipated/unsteady habits in self or family, exposure to cold on leaving taprooms, history of imprisonment, intemperance in self or spouse, mistreatment by spouse, prostitution, sleeping out/in shed/in stair, and smoking tobacco.

[102] Includes grief, poor work conditions, treatment by unlicensed practitioner, and sleeping in a strange bed.

[103] Includes teeth absent, decayed, dry, small, or sordes-covered, as well as gums bleeding, livid, orange, red, retracted, spongy, or ulcerated.

[104] Includes aphonia, as well as voices described as diminished, faint, harsh, husky, lost, whispering, rough, or weak.

[105] Includes dysphagia, pain on swallowing, and sensation of object in throat/blockage/choking.

[106] Includes congested, highly vascular, and injected.

[107] Includes hypertrophy and swelling.

[108] Includes cracked and furrowed.

[109] Includes covered, glazed, loaded, and streaked.

[110] Includes black, brownish, dark, florid, livid, and spotted.

[111] Includes grey, pale, pasty, and yellow.

[112] Includes flabby, irritable-looking, large, papillae enlarged, preternaturally clean, protruded, soft, tender, and tremulous.

[113] Includes bad breath, dry throat, rough epiglottis, sneeze, ulcers, and uvula anomalies, as well as lips described as blue, dry, fissured, sordes-covered, or thick, and mouths with abnormal tastes present, drawn-down corners, or dry.

[114] Includes catching, considerable, cutting, darting excessive, frequent, great, griping, heavy, intense, lancinating, much, neuralgic, racking, ripping, sharp, shooting, stabbing, and stitch.

[115] Includes aching, burning, constant, cramped, fixed, gnawing, longstanding, not severe, slight, some, and throbbing.

[116] Includes face and sensation load/fullness in head.

[117] Includes pain increased by long breath/expansion of chest/respiration, affecting respiration, and preventing full inspiration.

[118] Includes exercise, movement, stooping, and walking.

[119] Includes pain on percussion or touch and pain increased by a blow.

[120] Includes pain increased by bending, cold, eating, position, or standing, as well as increased in the morning, afternoon, or night.

[121] Includes loins.

[122] Includes kidney.

[123] Includes cardiac region, diaphragm, false ribs, heart, hypochondrium, and precordial region.

[124] Includes abdomen, epigastric, gastric, hypogastric, iliac fossa, liver, menstrual, spleen, side, and stomach.

[125] Includes body, intermittent, muscular, neck, occasional, pricking, relieved by blisters, relieved by pressure, skin, tumor/nodule, sudden, surface, and unspecified location.

[126] Includes dumb, half-comatose, impaired memory, incoherent, speech confused/difficult/incoherent/languid, and wandering mind.

[127] Includes deafness, Eustachian tube inflammation, hearing impaired/lost, noises/buzzing, pain in pars petrosa of the temporal bone, and tinnitus aurium.

[128] Includes amblyopia, blindness, eye opacity, flashes, floating objects, insensitivity/ hypersensitivity to light, muscae volitantes, myopia, pupil contraction/dilatation, staring, and vision dim/failing/imperfect/weak.

[129] Includes muscles flabby/small/flattened/wasted, sensation of powerlessness, and weakness in limbs/back/chest.

[130] Includes cramps, fits, increased sensibility of skin, irritable, legs failed, loss of smell/taste, nervous, muscular contraction, numbness, subsultus tendinem/twitching/jactitation, throbbing sensation in head, tingling sensation, tremor, and unsteady gait.

[131] Includes aggravated, bad, considerable, constant, continual, distressing, exhausting, frequent, good/great deal, hacking, hard, harsh, incessant, increased, long, loud, much, painful, paroxysmal, prolonged, reverberating, ringing, severe, spasmodic, troublesome, urgent, and violent.

[132] Includes occasional, scarce, short, slight, some, and tickling.

[133] Includes coughs described as dry, hollow, increased by activity/climate/dyspnea/posture, loose, moist, peculiar, and prevented by weakness.

[134] Includes considerable, copious, frequent, good/great deal, increased, large quantity, much, plentiful, and profuse.

[135] Includes rusty, brick-dust color, brownish, reddish, and tinged red.

[136] Includes adherent to vessel, difficult, tenacious, thick, tough, and unable to expel.

[137] Includes expectoration described as aerated, thin, or watery.

[138] Includes phlegm.

[139] Includes muco-purulent.

[140] Includes inconsiderable, little, moderate, not abundant, not great extent, not much, not very copious, occasional, slight, small quantity, and some.

[141] Includes straw hue, whitish yellow, and yellowish green.

[142] Includes black, bluish, characteristic (i.e., of TB-related disorders), dark colored, fetid, gelatinous, grayish, glairy, gum-like, not rusty, nummular, semitransparent, and unspecified.

[143] Includes diaphragmatic.

[144] Includes almost imperceptible, difficult, feeble, groaning, impeded, jerking, labored, moaning, noisy, oppressive, shallow, short, troublesome, very bad, and wheezing.

[145] Includes accelerated, frequent, and quick.

[146] Includes dilating alae nasi, laryngeal, and quiet.

[147] Includes two cases in which it is simply stated that vomicae are present.

[148] These describe normal breath sounds.

[149] Includes coughs described as laryngeal, pharyngeal, and reverberating.

[150] Includes crepitus/rales described as considerable, distinct, large, and well-marked.

[151] Includes crepitus/rales described as distant, faint, fine, obscure, occasional, and some.

[152] Includes crepitus/rales described as clicking, crackling, dry, loose, or moderate.

[153] Includes bubbling and splashing.

[154] Includes metallic click.

[155] Includes puffing and whiffling.

[156] Includes absent, distant, feeble, gone, imperfectly heard, inaudible, indistinct, obscured, and weak.

[157] Includes respiration described as coarse, exaggerated, harsh, intense, noisy, rough, sound increased, and wheezing.

[158] Includes altered, bisected, divided, interrupted, jerking, rapid, scarce air entering, and short.

[159] Includes vocal resonance described as exaggerated, loud, marked, pealing, or strong.

[160] Includes vocal resonance described as decreased, diminished, inaudible, or metallic.

[161] Includes vocal fremitus described as absent, diminished, impaired, indistinct, not felt, or wanting.

[162] Includes fremitus from moist sound, rhonchal fremitus, and increased or well-marked vocal fremitus.

[163] Includes jingling.

[164] Includes areas not clear, hard note, impaired clearness/note, more resistant, and resistance.

[165] Includes emphysematous, higher pitch, hyperresonance, indicative of vomicae, note increased, tympanitic, and unnaturally/unusually resonant.

[166] Includes chest expansion described as defective, deficient, difficult, diminished, en masse, feeble, impaired, less than normal, limited, little, much elevation on inspiration, not fully, not freely, not well, retracts instead, scarcely perceptible, and sides unequal.

[167] Includes anterior-posterior diameter increased, bulged/conical anteriorly, prominent/rounded near sternum, and pigeon breasted.

[168] Includes narrow, not well developed, and small.

[169] Includes barrel shape, bulging, prominent, and rounded.

[170] Includes depression, hollow, sunken, and softening.

[171] Includes congested-appearing (veins well-marked), intercostal spaces ill-marked, and winged scapulae.

[172] Includes sensation of rattling in trachea.

[173] Includes sensations of choking, smothering, and stifling.

[174] Includes ascites, belly dropsy, contains fluid, distended, edema of side/hypogastrium/ epigastrium/hypochondrium, effusion into, great, hard, pot belly, prominent, resonant, sensation of tightness, swollen, and tympanitic.

[175] Includes enlargement.

[176] Includes dullness anomalies at the hypochondrium or liver and measurements of the extent of dullness at the liver or spleen.

[177] Includes abnormal formation, absence, ankylosis, irritation, prominence, redness, roughness, spinal curvature, or swelling of bones and joints, as well as sensation of weight in limbs.

[178] Includes fingers clubbed, nails clavate/filbert-shaped, nails and fingertips blue, and nail nearly destroyed.

[179] Includes edema or swelling of the cardiac region, extremities, face, genitals, glands, head, mamma, posterior chest, or unspecified location, as well as swelling in the evening and an appearance of fullness.

APPENDIX G

SIGNS AND SYMPTOMS RECORDED IN SAMPLED CASES

FOR THE BACTERIOLOGICAL PERIOD, 1881 – 1905

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
1. APPEARANCE AND COMPORTMENT	283	107	146	536
Anemic	*12*	*0*	*2*	*14*
Anxiety[1]	*7*	*0*	*8*	*15*
Appearance, Healthy[2]	*12*	*21*	*18*	*51*
Appearance, Unhealthy[3]	*8*	*2*	*1*	*11*
Constitution	—	—	—	—
Delicate/Weak[4]	*4*	*0*	*0*	*4*
Phthisical[5]	*1*	*0*	*0*	*1*
Other[6]	*0*	*0*	*2*	*2*
Complexion	—	—	—	—
Dark[7]	*2*	*2*	*3*	*7*
Sallow/Yellow[8]	*4*	*0*	*7*	*11*
Countenance	—	—	—	—
Collapsed/Sunk[9]	*3*	*3*	*0*	*6*
Suffering[10]	*2*	*1*	*4*	*7*
Tranquil[11]	*1*	*0*	*0*	*1*
Debility[12]	*52*	*12*	*15*	*79*
Dizziness[13]	*9*	*6*	*14*	*29*
Emaciation[14]	*97*	*23*	*22*	*142*
Eyes, Abnormal Appearance[15]	*8*	*1*	*8*	*17*
Languor[16]	*5*	*0*	*3*	*8*
Pallor[17]	*33*	*12*	*21*	*66*
Position	—	—	—	—
Back[18]	*2*	*8*	*6*	*16*
Difficulty, Left Side	*0*	*0*	*1*	*1*
Difficulty, Right Side	*4*	*1*	*0*	*5*
Head Raised[19]	*0*	*8*	*1*	*9*
Right Side	*1*	*1*	*0*	*2*
Unaffected Side	*1*	*1*	*1*	*3*
Other[20]	*5*	*1*	*0*	*6*
Other Appearance[21]	*10*	*4*	*9*	*23*
2. CIRCULATORY	243	205	154	602
Auscultation	—	—	—	—
Heart, Action Decreased[22]	*17*	*17*	*6*	*40*
Heart, Action Increased[23]	*2*	*2*	*2*	*6*
Heart, 1st Sound Anomalies[24]	*11*	*13*	*27*	*51*
Heart, 2nd Sound Anomalies[25]	*9*	*9*	*12*	*30*
Heart/Blood Vessels, Other[26]	*1*	*3*	*5*	*9*

Appendix G (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
2. CIRCULATORY, CONT.				
Beat/Impulse	—	—	—	—
Impulse Decreased[27]	*10*	*13*	*10*	*33*
Impulse Increased[28]	*4*	*3*	*3*	*10*
Other Beat Anomalies[29]	*15*	*10*	*18*	*43*
Blood Count	—	—	—	—
Hemoglobin[30]	*2*	*0*	*0*	*2*
Red Blood Cells[31]	*4*	*1*	*0*	*5*
White Blood Cells[32]	*5*	*3*	*0*	*8*
Heart, Oppression/Weakness	*0*	*2*	*2*	*4*
Heart, Palpitation[33]	*1*	*10*	*7*	*18*
Percussion, Anomalies[34]	*36*	*23*	*24*	*83*
Pulse	—	—	—	—
≤ 74	*9*	*7*	*14*	*30*
75 – 84	*13*	*12*	*8*	*33*
85 – 94	*13*	*1*	*2*	*16*
95 – 104	*10*	*5*	*4*	*19*
105 – 114	*12*	*6*	*3*	*21*
≥ 115	*13*	*13*	*3*	*29*
Bounding[35]	*4*	*1*	*1*	*6*
Feeble/Weak[36]	*24*	*15*	*10*	*49*
Hard/Sharp[37]	*10*	*17*	*14*	*41*
Other Pulse Anomalies[38]	*6*	*11*	*6*	*23*
Other Circulatory[39]	*12*	*8*	*8*	*28*
3. DERMATOLOGICAL	165	83	55	303
Encrustation/Discharge[40]	*10*	*4*	*2*	*16*
Flushing/Redness[41]	*22*	*26*	*10*	*58*
Perspiration	—	—	—	—
Frequent/Profuse[42]	*27*	*13*	*7*	*47*
Night	*45*	*6*	*3*	*54*
Other[43]	*13*	*5*	*6*	*24*
Rash[44]	*6*	*7*	*7*	*20*
Node/Mass[45]	*3*	*0*	*1*	*4*
Skin	—	—	—	—
Cool[46]	*0*	*3*	*4*	*7*
Dry/Harsh[47]	*10*	*4*	*4*	*18*
Warm/Hot[48]	*7*	*8*	*1*	*16*
Other[49]	*18*	*5*	*9*	*32*

Appendix G (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
3. DERMATOLOGICAL, CONT.				
Ulcer	—	—	—	—
Limbs	*0*	*1*	*0*	*1*
Neck/Chest[50]	*1*	*0*	*0*	*1*
Oral/Facial[51]	*1*	*1*	*1*	*3*
Lupus/Scrofulous[52]	*2*	*0*	*0*	*2*
4. DIGESTIVE	200	67	113	380
Abdominal Fluctuation[53]	*3*	*1*	*4*	*8*
Acidity[54]	*4*	*3*	*5*	*12*
Anorexia[55]	*45*	*17*	*9*	*71*
Appetite, Increased	*0*	*0*	*1*	*1*
Bowels	—	—	—	—
Constipation[56]	*32*	*17*	*22*	*71*
Diarrhea[57]	*12*	*2*	*3*	*17*
Gripes/Tenesmus[58]	*3*	*0*	*1*	*4*
Stool Anomalies[59]	*9*	*1*	*2*	*12*
Eructation, Non-Acid[60]	*1*	*1*	*0*	*2*
Flatulence[61]	*6*	*3*	*6*	*15*
Nausea/Retching/Vomiting[62]	*58*	*15*	*36*	*109*
Other Digestive[63]	*27*	*7*	*24*	*58*
5. GENERAL	112	69	49	230
General Health Impaired[64]	*5*	*0*	*0*	*5*
Sleep, Disturbed[65]	*14*	*13*	*6*	*33*
Hectic	*3*	*0*	*0*	*3*
Temperature	—	—	—	—
Alternations[66]	*1*	*2*	*0*	*3*
Fever/Heat[67]	*7*	*6*	*2*	*15*
Shivering[68]	*15*	*5*	*8*	*28*
Thermometer	—	—	—	—
< 98°F[69]	*3*	*5*	*9*	*17*
98°F – 99°F[70]	*11*	*6*	*9*	*26*
> 99°F[71]	*40*	*25*	*10*	*75*
Thirst, Increased	*13*	*7*	*5*	*25*

Appendix G (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
6. GENITOURINARY	246	165	171	582
Amenorrhea[72]	*10*	*3*	*3*	*16*
Urinalysis	—	—	—	—
Acid	*52*	*33*	*25*	*110*
Albuminous	*23*	*22*	*16*	*61*
Alkaline	*1*	*0*	*0*	*1*
Chemical Reaction[73]	*0*	*1*	*2*	*3*
Components, Diminished[74]	*1*	*5*	*0*	*6*
Components, Other[75]	*7*	*8*	*4*	*19*
Deposit, Biological[76]	*22*	*12*	*16*	*51*
Deposit, Other[77]	*12*	*9*	*5*	*26*
Specific Gravity, Normal	*48*	*35*	*28*	*111*
Specific Gravity, Abnormal[78]	*4*	*1*	*4*	*9*
Sugar Present	*1*	*0*	*3*	*4*
TB Present	*2*	*0*	*0*	*2*
Urine Color	—	—	—	—
Dark[79]	*24*	*19*	*11*	*54*
Light[80]	*7*	*3*	*11*	*21*
Other[81]	*1*	*2*	*0*	*3*
Urine, Difficult/Obstructed[82]	*5*	*2*	*10*	*17*
Urine, Quantity, Decreased[83]	*3*	*5*	*3*	*11*
Urine, Quantity, Increased	*1*	*1*	*2*	*4*
Urination, Frequent Urge	*7*	*2*	*6*	*15*
Urination, Incontinence	*0*	*1*	*4*	*5*
Vagina, Physical Exam	*3*	*0*	*9*	*12*
Vagina, Discharge[84]	*3*	*0*	*2*	*5*
Other Genitourinary[85]	*9*	*1*	*7*	*17*
7. HISTORY, MEDICAL	247	137	123	507
Other Disorders	—	—	—	—
Cold[86]	*20*	*3*	*3*	*26*
OB/GYN[87]	*14*	*3*	*11*	*28*
Pulmonary	*46*	*29*	*9*	*84*
Strumous/Gland Swellings[88]	*15*	*4*	*2*	*21*
Venereal[89]	*6*	*2*	*2*	*10*
Other	*72*	*48*	*60*	*180*
Seasonal Disorder, Fall/Winter	*6*	*8*	*3*	*17*

Appendix G (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
7. HISTORY, MEDICAL, CONT.				
Symptoms	—	—	—	—
Debility[90]	*9*	*2*	*4*	*15*
Digestive[91]	*16*	*3*	*9*	*28*
Edema	*2*	*3*	*0*	*5*
Pulmonary	—	—	—	—
Cough	*1*	*0*	*2*	*3*
Dyspnea	*1*	*1*	*0*	*2*
Expectoration	*1*	*1*	*0*	*2*
Hemoptysis[92]	*6*	*6*	*0*	*12*
Pain/Uneasiness, Trunk	*4*	*1*	*3*	*8*
Pain/Uneasiness, Other[93]	*3*	*1*	*3*	*7*
Perspiration, Night	*3*	*0*	*0*	*3*
Shivering[94]	*8*	*12*	*3*	*23*
Other[95]	*9*	*6*	*5*	*20*
Treatment, Cod Liver Oil	*1*	*0*	*0*	*1*
Treatment, Other[96]	*4*	*4*	*4*	*12*
8. HISTORY, PERSONAL	445	236	207	888
Disease Duration	—	—	—	—
< 1 Month	*11*	*25*	*9*	*45*
1 – 4 Months	*40*	*10*	*21*	*71*
5 – 8 Months	*23*	*4*	*7*	*34*
≥ 9 Months	*28*	*13*	*13*	*54*
Family History	—	—	—	—
Death, Childhood/Infancy[97]	*35*	*18*	*16*	*69*
Death, Pulmonary[98]	*34*	*21*	*10*	*65*
TB-Related[99]	*24*	*6*	*3*	*33*
TB-Related Unknown[100]	*48*	*20*	*24*	*92*
Other[101]	*63*	*29*	*25*	*117*
Habits	—	—	—	—
Diet, Poor[102]	*4*	*1*	*1*	*6*
Diet, Other[103]	*1*	*2*	*0*	*3*
Temperate[104]	*22*	*24*	*21*	*67*
Housing, Unhealthy[105]	*12*	*8*	*4*	*24*

Appendix G (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
8. HISTORY, PERSONAL, CONT.				
Off Work	—	—	—	—
< 1 Month	*14*	*14*	*4*	*32*
1 – 4 Months	*11*	*1*	*7*	*19*
5 – 8 Months	*0*	*1*	*1*	*2*
≥ 9 Months	*4*	*0*	*1*	*5*
Precipitating Factors	—	—	—	—
Exposure to Elements[106]	*22*	*15*	*6*	*43*
Fatigue[107]	*5*	*2*	*5*	*12*
Immorality[108]	*28*	*18*	*17*	*63*
Injury	*10*	*1*	*5*	*16*
Other[109]	*6*	*3*	*7*	*16*
9. MOUTH AND THROAT	136	83	64	283
Dental/Alveolar Disease[110]	*15*	*5*	*7*	*27*
Difficulty Speaking/Hoarseness[111]	*8*	*7*	*3*	*18*
Difficulty Swallowing[112]	*1*	*0*	*4*	*5*
Livid/Livid[113]	*7*	*2*	*2*	*11*
Edema/Enlargement[114]	*2*	*2*	*0*	*4*
Pain/Soreness	*2*	*0*	*1*	*3*
Tongue	—	—	—	—
Dry/Fissured[115]	*8*	*7*	*3*	*18*
Foul/Furred[116]	*34*	*26*	*17*	*77*
Red[117]	*9*	*4*	*5*	*18*
White[118]	*19*	*14*	*6*	*39*
Other Tongue[119]	*12*	*2*	*4*	*18*
Other Oral[120]	*19*	*14*	*12*	*45*
10. PAIN	224	169	161	554
Acute/Severe/Violent[121]	*19*	*16*	*23*	*58*
Dull/General/Unspecified[122]	*18*	*6*	*6*	*30*
Head[123]	*20*	*14*	*19*	*53*
Increased by	—	—	—	—
Cough/Inspiration[124]	*23*	*33*	*2*	*58*
Exertion/Motion[125]	*8*	*7*	*12*	*27*
Pressure[126]	*12*	*3*	*13*	*28*
Other[127]	*2*	*0*	*7*	*9*
Limbs[128]	*10*	*10*	*14*	*34*

Appendix G (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
10. PAIN, CONT.				
Trunk, Back/Shoulders	*23*	*13*	*11*	*47*
Trunk, Chest/Thorax[129]	*26*	*30*	*10*	*66*
Trunk, Other[130]	*47*	*23*	*25*	*95*
Other Pain[131]	*16*	*14*	*19*	*49*
11. NEUROLOGICAL	89	42	131	262
Confusion/Delirium[132]	*4*	*8*	*19*	*31*
Ear/Hearing Anomalies[133]	*0*	*5*	*4*	*9*
Eye/Vision Anomalies[134]	*16*	*14*	*27*	*57*
Muscular Weakness[135]	*17*	*3*	*12*	*32*
Reflex Anomalies[136]	*24*	*5*	*30*	*59*
Other Neurological[137]	*28*	*7*	*39*	*74*
12. RESPIRATORY, GENERAL	491	303	102	896
Cough	—	—	—	—
Distressing[138]	*64*	*34*	*10*	*108*
Disturbs Sleep	*3*	*1*	*0*	*4*
Little[139]	*24*	*18*	*8*	*50*
Longstanding	*4*	*2*	*0*	*6*
Morning	*10*	*5*	*1*	*16*
Night	*17*	*5*	*0*	*22*
Unspecified	*21*	*10*	*3*	*34*
Other[140]	*12*	*14*	*3*	*29*
Dyspnea, All Types	*37*	*43*	*18*	*98*
Expectoration	—	—	—	—
Abundant[141]	*33*	*18*	*1*	*52*
Bacteria, Pneumococci	*3*	*6*	*0*	*9*
Bacteria, TB	*28*	*0*	*0*	*28*
Bloody[142]	*11*	*14*	*2*	*27*
Dense[143]	*16*	*15*	*3*	*34*
Easy/Frothy[144]	*19*	*20*	*3*	*42*
Mucus	*8*	*8*	*2*	*18*
Opaque/White	*12*	*12*	*2*	*26*
Pus[145]	*23*	*15*	*0*	*38*
Scanty[146]	*13*	*15*	*6*	*34*
Viscid	*3*	*2*	*0*	*5*

Appendix G (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
12. RESPIRATORY, GENERAL, CONT.				
Expectoration, Cont.				
Yellow/Greenish[147]	*31*	*7*	*1*	*39*
Other[148]	*16*	*14*	*2*	*32*
Hemoptysis, All Types	*19*	*3*	*1*	*23*
Phthisis Pulmonalis[149]	*2*	*0*	*0*	*2*
Respiration	—	—	—	—
Abdominal	*0*	*0*	*1*	*1*
Impaired[150]	*6*	*22*	*9*	*37*
Rapid[151]	*5*	*4*	*2*	*11*
Other[152]	*10*	*14*	*0*	*24*
Rate, ≤ 29	*26*	*24*	*18*	*68*
Rate, 30 – 39	*12*	*9*	*4*	*26*
Rate, ≥ 40	*3*	*8*	*1*	*12*
13. RESPIRATORY, AUSCULTATION AND PERCUSSION	369	173	56	598
Auscultation	—	—	—	—
Amphoric/Cavernous[153]	*7*	*3*	*0*	*10*
Bronchial/Puerile[154]	*12*	*8*	*3*	*23*
Bronchophony/Aegophony[155]	*1*	*3*	*0*	*4*
Crepitus	—	—	—	—
Bronchitic[156]	*5*	*1*	*2*	*8*
Coarse/Loud[157]	*3*	*2*	*2*	*7*
Cooing/Musical[158]	*5*	*7*	*3*	*15*
Slight/Small[159]	*12*	*8*	*5*	*25*
Subcrepitus/Other[160]	*25*	*12*	*4*	*41*
Friction/Frottement[161]	*7*	*14*	*0*	*21*
Gurgling/Gargouillement[162]	*7*	*4*	*0*	*11*
Metallic Tinkle[163]	*3*	*0*	*0*	*3*
Mucus[164]	*39*	*23*	*8*	*70*
Pectoriloquy	*2*	*0*	*0*	*2*
Rales, Unspecified[165]	*9*	*8*	*3*	*20*

Appendix G (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
13. RESPIRATORY, AUSCULTATION AND PERCUSSION, CONT.				
Auscultation, Cont.				
Respiration	—	—	—	—
Blowing	*0*	*2*	*1*	*3*
Faint/Obscure[166]	*17*	*21*	*7*	*45*
Loud/Raucous[167]	*21*	*23*	*4*	*48*
Prolonged	*23*	*15*	*0*	*38*
Tubular[168]	*17*	*9*	*2*	*28*
Other[169]	*11*	*7*	*0*	*18*
Sibilus	*3*	*4*	*1*	*8*
Sonorous	*5*	*9*	*2*	*16*
Vocal Resonance, Increased[170]	*24*	*9*	*3*	*36*
Vocal Resonance, Other[171]	*7*	*7*	*1*	*15*
Palpation, Vocal Fremitus	—	—	—	—
Decreased[172]	*6*	*12*	*1*	*19*
Increased[173]	*26*	*11*	*2*	*39*
Percussion	—	—	—	—
Dull/Diminished Resonance[174]	*62*	*35*	*12*	*109*
Hollow/Tympanitic[175]	*10*	*11*	*5*	*26*
14. RESPIRATORY, CHEST FORM/SENSATION	69	53	11	133
Expansion, Imperfect[176]	*20*	*19*	*1*	*40*
Form	—	—	—	—
Arched[177]	*3*	*1*	*1*	*5*
Contracted[178]	*13*	*4*	*1*	*18*
Expanded/Broad[179]	*6*	*9*	*1*	*16*
Flattening[180]	*11*	*4*	*1*	*16*
Other[181]	*6*	*4*	*0*	*10*
Measurement, Sides Unequal	*3*	*7*	*2*	*12*
Sensation	—	—	—	—
Constriction/Tightness[182]	*2*	*2*	*2*	*6*
Suffocation[183]	*5*	*2*	*2*	*9*
Surgical Aspiration[184]	*0*	*1*	*0*	*1*

Appendix G (*continued*)

Signs and Symptoms	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
15. MISCELLANEOUS	177	87	117	380
Abdomen	—	—	—	—
Edema[185]	*44*	*14*	*26*	*84*
Liver Anomalies[186]	*9*	*7*	*8*	*24*
Percussion, Dullness[187]	*36*	*18*	*21*	*75*
Tumor[188]	*7*	*3*	*7*	*17*
Other[189]	*8*	*2*	*6*	*16*
Bone and Joint	—	—	—	—
Pain	*1*	*5*	*6*	*12*
Rheumatism	*3*	*2*	*5*	*10*
Other[190]	*19*	*10*	*19*	*38*
Fingers/Nails, Anomalies[191]	*21*	*0*	*1*	*22*
Edema, Limbs, Lower	*11*	*12*	*19*	*42*
Edema, Other[192]	*18*	*13*	*9*	*40*
Percussion, Other[193]	*0*	*1*	*0*	*1*
TOTAL	3467	1948	1647	6232

[1] Includes patients described as distressed, excited/excitable, ill-natured, irritable, or nervous.

[2] Includes patients described as having a good layer of subcutaneous fat or good muscular development, as well as powerfully/strongly/well made/built/developed, stout, or strong.

[3] Includes patients described as having poor muscular development, as well as acutely/very ill, ill, neglected, or poorly built/developed.

[4] Includes patients described as delicate, having a delicate build, or weak-looking.

[5] Includes eyes suggestive of strumous diathesis.

[6] Includes melancholic or nervous temperament.

[7] Includes brownish, dingy, dusky, and swarthy.

[8] Includes jaundice.

[9] Includes cheeks hollow, eyes sunken, face drawn, malar bones prominent, and neck hollows exceedingly marked.

[10] Includes patients described as crying when questioned, dour, emotional, having a strained expression, sighing without apparent reason, or worn.

[11] Includes comfortable.

[12] Includes patients described as collapsed, disinclined for exertion, feeble, feeling quite done out, having lost power/strength, or weak.

[13] Includes collapse on getting up, dazed, dizziness, giddiness, lightheaded, semiconscious, and unconscious.

[14] Includes cachectic, flesh loss, ill-nourished, little subcutaneous fat, slightly built, spare, thin, and wasting.

[15] Includes conjunctivae described as bile-tinged, bloodshot, congested, livid, pale, red, suffused, or yellow, as well as dark lines under eyes, dull, dim, puffy lids, and red.

[16] Includes lethargic, listless, and tired.

[17] Includes color lost/poor, pale, pallid, and pasty.

[18] Includes mostly back, recumbent, and side or back where no specific difficulty was described with the opposite side.

[19] Includes must sit to breathe, prefers to sit, propped with pillows, semirecumbent, and unable to lie.

[20] Includes cannot sit or stand, leg(s) drawn up, and unable to sit.

[21] Includes alert, asymmetrical face, attempts to get out of bed, blotched, clear intellect, complexion not robust but mucus membranes well colored, contradictory statements, countenance blank, dislikes noise, facile, older/younger than years, pinched face, seems unintelligent (perhaps due to deafness), soft and flabby, talkative, undersized/small stature, very intelligent, and weight gain.

[22] Includes heart sounds described as distant, faint, feeble, muffled, not loud, obscured (often by lung sounds), small like a child's, toneless, or weak.

[23] Includes heart sounds described as accentuated, amplified, loud, rapid, thumping, or tachycardia.

[24] Includes first, systolic or VS (ventricular systole) sounds described as abrupt, accentuated, blowing, booming, closure indefinite, dull, faint, feeble, loud, prolonged, reduplicated,

ringing, roughened, slapping, thumping, toneless, weak, or accompanied by a bruit or murmur, as well as heard in swelling or thyroid.

[25] Includes second, AS (atrial systole) or VD (ventricular diastole) sounds described as accentuated, better heard than first, booming, lacking tone, loud, metallic, reduplicated, ringing, weak, or accompanied by a murmur.

[26] Includes anti-diastolic [murmur], continuous murmur, pericardial friction, Russell murmur, and sounds described as deep in tone or displaced.

[27] Includes apex beat, impulse, or action of heart described as heaving, punctuate, or rapid.

[28] Includes apex beat, impulse, or action of heart described as difficult to locate, diffuse, faint, imperceptible, indistinct, intangible, not felt, not felt accurately, or not well marked.

[29] Includes apex beat, impulse, or action of heart described as displaced (including location other than 5th interspace), irregular, or transmitted to trachea.

[30] Recorded values include 50% and 60%.

[31] Recorded values include 3.8 million to 6 million.

[32] Recorded values include 8125 to 31500.

[33] Includes throbbing neck vessels and throbbing sensation in temples.

[34] Includes patients for whom the extent of cardiac dullness was reported as expanded, diminished, displaced, obscured by lung percussion anomalies, or with measurements.

[35] Includes pulses described as rapid or running.

[36] Includes pulses described as low tension, not very full, not well sustained, poor volume, or thin.

[37] Includes pulses described as easily/unduly palpable, engorged, enlarged, expansile, high tension, large, marked, tough, very evident, violent, or visible.

[38] Includes pulses described as atheromatous, dicrotic, flickering, irregular, shotty, slapping, slow, or whip-end, as well as capillary pulse present, epigastric pulse absent, pulse felt within mass, or pulses unequal between sides.

[39] Includes bruit de diable (indicative of anemia), valvular disease, and vessels described as dilated, enlarged, engorged, full, irregular, prominent, rolling, sclerosed, static, thickened, tortuous, or varicose.

[40] Includes discharge from the breast, ear, eyes, eruption, nose (coryza, epistaxis), or metatarsal.

[41] Includes appears hot, blush, capillary dilation/ingestion, congestion, florid, high color, infiltrated, inflammation, injected, and red/herpetic spots.

[42] Includes perspiration described as all over, copious, good/great deal, heavy, severe, or whole body.

[43] Includes unspecified perspiration, as well as that accompanying cough, palpitation, or pain; described as clammy, cold, little/slight, or having a sour evil odor; or occurring on the face or head.

[44] Includes eruptions of acne, eczema, herpes, papules, psoriasis, or red/hemorrhagic spots; or described as itchy, mottled, patchy, or scaly.

[45] Includes bean-sized swellings or nodules on the tibiae, nevi, or warty eruptions.

[46] Includes clammy.

[47] Includes desquamated and scaly skin.

[48] Includes pungent skin.

[49] Includes bedsore, cyanosed, deep/brown pigmentation on abdomen, dermography, dusky hue of affected limb, ectropion, extremities blue, hair scanty/lost, hemorrhagic spot, lineal albicantes (stretch marks), moist, mottled, neglected, parchment-like, scalp wound, scratched, sloughing, and wrinkled.

[50] Includes sternal abscess.

[51] Includes granulative tissue on eyelid and mouth ulcer.

[52] Includes tubercular dactylitis and tubercular sore on hand.

[53] Includes wave or succussion on percussion.

[54] Includes acid eructation, acid stomach, heartburn, and waterbrash.

[55] Includes all descriptions of reduced appetite.

[56] Includes bowels described as confined, costive, irregular, or not moved since admission.

[57] Includes bowels described as loose.

[58] Includes pain localized to the sigmoid flexure.

[59] Includes stool described as black, bloody, foul smelling, having an offensive odor, mucus, tarry, or including worms.

[60] Includes eructation of clear, watery material.

[61] Includes wind.

[62] Includes sickness.

[63] Includes abdominal tightness, atonic stomach, colicky pain, digestion easily deranged, disagreeable sensations after food (burning, distension, heaviness, load, pain, swelling, weight), dyspepsia, epigastric heaviness, hemorrhoids, hiccough, indigestion, paralysis of sphincter ani, rectal prolapse, stomach troubles him/her, stool passed involuntarily, and tubercular peritonitis.

[64] Includes patients described as dying, in failing health, in poor condition, and weakly child.

[65] Includes patients described as sleeping badly, little, or not well, as well as insomniac, restless, sleepless, or unable to sleep.

[66] Includes patients reporting a sensation of cold after perspiration.

[67] Includes feverish, heat, overheated, and sensation of fever or heat.

[68] Includes chills, rigors, and sensation of cold.

[69] Includes temperature ranges extending below 98°F, those designated subnormal or normal and subnormal without numerical values, one case in which the thermometer wouldn't rise, and one case ranging 97°F – 100°F which was also counted among the elevated temperatures below.

[70] Includes temperatures designated as normal without a numerical value.

[71] Includes temperature ranges extending above 99°F, as well as one case also included among the low temperatures above owing to a range of 97°F – 100°F.

[72] Includes irregular menses and menopausal.

[73] Includes all cases in which a reaction (e.g., appearance of a coagulum or precipitate) occurred following exposure of the urine to boiling, perchloride of iron, or silver nitrate.

[74] Includes chlorides.

[75] Includes the presence of phosphates, urates, urea, and uric acid.

[76] Includes deposits of blood, blood cells (red or white), cylindroids (uncertain tube casts), epithelial cells, mucus, pus, or tube casts.

[77] Includes urine described as cloudy, glairy, muddy, smoky, translucent, or turbid, as well as deposits described as amorphous, flocculent, or white.

[78] All abnormal densities are higher than the normal range.

[79] Includes urine described as bright, high colored, or dark, deep or rich shades of amber.

[80] Includes urine described as clear, light, pale, partly whitish, or light or pale shades of amber, lemon, straw, or yellow.

[81] Includes red urine.

[82] Includes delayed, painful, requiring a catheter, retained, and scalding.

[83] Includes quantities described as diminished, scanty, or small, as well as measured quantity less than 45 ounces.

[84] Includes menstrual flooding, pieces of tissue, scanty menses, and uterine hemorrhage.

[85] Includes antemenstrual mammary swelling, antemenstrual pain, atrophied breasts, dysmenorrhea, dyspareunia, frequent menstruation, genital pain, inguinal pain, itching vulva, and urine described as irregular, thick, or containing crystalline bodies.

[86] Includes chill and cold with cough

[87] Includes abnormal vaginal discharge, amenorrhea, childbirth requiring five years of bed rest to recover, childbirth with instruments, flooding after childbirth, inflammation of womb, menstrual pain/headache, miscarriage, origin of disorder (tubercular) traced to childbirth, uterine displacement/prolapse, weakness after childbirth, and weaning a child.

[88] Includes diseased knee, spinal disease, and water in head, as well as glands or lymph nodes described as swollen, enlarged, or suppurating.

[89] Includes gonorrhea, specific history, and syphilis.

[90] Includes fainting, never healthy/robust/strong, tired, and weakness.

[91] Includes bilious attacks, bowel complaints, dysentery, dyspepsia, enteric fever, gastric fever, gastric ulcer, gastritis, indigestion, liver abscess, loss of appetite, sickness, stomach cramps, stool anomalies, and vomiting.

[92] Includes expectoration described as blood-streaked, bloody, or having a blood reaction.

[93] Includes sore throat.

[94] Includes chills and rigors.

[95] Includes albumen in urine, choking sensation, deafness, delirium, discharge from ears, dizziness, enlarged head, epistaxis, fits, hair loss, mitral murmur, perspiration other than night, pimple, temperature anomalies, tickling in throat, tube casts in urine, urine decreased and dark colored, and wheezing.

[96] Includes amputation, asylum, change of climate, Easton's syrup overdose, paracentesis, poultice, sanitorium, tooth drawn, and tonsils removed.

[97] Includes miscarriages; those reported as having died in childhood from pulmonary causes are included under family history of pulmonary disorders, below.

[98] Includes cold, as well as family with pulmonary disorders during childhood.

[99] Includes hemoptysis, hydrocephalus or water in the head, and swollen neck glands, as well as one decline noted "no other history," i.e., of TB-related illness.

[100] Includes records noting deaths from causes not given or unknown; family histories without chest/lung problems, consumption, constitutional disorder, hereditary taint, lung/bone/brain/gland swellings, phthisis, TB-related, tubercular taint; and family histories described as not bearing on case (i.e., not tubercular), obscure, unimportant, or unknown.

[101] Includes accident, acute illness, alcoholism, apoplexy/stroke, blood poisoning, bone disease, brain/nervous disease, Bright's/kidney disease, British cholera, broken heart, cancer, cardiac disease, cerebral hemorrhage, childbed, chill, cramps, debility, decline, diabetes, diphtheria, dropsy, enteric, erysipelas, fever, growth in bowels, headache, poor health, inflammation of bowels, influenza, internal growth, none of nervous disease, paralysis, pelvic trouble, rheumatics/rheumatism, rickets, scarlet fever, sciatica, suicide following head injury, syphilis, tonsils removed, tumor, typhus, and weak legs.

[102] Includes diets described as irregular, largely bread and tea, poor, and unsatisfactory.

[103] Includes sickness from meat or fat and cases in which sour milk or vegetables agree best.

[104] Includes moderate or absent use of alcohol and tobacco as well as steady habits.

[105] Includes housing described as airless, cold, damp, lodgings, poorly ventilated, those in which the windows are closed at night, and those with more than two occupants per room (e.g., five or more occupants in a "room and kitchen house"), following the definition for overcrowded housing given in Beevor (1899, 1011).

[106] Includes exposure to all weather, alternations of temperature, cold, draughts or cold after being heated, wet, as well as catching chill, putting off thick winter coat, waking in snow, and working in a draughty space.

[107] Includes exertion, heavy lifting, overuse injury, overwork, standing stationary to work, standing to watch football match, strain, and too much school work.

[108] Includes indications of current or former overindulgence in alcohol such as alcoholic, free/heavy drinking, injury while drunk, intemperance (self or spouse), too much alcohol, and trembling hands attributed to drinking; tobacco use described as heavy or comprising 3 ounces or more per week (the equivalent of about 4.5 modern packs of cigarettes); exposed self to risk of sexually transmitted infection; taking insufficient care of self, and improper and careless dieting.

[109] Includes chemical exposure, confined/impure atmosphere at work, does not take much exercise, family and business trouble, grief, infection from spouse, mother out all day, neglect, pressure (on affected part), and travel.

[110] Includes teeth described as anomalously erupted, carious, decayed, dirty, double-notched, in bad condition, lost, neglected, sordes-covered, or stunted, as well as gumboils and pale gums.

[111] Includes voices described as husky, muffled, thick, or weak.

[112] Includes pain on swallowing.

[113] Includes livid and a sensation of roughness in the throat.

[114] Includes congestion and swelling.

[115] Includes furrowed.

[116] Includes caked, coated, dirty, and glazed.

[117] Includes black line down center.

[118] Includes yellow.

[119] Includes bald patch, fibrillar contractions, flabby, indented by teeth, large, leathery, papillae congested, protruded, raw, rough, ruffed, protruding to one side, and tremulous.

[120] Includes bad breath, dry throat, high-pitched voice, vertical displacement of trachea, scanty saliva, sensation of foreign body in windpipe, stomatitis, and thrill on palpation of thyroid, as well as lips described as blue/cyanosed, cracked, dry, dusky, having a pale mucus membrane, pale, parched, pink and parted, or sordes-covered; palates described as high and narrow or perforated, and mouths with bad taste present, or foul.

[121] Includes acute, cutting, darting, great, lancinating, like sword being run through, making him cry out, making him draw himself together, often, racking, severe, sharp, shooting, so bad he thought he'd die, tearing, troublesome, and violent.

[122] Includes aching, bodily, burning, constant, mild, moderate, slight, throbbing, and transitory.

[123] Includes eyes, jaw, and tongue.

[124] Includes pain increased by breathing or deep/long breaths and that preventing or impeding cough, deep breath, or respiration.

[125] Includes pain increased by hip flexion, movement, stooping or rising from stooping, and that preventing walking.

[126] Includes pain on palpation, percussion, or touch.

[127] Includes pain increased at night or in the morning, in particular positions, and in wet weather.

[128] Includes pain in the "caput region" assumed to be the femoral head and in the loins.

[129] Includes pain referred to the axilla, breast, cardiac region, costal margin, hepatic region, hypochondrium, or sternum; infraclavicular, inframammary, mammary, or precordial pain; and needle angina.

[130] Includes pain two inches below the xiphoid as well as that referred to the abdomen, belly, epigastrium, flank, gastric region, hypogastrium, iliac region/fossa, liver region, lower anterior lateral region, pelvis, side, stomach.

[131] Includes pain described as dragging, general, like an open sore, occasional, paroxysmal, pins and needles, pricking, radiating, recurrent, rheumatic, stinging, sudden, or wandering; that referred to gland, mass, muscle, neck; that preventing school, reading, standing, or work; and that occasioned by fits or relieved by cold water, drink, food, motion, position, or pressure.

[132] Includes aphasia, dense, difficult to communicate with, judgment feeble, memory failure/impairment, mind wanders, not very bright, raving, reason affected, talks nonsense, and unable to understand speech, as well as speech anomalies including defective articulation and speech described as hesitating, indistinct, jerky, not plain, slowed, slurring, thick, or unintelligible.

[133] Includes deaf, deficient/dull hearing, and ringing in ears.

[134] Includes blind, corneitis, diplopia, eyes bright, hurt by light, iritis, leucoma, nystagmus, ophthalmia tarsi, optic neuritis, protruded, seeing spots/black dots, sight poor/failing, spot near macula, squint, strabismus/convergent, or weakness, as well as pupils described as contracted, dilated, reacting poorly to light or accommodation, or unequal.

[135] Includes muscles described as feeble, flabby, poorly developed, or wasted, as well as weakness of the abdominal wall, back, or legs, and atrophy, loss of power, thin abdominal wall, neck unable to hold up enlarged head, and unequal grip.

[136] Includes anomalies of abdominal, ankle clonus, cremasteric, knee/patellar, plantar, triceps tendon, wrist, and unspecified reflexes.

[137] Includes ataxia, cerebral irritation symptoms, coma, convulsions, cramps, difficulty standing, fits, impaired sense of smell, increased head size, Kernig's sign, lateral inequality of facial or neck muscles, limited movement, limpness, muscle twitching, numbness, paralysis, pins and needles/falling asleep, sciatica, sensation anomalies, spasm, staggering, or tremor, and gait anomalies including jerky, limping, stiff, unable to walk, or unsteady, as well as muscles described as distended, flexed, or rigid.

[138] Includes aggravated, bad, constant, frequent, good deal, hacking, hard, heavy, increasing, loud, paroxysmal, severe, troublesome, and violent.

[139] Includes moderate, not much, occasional, short, slight, some, and tickling.

[140] Includes coughs described as brassy, dry, irritating, laryngeal, loose, moist, or nearly causing vomiting, as well as those increased by cold, exertion, meals, and wet.
[141] Includes 1.5 inches in vessel, considerable, fair/great/large quantity, good/great deal, increasing, increased in morning or winter, and profuse.

[142] Includes blood-stained/streaked/tinged, brown spots, brownish, red-streaked, reddish, and rusty.

[143] Includes adhesive, difficult, sticky, tenacious, thick, and tough.

[144] Includes expectoration described as aerated, thin, or watery.

[145] Includes muco-purulent.

[146] Includes 0.25 inches in vessel, moderate quantity, not abundant, not much, one or two portions, partial layer, practically none, slight, small quantity, and some.

[147] Includes greenish yellow.

[148] Includes black/blackish, bronchial, dark-colored, fetid, foul-smelling, mass, non-aerated, nummular, and unspecified

[149] Includes tubercular empyema.

[150] Includes difficult, distressing, forced, grunting, labored, noisy, oppressed, sighing, stertorous, and wheezing.

[151] Includes hurried and quick.

[152] Includes crackling, dilating alae nasi/nostrils, gurgling, irregular, rattling, shallow, thoracic, and tumid.

[153] Includes bell sound from knocking coins together in front of chest, cavity breaking down, evidence of softening, extensive disease (i.e., tubercular) both lungs, and suspicious of cavity.

[154] These describe normal breath sounds and include sounds described as fairly good.

[155] Includes aegophony, bleating vocal resonance, and nasal vocal resonance.

[156] Includes lung sounds described as indicating a slight degree of bronchitis.

[157] Includes crepitus/rales described as abundant, considerable, distinct, large, and well-marked.

[158] Includes crepitus/rales described as high-pitched and piping.

[159] Includes crepitus/rales described as distant, faint, fine, obscure, occasional, and some.

[160] Includes crepitus/rales described as clicking, crackling, dry, hollow, low-pitched, mixed, or rough.

[161] Includes sounds described as friction-like and suggestive of friction.

[162] Includes bubbling, splashing, and splashing on shaking patient.

[163] Includes metallic tinkling.

[164] Includes sounds described as indicating catarrh, mucocrepitant, mucopurulent, and submucous.

[165] Includes unspecified crepitation.

[166] Includes defective, deficient, diminished, distant, feeble, gone, inaudible, lessened, loss, and weak.

[167] Includes respiration described as coarse, exaggerated, harsh, marked, noisy, rough, and wheezing.

[168] Includes subtubular.

[169] Includes cogwheel rhythm, heightened pitch, hoarse, hollow, jerky, short, superficial, tracheal, tubercular element, and wavy.

[170] Includes vocal resonance described as distinct, exaggerated, great, marked, pronounced, or relatively stronger, as well as whispered voice heard through stethoscope distantly.

[171] Includes vocal resonance described as decreased, diminished, or not much increased.

[172] Includes vocal fremitus described as absent, diminished, doubtful, impaired, indistinct, lost, not readily/well felt, or slight.

[173] Includes bronchial or rhonchal fremitus, thrill on inspiration, and vocal fremitus described as considerable, distinct, due to pleural friction, great, increased slightly/not greatly, or well felt/marked.

[174] Includes defective, flat, impaired, less clear, loss of tone/toneless, note not very good, reduced, seeming difference, skodiac, and wooden.

[175] Includes heightened pitch, hyperresonance, and tympanitic.

[176] Includes chest expansion described as deficient, diminished, limited, little range of motion, not full, retracting/indrawing, poor, or sides unequal

[177] Includes anterior-posterior curvature, coming out in front, and pigeon-breasted.

[178] Includes poorly developed, wasted, and walls thin.

[179] Includes barrel shape, bowl shape, bulging, and increased laterally.

[180] Includes depression and retraction.

[181] Includes clavicles raised, emphysematous swelling, grooved, ill-shaped, lobe shrinking, lung adhesion to chest wall, mass in axilla, one shoulder higher than the other, and suggestive of beading (i.e., rachitic).

[182] Includes gripping.

[183] Includes sensations of choking, heaviness, smothering, and weakness.

[184] Includes needle aspiration of a small quantity of clear, straw-colored fluid from the eighth intercostal space.

[185] Includes ascites, board-like, distended, enlarged, fullness, gaseous, hard, resistant to palpation, rigid, tense, and tympanitic.

[186] Includes diminished, enlarged (other than patients for whom specific liver dullness measurements were recorded, included under abdominal dullness), displaced, irregularly shaped, and margin indefinite.

[187] Includes measurements of the extent of dullness of the liver or spleen, as well as other percussion anomalies of the spleen.

[188] Includes area of resistance/thickening to palpation, globular swelling, irregularity suggestive of peritoneal TB, mass, and small hard lumps

[189] Includes flaccid, gurgling/splashing heard via auscultation, hardish ridge, lax, retracted, stomach dilated, thrill, throbbing, and umbilical hernia.

[190] Includes caries, disease, edema, enlargement, fracture, grating, loss, projection, restricted movement, sinus, weakness of the bones and joints, as well as acromegaly, flattened arches, Pott's disease of the spine, rickets, sensation of hump in caput region (assumed femur), spinal depression, spinal curvature, and unequal leg lengths.

[191] Includes fingers clubbed and nails curved.
[192] Includes edema or swelling of the arms, body, cardiac region, chest, face, hands, genitals, limbs, lumbar region, neck, thyroid, or wrists, as well as myoidema, pitting edema, tense and drum-like edema, and swelling reduced by bed rest or increased by coughing or at night.

[193] Includes tympanitic sound on percussion over tumor.

APPENDIX H

TREATMENTS RECORDED IN SAMPLED CASES

FOR THE ESSENTIALIST PERIOD, 1794 – 1820

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
1. APPLICATIONS	22	13	9	44
Anodyne	*6*	*3*	*1*	*10*
Blister	*11*	*9*	*4*	*24*
Camphor	*1*	*0*	*2*	*3*
Mustard Plaster	*2*	*0*	*0*	*2*
Other	*2*	*1*	*2*	*5*
2. BLEEDING	11	7	3	21
Cupping	*7*	*3*	*2*	*12*
Venesection	*4*	*4*	*1*	*9*
3. DIETS	8	6	2	16
Low Diet	*1*	*0*	*0*	*1*
Middle Diet	*1*	*0*	*0*	*1*
Milk Diet	*2*	*1*	*2*	*5*
Porter	*1*	*2*	*0*	*3*
Other[1]	*3*	*3*	*0*	*6*
4. DRUGS, OTHER	13	5	2	20
Assafoetida	*2*	*1*	*2*	*5*
Digitalis	*3*	*2*	*0*	*5*
Squill[2]	*8*	*2*	*0*	*10*
5. INHALATIONS	6	2	0	8
Water Vapor	*0*	*2*	*0*	*2*
Other[3]	*6*	*0*	*0*	*6*
6. LAXATIVES	30	18	12	60
Castor Oil	*9*	*4*	*6*	*19*
Enema	*3*	*6*	*0*	*9*
Mineral	*8*	*6*	*2*	*16*
Rhubarb	*7*	*2*	*2*	*11*
Other[4]	*3*	*0*	*2*	*5*
7. MERCURIALS	5	4	10	19
External	*0*	*1*	*5*	*6*
Internal	*5*	*3*	*5*	*13*
8. MIXTURES	18	4	4	26
Acid	*4*	*2*	*0*	*6*
Camphor	*2*	*0*	*0*	*2*
Mucilaginous	*4*	*1*	*3*	*8*
Other	*8*	*1*	*1*	*10*

Appendix H (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
9. OPIATES	14	7	5	26
External	*1*	*0*	*1*	*2*
Internal, Oral	*10*	*6*	*4*	*20*
Internal, Rectal[5]	*3*	*1*	*0*	*4*
10. VOMITS	8	1	2	11
Ipecac	*5*	*1*	*1*	*7*
Other/Unspecified[6]	*3*	*0*	*1*	*4*
11. WASHES	4	0	3	7
Vinegar	*1*	*0*	*0*	*1*
Water, Tepid/Warm	*3*	*0*	*3*	*6*
12. OTHER[7]	17	4	7	28
Bolus	*2*	*1*	*2*	*5*
Decoction	*2*	*0*	*0*	*2*
Electuary	*1*	*1*	*0*	*2*
Infusion	*3*	*0*	*1*	*4*
Powder	*4*	*1*	*1*	*6*
Miscellaneous	*5*	*1*	*3*	*9*
TOTAL	156	71	59	286

[1] Includes orders to provide the patient with beef tea, red meat, red or white wine, or rice.

[2] Squill was commonly prescribed as an expectorant.

[3] Includes inhalations of aether nitrosis spiritus, aether sulphericus, antimony, gentian, hydrocarbons, senna, sodae carbonis, and vinegar

[4] Includes aloe and senna

[5] Opium enemas were commonly prescribed for diarrhea.

[6] Includes vomit-inducing treatments containing antimony or sulfate.

[7] The treatments in this category include all preparations of the listed types not accounted for under the above classifications.

APPENDIX I

TREATMENTS RECORDED IN SAMPLED CASES

FOR THE PATHOLOGICAL PERIOD I, 1821 – 1840

Treatments	Diagnosis Groups			
	TB-Related	Non-TB Pulmonary	Other	TOTAL
1. ANODYNES	10	3	2	15
Anodyne, Draught	*7*	*2*	*1*	*10*
Anodyne, Enema	*3*	*1*	*1*	*5*
2. ANTACIDS[1]	4	2	5	11
Potassium, Acetate/Carbonate	*1*	*1*	*1*	*3*
Potassium, Hydroxide[2]	*0*	*0*	*1*	*1*
Soda, Bicarbonate/Carbonate	*3*	*1*	*3*	*7*
3. ANTISPASMODICS	16	4	10	30
Amber	*1*	*0*	*0*	*1*
Ammonia	*4*	*1*	*3*	*8*
Assafoetida	*5*	*1*	*5*	*11*
Camphor	*1*	*1*	*0*	*2*
Ether, All Types	*5*	*1*	*1*	*7*
Valerian	*0*	*0*	*1*	*1*
4. ASTRINGENTS	4	2	4	10
Chalk	*2*	*1*	*0*	*3*
Gall, Unguent	*0*	*0*	*1*	*1*
Iron	*0*	*0*	*2*	*2*
Lead	*1*	*1*	*1*	*3*
Rose	*1*	*0*	*0*	*1*
5. BLEEDING	23	23	14	60
Abrasion/Scarification	*1*	*1*	*0*	*2*
Cupping	*14*	*8*	*8*	*30*
Leeches	*5*	*10*	*5*	*20*
Venesection	*3*	*4*	*1*	*8*
6. CATHARTICS[3]	29	24	37	90
Aloe	*0*	*2*	*6*	*8*
Bolus, Purgative	*1*	*1*	*1*	*3*
Castor Oil	*10*	*5*	*9*	*24*
Colocynth	*2*	*4*	*2*	*8*
Draught, Unspecified	*0*	*1*	*0*	*1*
Elaterium	*1*	*0*	*1*	*2*
Enema	*3*	*1*	*3*	*7*
Jalap	*1*	*1*	*1*	*3*
Magnesium	*7*	*7*	*7*	*21*
Potassium Tartrate	*0*	*0*	*1*	*1*
Rhubarb	*4*	*2*	*2*	*8*
Senna	*0*	*0*	*4*	*4*

Appendix I (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
7. CORROSIVES	5	5	4	14
Acid, Nitric	*1*	*0*	*0*	*1*
Acid, Sulphuric	*4*	*3*	*2*	*9*
Silver, Nitrate, Application[4]	*0*	*2*	*2*	*4*
8. DEMULCENTS	8	9	2	19
Mucilage, Gum Arabic	*1*	*0*	*0*	*1*
Mucilage, Unspecified	*7*	*9*	*2*	*18*
9. DIAPHORETICS	9	5	3	17
Antimony, Unguent	*1*	*1*	*0*	*2*
Dover's Powder[5]	*5*	*2*	*2*	*9*
Guaiaci[6]	*0*	*1*	*0*	*1*
Mixture, Unspecified	*1*	*1*	*0*	*2*
Powder, Unspecified	*0*	*0*	*1*	*1*
Wine,[7] *Rectified/Rub*	*2*	*0*	*0*	*2*
10. DIURETICS	13	22	13	48
Cantharis	*4*	*7*	*5*	*16*
Digitalis	*2*	*6*	*2*	*10*
Potassium, Bitartrate[8]	*3*	*4*	*2*	*9*
Potassium, Nitrate[9]	*0*	*3*	*0*	*3*
Potassium, Hydriodic[8]	*0*	*0*	*2*	*2*
Powder, Unspecified	*4*	*2*	*2*	*8*
11. EMETICS	17	10	7	34
Antimony, Various[10]	*8*	*10*	*5*	*23*
Ipecac	*7*	*0*	*2*	*9*
Mixture, Unspecified	*1*	*0*	*0*	*1*
Zinc	*1*	*0*	*0*	*1*
12. EMOLLIENTS	1	1	1	3
Application, Unspecified	*1*	*1*	*1*	*3*
13. EXPECTORANTS	17	6	1	24
Squill	*14*	*6*	*1*	*21*
Balsam, Tolu[11]	*2*	*0*	*0*	*2*
Benzoin[10]	*1*	*0*	*0*	*1*
14. MERCURY[12]	12	14	18	44
External	*0*	*1*	*5*	*6*
Internal	*12*	*13*	*13*	*38*

Appendix I (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
15. OPIUM[13]	36	13	7	56
Internal, Oral	*35*	*13*	*7*	*55*
Internal, Rectal	*1*	*0*	*0*	*1*
16. SEDATIVES	3	1	0	4
Belladonna	*0*	*1*	*0*	*1*
Hyoscyamus	*3*	*0*	*0*	*3*
17. STIMULANTS	18	9	14	41
Cascarilla	*1*	*0*	*1*	*2*
Cassia	*0*	*1*	*1*	*2*
Cinnamon	*2*	*2*	*1*	*5*
Copaiba	*1*	*1*	*0*	*2*
Ginger	*1*	*0*	*1*	*2*
Juniper	*3*	*1*	*2*	*6*
Lavender	*1*	*0*	*0*	*1*
Mint	*0*	*0*	*1*	*1*
Mustard, Plaster	*3*	*3*	*4*	*10*
Myrrh	*3*	*1*	*0*	*4*
Turpentine	*3*	*0*	*3*	*6*
18. TONICS	9	2	6	17
Chamomile	*1*	*1*	*1*	*3*
Gentian	*6*	*1*	*3*	*10*
Quassia	*1*	*0*	*0*	*1*
Quinia, Sulphate	*1*	*0*	*2*	*3*
19. INDETERMINATE[14]	9	1	7	17
Acetate	*1*	*0*	*0*	*1*
Acid[15]	*2*	*0*	*0*	*2*
Alcohol, Barley Spirit	*0*	*0*	*1*	*1*
Draught	*1*	*0*	*1*	*2*
Enema	*0*	*0*	*1*	*1*
Pill	*0*	*0*	*1*	*1*
Potassium	*0*	*0*	*3*	*3*
Powder, Aromatic	*1*	*0*	*0*	*1*
Saline	*1*	*0*	*0*	*1*
Tincture	*2*	*0*	*0*	*2*
Water, Cold/Spring	*1*	*1*	*0*	*2*

Appendix I (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
20. UNCLASSIFIED[16]	19	7	15	41
Aconite	*0*	*1*	*1*	*2*
Application, Bath, Tepid	*2*	*0*	*3*	*5*
Application, Bread Crumb	*1*	*0*	*0*	*1*
Application, Curcurbitaceae	*2*	*0*	*1*	*3*
Application, Gargle, Egg	*1*	*0*	*0*	*1*
Application, Plaster, Various[17]	*2*	*2*	*3*	*7*
Avenae	*0*	*0*	*1*	*1*
Bolus, Common/Unspecified	*3*	*1*	*2*	*6*
Catechu	*1*	*0*	*0*	*1*
Colato	*2*	*0*	*0*	*2*
Creosote	*0*	*1*	*0*	*1*
Cyatho Aquae	*0*	*1*	*0*	*1*
Inhalation, Vinegar	*0*	*1*	*0*	*1*
Iodine	*0*	*0*	*2*	*2*
Olein Cola	*1*	*0*	*0*	*1*
Sarsa	*2*	*0*	*1*	*3*
Vinegar	*2*	*0*	*0*	*2*
Yeast, Brewer's	*0*	*0*	*1*	*1*
TOTAL	262	163	170	595

[1] Categories two through eighteen follow Cullen's (1789) treatments classification, with the exceptions of bleeding, mercury, and opium. Where drugs fell into multiple categories, I estimated the appropriate category from context.

[2] Cullen (1789) also classifies potassium hydroxide as a diuretic.

[3] Cathartics act as laxatives.

[4] Silver nitrate is also classified as an antiseptic.

[5] Dover's powder, while not classified by Cullen (1789), was a commonly prescribed diaphoretic comprising opium and ipecac.

[6] Guaiaci is also classified as a stimulant.

[7] Wine is also classified as a sedative.

[8] Potassium bitartrate is commonly known as cream of tartar.

[9] Potassium nitrate and hydriodic potassium are also classified as antacids.

[10] Includes antimony powder, antimony wine, and tartarated antimony.

[11] Tolu balsam and benzoin are also classified as stimulants.

[12] Mercury is classified as a cathartic, corrosive, diaphoretic, emetic, errhina (nasal decongestant), menagoga (menstruation agent), and sialogoga (salivation agent).

[13] Opium is classified as an antiseptic and diaphoretic, while poppy is classified as a sedative.

[14] Drugs in this category are classified in multiple categories that I was unable to resolve from context, or were not specific or legible enough to allow classification.

[15] Includes a boracic acid gargle and hydrocyanic acid.

[16] Cullen (1789) does not classify the drugs in this category.

[17] Includes poultices and warm and unspecified plasters.

APPENDIX J

TREATMENTS RECORDED IN SAMPLED CASES FOR THE PATHOLOGICAL PERIOD II, 1841 – 1880, AT GLASGOW ROYAL INFIRMARY

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
1. ANODYNES	0	0	0	0
2. ANTACIDS[1]	1	2	6	9
Potassium, Acetate/Carbonate[2]	*1*	*0*	*4*	*5*
Soda, Bicarbonate/Carbonate[3]	*0*	*2*	*2*	*4*
3. ANTISPASMODICS	16	13	6	35
Ammonia[4]	*2*	*3*	*0*	*5*
Assafoetida	*0*	*2*	*3*	*5*
Camphor	*6*	*6*	*1*	*13*
Ether, All Types	*8*	*2*	*2*	*12*
4. ASTRINGENTS	14	2	11	27
Alum[5]	*1*	*0*	*0*	*1*
Bismuth[6]	*1*	*1*	*1*	*3*
Chalk	*2*	*1*	*1*	*4*
Iron	*8*	*0*	*8*	*16*
Kino	*1*	*0*	*0*	*1*
Rose	*0*	*0*	*1*	*1*
Starch, Enema	*1*	*0*	*0*	*1*
5. BLEEDING	1	0	0	1
Leeches	*1*	*0*	*0*	*1*
6. CATHARTICS[7]	14	8	28	50
Aloe	*0*	*1*	*5*	*6*
Castor Oil	*7*	*4*	*8*	*19*
Colocynth	*2*	*0*	*3*	*5*
Elaterium	*0*	*0*	*1*	*1*
Gregory's Powder[8]	*1*	*0*	*0*	*1*
Hellebore	*0*	*0*	*1*	*1*
Jalap	*0*	*0*	*1*	*1*
Magnesium	*1*	*0*	*2*	*3*
Rhubarb	*3*	*1*	*5*	*9*
Senna	*0*	*1*	*2*	*3*
Sulphur, Sublimated	*0*	*1*	*0*	*1*
7. CORROSIVES	9	4	2	15
Acid, Nitric	*3*	*3*	*2*	*8*
Acid, Sulphuric	*6*	*1*	*0*	*7*
8. DEMULCENTS	3	1	0	4
Mucilage, Acacia	*3*	*0*	*0*	*3*
Mucilage, Unspecified	*0*	*1*	*0*	*1*

Appendix J (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
9. DIAPHORETICS	17	13	9	39
Acid, Gallic/Tannic[9]	*2*	*0*	*1*	*3*
Alcohol[10]	*15*	*9*	*7*	*31*
Dover's Powder[11]	*0*	*0*	*1*	*1*
Senega[12]	*0*	*4*	*0*	*4*
10. DIURETICS	1	1	10	12
Cantharis	*1*	*0*	*0*	*1*
Digitalis	*0*	*0*	*6*	*6*
Potassium, Bitartrate	*0*	*1*	*2*	*3*
Potassium, Citrate/Nitrate[13]	*0*	*0*	*2*	*2*
11. EMETICS	8	4	1	13
Antimony, Various[14]	*1*	*2*	*1*	*4*
Ipecac	*7*	*2*	*0*	*9*
12. EMOLLIENTS	0	0	0	0
13. EXPECTORANTS	4	2	2	8
Potassium Iodide[15]	*1*	*0*	*2*	*3*
Squill	*3*	*2*	*0*	*5*
14. MERCURY[16]	3	1	3	7
External[17]	*2*	*0*	*0*	*2*
Internal	*1*	*1*	*3*	*5*
15. OPIUM[18]	27	3	2	32
Internal, Oral[19]	*26*	*3*	*2*	*31*
Internal, Rectal	*1*	*0*	*0*	*1*
16. SEDATIVES	6	2	7	15
Belladonna[20]	*3*	*0*	*2*	*5*
Datura	*0*	*1*	*0*	*1*
Hemlock[21]	*0*	*1*	*2*	*3*
Hyoscyamus	*3*	*1*	*3*	*7*
17. STIMULANTS	23	10	11	44
Cardamom	*0*	*2*	*1*	*3*
Cinnamon	*1*	*0*	*0*	*1*
Easton's Syrup[22]	*1*	*0*	*1*	*2*
Ginger	*3*	*4*	*1*	*8*
Juniper	*1*	*0*	*0*	*1*
Lavender	*1*	*0*	*0*	*1*
Mint	*14*	*1*	*1*	*16*
Mustard, Plaster	*2*	*2*	*1*	*5*

Appendix J (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
17. STIMULANTS, CONT.				
Myrrh	*0*	*0*	*3*	*3*
Nutmeg	*0*	*0*	*1*	*1*
Turpentine	*0*	*1*	*2*	*3*
18. TONICS	21	7	7	35
Calumba[23]	*1*	*0*	*0*	*1*
Dandelion[24]	*0*	*1*	*0*	*1*
Gentian	*5*	*2*	*1*	*8*
Orange[25]	*3*	*0*	*1*	*4*
Quassia	*2*	*1*	*3*	*6*
Quinia, Sulphate	*9*	*3*	*2*	*14*
Strychnine[26]	*1*	*0*	*0*	*1*
19. INDETERMINATE[27]	10	6	7	23
Acid[28]	*0*	*1*	*2*	*3*
Alcohol, Barley Spirit	*9*	*3*	*3*	*15*
Aromatic, Powder	*1*	*1*	*1*	*3*
Draught	*0*	*1*	*0*	*1*
Pill	*0*	*0*	*1*	*1*
20. UNCLASSIFIED[29]	34	12	11	57
Applications	—	—	—	—
Bath, Tepid[30]	*0*	*0*	*2*	*2*
Blister	*0*	*1*	*1*	*2*
Borax Glycerin	*0*	*0*	*1*	*1*
Colloidion, Flexible[31]	*0*	*0*	*1*	*1*
Oil, Olive	*0*	*1*	*0*	*1*
Plaster, Various[32]	*2*	*1*	*1*	*4*
Sulphur	*1*	*0*	*0*	*1*
Unguent, Simple	*0*	*1*	*1*	*2*
Behavioral	*1*	*0*	*0*	*1*
Catechu	*1*	*0*	*0*	*1*
Chlorodyne	*2*	*2*	*0*	*4*
Chloroform	*5*	*2*	*0*	*7*
Cod Liver Oil	*16*	*1*	*1*	*18*
Creosote	*1*	*0*	*0*	*1*
Croton Oil	*0*	*1*	*1*	*2*
Diet, Special[33]	*3*	*0*	*0*	*3*
Egg Yolk	*0*	*1*	*0*	*1*
Lactose	*1*	*0*	*0*	*1*
Lime, Sulphuretted[34]	*1*	*0*	*0*	*1*

Appendix J (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
20. UNCLASSIFIED, CONT.				
Lobelia	*0*	*1*	*0*	*1*
Sodium Salicylate	*0*	*0*	*1*	*1*
Water Bed	*0*	*0*	*1*	*1*
TOTAL	212	91	123	426

[1] Categories two through eighteen follow Cullen's (1789) treatments classification, with the exceptions of bleeding, mercury, and opium. Where drugs fell into multiple categories, I estimated the appropriate category from context.

[2] Includes potassium bicarbonate.

[3] Includes one simply "soda."

[4] Includes ammonium acetate, ammonium carbonate (smelling salts), and acet. sulp. amm. Cullen (1789) also classified ammonia alone as a diaphoretic, while Bartholow (1879) also classified ammonium carbonate as a stimulant and expectorant (173–76).

[5] Cullen (1789) also classifies alum as a refrigerant.

[6] Classification of bismuth as an astringent is based on its use today in antidiarrheals such as Pepto-Bismol.

[7] Cathartics act as laxatives.

[8] Gregory's powder, while not classified by Cullen (1789), was a laxative compound of rhubarb, magnesia, and ginger.

[9] Classification of gallic and tannic acids follows Cullen's (1789) "vegetable acids," as well as "Treatment of Nephritis" (1885, 247).

[10] Includes brandy, port, sherry, whisky, and wine (rectified, rub).

[11] Dover's powder, while not classified by Cullen (1789), was a commonly prescribed diaphoretic comprising opium and ipecac.

[12] Cullen (1789) also classifies senega as a diuretic.

[13] Potassium nitrate is also classified as an antacid; potassium citrate is used as a diuretic today.

[14] Includes antimony powder, antimony wine, and tartarated antimony.

[15] I inferred the classification of potassium iodide from modern pharmaceutical applications.

[16] Mercury is classified as a cathartic, corrosive, diaphoretic, emetic, errhina (nasal decongestant), menagoga (menstruation agent), and sialogoga (salivation agent).

[17] Includes a subchloride unguent I assumed to be mercury subchloride, as well as McKenzie's eye lotion, a mercury-containing application ("McKenzie's Eye-Lotion" 1887, 559).

[18] Opium is classified as an antiseptic and diaphoretic, while poppy is classified as a sedative.

[19] Includes one "nepenthe," a poetic reference to opiate sedatives.

[20] Includes atropine.

[21] Includes cataplasma conii (hemlock poultice).

[22] Easton's syrup was a compound of strychnine, iron phosphate, and quinine.

[23] Classification from *The Edinburgh New Dispensatory* (Duncan 1830, 393).

[24] Classification from *The British Pharmaceutical Codex* (Pharmaceutical Society of Great Britain 1907, 1149).

[25] Classified as a tonic, but most often used as a flavoring agent per *King's American Dispensatory* (Felter and Lloyd 1909, 311).

[26] Classification is based on Cullen's (1789) placement of St. Ignatius' bean as a tonic.

[27] Drugs in this category are classified in multiple categories that I was unable to resolve from context, or were not specific or legible enough to allow classification.

[28] Includes acetic, hydrochloric, and phosphoric acids.

[29] Cullen (1789) does not classify the drugs in this category.

[30] Includes hot bath.

[31] Presumed to be an application rather than prescribed for internal use.

[32] Includes poultices and warm and unspecified plasters.

[33] Includes prescriptions for lemonade, milk, and regulated diet.

[34] Recommended for treating pulmonary tuberculosis by Wells (1893).

APPENDIX K

TREATMENTS RECORDED IN SAMPLED CASES FOR THE PATHOLOGICAL PERIOD II, 1841 – 1880, AT THE ROYAL INFIRMARY OF EDINBURGH

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
1. ANODYNES	1	1	0	2
Anodyne, Enema	*1*	*1*	*0*	*2*
2. ANTACIDS[1]	29	13	12	54
Lime Water/Aqua Calcis	*3*	*1*	*2*	*6*
Potassium, Acetate/Carbonate[2]	*23*	*10*	*10*	*43*
Soda, Bicarbonate/Carbonate[3]	*4*	*3*	*2*	*9*
Unspecified Alkaline[4]	*2*	*0*	*0*	*2*
3. ANTISPASMODICS	183	98	59	340
Ammonia[5]	*52*	*29*	*18*	*99*
Assafoetida[6]	*1*	*2*	*0*	*3*
Camphor	*61*	*22*	*14*	*97*
Ether, All Types	*63*	*44*	*25*	*132*
Valerian	*6*	*1*	*2*	*9*
4. ASTRINGENTS	138	51	216	405
Alum[7]	*1*	*0*	*0*	*1*
Bismuth[8]	*11*	*2*	*6*	*19*
Chalk	*27*	*7*	*6*	*40*
Fern[9]	*0*	*0*	*2*	*2*
Iron	*31*	*6*	*13*	*50*
Kino	*5*	*0*	*1*	*6*
Lead[10]	*33*	*8*	*10*	*51*
Logwood	*3*	*1*	*0*	*4*
Rose	*10*	*6*	*11*	*27*
Starch, Enema	*11*	*3*	*2*	*16*
Zinc[11]	*4*	*0*	*3*	*7*
Unspecified Astringent	*2*	*0*	*0*	*2*
5. ATTENUANTS	7	4	4	15
Licorice	*1*	*0*	*0*	*1*
Soap	*6*	*4*	*4*	*14*
6. BLEEDING	17	17	18	52
Cupping	*3*	*3*	*5*	*11*
Leeches	*12*	*9*	*10*	*31*
Venesection[12]	*2*	*5*	*3*	*10*

Appendix K (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
7. CATHARTICS	68	43	65	176
Aloe	*4*	*2*	*3*	*9*
Castor Oil	*20*	*8*	*20*	*48*
Colocynth	*5*	*6*	*6*	*17*
Elaterium	*1*	*0*	*0*	*1*
Enema	*1*	*2*	*6*	*9*
Gregory's Powder[13]	*0*	*0*	*1*	*1*
Hellebore[14]	*0*	*0*	*2*	*2*
Honey[15]	*2*	*0*	*0*	*2*
Jalap	*2*	*5*	*2*	*9*
Magnesium	*12*	*1*	*6*	*19*
Rhubarb	*7*	*8*	*6*	*21*
Scammony	*2*	*2*	*1*	*5*
Senna	*1*	*1*	*4*	*6*
Sulphur	*4*	*3*	*0*	*7*
Unspecified Cathartic[16]	*7*	*8*	*8*	*23*
8. CORROSIVES	51	12	21	84
Acid, Nitric	*3*	*2*	*6*	*11*
Acid, Sulphuric	*31*	*7*	*7*	*45*
Arsenic	*1*	*0*	*4*	*5*
Silver Nitrate, Application	*16*	*3*	*4*	*23*
9. DEMULCENTS	14	9	3	26
Gum Tragacanth	*0*	*0*	*1*	*1*
Mucilage, Acacia	*2*	*0*	*0*	*2*
Mucilage, Unspecified	*12*	*9*	*3*	*24*
10. DIAPHORETICS	132	64	58	254
Acid, Vegetabilis[17]	*13*	*4*	*3*	*20*
Alcohol[18]	*92*	*31*	*40*	*163*
Crocus[19]	*2*	*5*	*3*	*10*
Dover's Powder	*7*	*4*	*3*	*14*
Senega[20]	*17*	*20*	*7*	*44*
Unspecified Diaphoretic	*1*	*0*	*2*	*3*
11. DIURETICS	18	11	30	59
Broom	*1*	*0*	*2*	*3*
Cantharis[21]	*1*	*0*	*5*	*6*
Digitalis	*8*	*6*	*11*	*25*
Potassium, Bitartrate	*6*	*4*	*9*	*19*
Potassium, Citrate/Nitrate[22]	*2*	*1*	*3*	*6*

Appendix K (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
12. EMETICS	63	45	21	129
Antimony, Various[23]	*29*	*22*	*10*	*61*
Horseradish	*1*	*0*	*0*	*1*
Ipecac	*22*	*23*	*11*	*56*
Zinc	*11*	*0*	*0*	*11*
13. EMOLLIENTS	1	0	1	2
Linseed	*1*	*0*	*1*	*2*
14. EXPECTORANTS	39	26	32	97
Balsam, Tolu	*1*	*1*	*0*	*2*
Benzoin	*1*	*0*	*0*	*1*
Potassium Iodide[24]	*12*	*2*	*14*	*28*
Squill	*25*	*23*	*18*	*66*
Unspecified Expectorant[25]	*4*	*2*	*0*	*6*
15. MERCURY[26]	13	13	16	42
External[27]	*2*	*0*	*1*	*3*
Internal	*11*	*13*	*15*	*39*
16. OPIUM[28]	127	71	55	253
Internal, Oral[29]	*109*	*66*	*47*	*222*
Internal, Rectal	*18*	*5*	*8*	*31*
External/Other[30]	*7*	*8*	*7*	*22*
17. SEDATIVES	24	10	13	47
Belladonna[31]	*1*	*0*	*2*	*3*
Datura	*0*	*1*	*1*	*2*
Hyoscyamus	*23*	*10*	*10*	*43*
Potassium Bromide[32]	*1*	*1*	*4*	*6*
18. STIMULANTS	98	44	29	171
Capsicum	*1*	*0*	*0*	*1*
Cardamom	*27*	*3*	*3*	*33*
Cassia	*5*	*3*	*1*	*9*
Cinnamon[33]	*20*	*7*	*5*	*32*
Clove	*3*	*0*	*0*	*3*
Copaiba	*1*	*2*	*0*	*3*
Dill	*1*	*0*	*0*	*1*
Ginger	*6*	*2*	*3*	*11*
Juniper	*0*	*2*	*2*	*4*
Lavender	*2*	*2*	*4*	*8*
Mint	*11*	*4*	*2*	*17*
Mustard, Plaster	*12*	*7*	*5*	*24*

Appendix K (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
18. STIMULANTS, CONT.				
Myrrh	*2*	*1*	*0*	*3*
Turpentine	*6*	*10*	*4*	*20*
Unspecified Stimulant	*1*	*1*	*0*	*2*
19. TONICS	101	25	38	164
Calumba[34]	*20*	*4*	*7*	*31*
Chamomile	*1*	*0*	*0*	*1*
Dandelion[35]	*2*	*0*	*3*	*5*
Gentian	*24*	*5*	*5*	*34*
Orange[36]	*16*	*3*	*3*	*22*
Quassia	*20*	*1*	*8*	*29*
Quinia[37]	*13*	*7*	*9*	*29*
Strychnine[38]	*5*	*5*	*3*	*13*
20. INDETERMINATE[39]	73	16	41	130
Acid	*18*	*6*	*13*	*37*
Application[40]	*3*	*1*	*5*	*9*
Aromatic, Powder[41]	*10*	*5*	*5*	*20*
Botanical	*10*	*1*	*6*	*17*
Chemical	*7*	*1*	*3*	*11*
Enema[42]	*5*	*0*	*2*	*7*
Patent Formulations[43]	*0*	*0*	*2*	*2*
Potassium	*16*	*1*	*3*	*20*
Saline[44]	*4*	*1*	*2*	*7*
21. UNCLASSIFIED[45]	493	153	162	808
Aconite	*0*	*3*	*2*	*5*
Applications	—	—	—	—
Bath, Tepid[46]	*3*	*1*	*9*	*13*
Blister	*51*	*34*	*16*	*101*
Cold[47]	*9*	*1*	*7*	*17*
Colloidion, Flexible	*0*	*1*	*0*	*1*
Curcurbitaceae	*0*	*1*	*0*	*1*
Dry Cupping	*2*	*2*	*5*	*9*
Gargle	*4*	*4*	*4*	*12*
Oil, Olive	*1*	*1*	*1*	*3*
Plaster, Various[48]	*33*	*15*	*25*	*73*
Sulphur[49]	*5*	*1*	*2*	*8*

Appendix K (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
21. UNCLASSIFIED, CONT.				
Aluminis	*4*	*2*	*0*	*6*
Behavioral[50]	*2*	*0*	*1*	*3*
Belae	*0*	*1*	*1*	*2*
Cannabis	*2*	*0*	*1*	*3*
Catechu	*22*	*7*	*4*	*33*
Chlorodyne	*18*	*5*	*2*	*25*
Chloroform	*6*	*7*	*2*	*15*
Cod Liver Oil	*103*	*5*	*2*	*110*
Creosote	*5*	*1*	*1*	*7*
Croton Oil	*3*	*5*	*2*	*10*
Diet, Special[51]	*161*	*46*	*46*	*253*
Egg	*3*	*0*	*1*	*4*
Ergot	*4*	*0*	*1*	*5*
Head Shaved	*2*	*1*	*3*	*6*
Inhalation	*1*	*0*	*11*	*2*
Iodine	*11*	*3*	*8*	*22*
Lobelia	*1*	*0*	*0*	*1*
Naphtha	*25*	*1*	*2*	*28*
Surgical Intervention[52]	*9*	*4*	*10*	*23*
Tannin	*3*	*1*	*2*	*6*
Water Bed	*0*	*0*	*1*	*1*
TOTAL	1686	723	892	3301

[1] Categories two through eighteen follow Cullen's (1789) treatments classification, with the exceptions of bleeding, mercury, and opium. Where drugs fell into multiple categories, I estimated the appropriate category from context.

[2] Includes potassium bicarbonate.

[3] Includes two alkaline baths containing carbonate of soda.

[4] Includes alkaline tonic and alkaline lotion.

[5] Includes ammonium carbonate (smelling salts). Cullen (1789) also classified ammonia alone as a diaphoretic, while Bartholow (1879) also classified ammonium carbonate as a stimulant and expectorant (173–76).

[6] Includes one enema foetidum.

[7] Cullen (1789) also classifies alum as a refrigerant.

[8] Classification of bismuth as an astringent is based on its use today in antidiarrheals such as Pepto-Bismol.

[9] Includes shield fern, based on Cullen's (1789) inclusion of other ferns as astringents.

[10] Includes a lead acetate application.

[11] Includes all topical uses of zinc; I classified zinc used internally as an emetic.

[12] Includes one bleeding specified only as a volume of six ounces.

[13] Gregory's powder, while not classified by Cullen (1789), was a laxative compound of rhubarb, magnesia, and ginger.

[14] White hellebore is also classified as an errhina by Cullen (1789).

[15] Cullen (1789) also classifies honey as an antiseptic and attenuant.

[16] Includes cathartic/laxative electuary, enema, powder, pill, and simple/warm water/soap and water enema.

[17] Includes gallic acid, tannic acid, and vegetable acid.

[18] Cullen (1789) also classifies alcohol as a sedative.

[19] Cullen (1789) also classifies crocus as an antiseptic, diaphoretic, menagogue, and sedative.

[20] Includes black cohosh/snakeroot and serpentariae.

[21] Includes lyttae.

[22] Potassium nitrate is also classified as an antacid; potassium citrate is used as a diuretic today.

[23] Includes antimony powder, antimony wine, and tartarated antimony, as well as one tartar emetii.

[24] I inferred the classification of potassium iodide from modern pharmaceutical applications; as it was prescribed as an application in four cases, it may not have been used as an expectorant exclusively during the nineteenth century.

[25] These were probably a squill and opium mixture.

[26] Mercury is classified as a cathartic, corrosive, diaphoretic, emetic, errhina (nasal decongestant), menagoga (menstruation agent), and sialogoga (salivation agent).

[27] Includes a subchloride unguent assumed to be mercury subchloride, McKenzie's eye lotion, a mercury-containing application ("McKenzie's Eye-Lotion" 1887, 559).

[28] Opium is classified as an antiseptic and diaphoretic, while poppy is classified as a sedative.

[29] Includes codeias (codeine) as well as the opiate-based Battely's solution.

[30] Includes inhalation and non-rectal injection.

[31] Includes belladonna prescribed as an application.

[32] Potassium bromide was not classified by Cullen (1789), but was used widely as a sedative during this time period.

[33] Cinnamon may be prescribed largely to improve the palatability of cod liver oil.

[34] Classification from *The Edinburgh New Dispensatory* (Duncan 1830, 393).

[35] Classification from *The British Pharmaceutical Codex* (Pharmaceutical Society of Great Britain 1907, 1149).

[36] Classified as a tonic, but most often used as a flavoring agent per *King's American Dispensatory* (Felter and Lloyd 1909, 311).

[37] Includes cinchon.

[38] Classification is based on Cullen's (1789) placement of St. Ignatius' bean as a tonic.

[39] Drugs in this category are classified in multiple categories that I was unable to resolve from context, or were not specific or legible enough to allow classification.

[40] Includes flour, picis/pitch/tar unguent, red lotion, and illegible applications.

[41] Includes aromatic confectiones.

[42] Includes beef tea, brandy, glycerin, sherry, and vitelli ovi.

[43] Includes Kaufsons and Murray's specific.

[44] Includes effervescing salts and dose of salts, the latter likely a laxative.

[45] Cullen (1789) does not classify the drugs in this category.

[46] Includes a patient to be well washed for lice.

[47] Includes applications of ice, evaporating lotion, and spirit.

[48] Includes linseed poultice and hot air bath.

[49] Includes copper sulphate and sulphur collyrium.

[50] Includes rest and frequent walks in open air.

[51] Includes prescriptions for American beans, arrowroot, beef/steak, beef tea, bread, bread pudding, butter, cabbage, chicken/chicken soup, coffee, cooling drinks, currant jam, egg, fat bacon, fish, lemon juice, lemonade, marmalade, milk, milk pudding, mutton, porridge, rice, rice pudding, soda water, soup, and toast, as well as diets described as good/full, farinaceous, increased vegetable, mild, nourishing, low, mixed, and without meat.

[52] Includes acupuncture, adhesive plaster, bandaging, catheter, faradization, galvinization, hot iron (possibly a feigned prescription), surgical draining, tracheotomy, and water dressing.

APPENDIX L

TREATMENTS RECORDED IN SAMPLED CASES

FOR THE BACTERIOLOGICAL PERIOD, 1881 – 1905

Treatments	Diagnosis Groups			
	TB-Related	Non-TB Pulmonary	Other	TOTAL
1. ANODYNES	—	—	—	—
2. ANTACIDS[1]	12	6	6	24
Potassium, Acetate/Carbonate[2]	*6*	*5*	*2*	*13*
Soda, Bicarbonate/Carbonate[3]	*6*	*1*	*3*	*10*
Unspecified Alkaline[4]	*0*	*0*	*1*	*1*
3. ANTISPASMODICS	36	36	18	90
Ammonia[5]	*21*	*21*	*7*	*49*
Camphor	*11*	*6*	*2*	*19*
Ether, All Types	*4*	*7*	*8*	*19*
Musk[6]	*0*	*2*	*0*	*2*
Savin	*0*	*0*	*1*	*1*
4. ASTRINGENTS	34	17	63	114
Alum[7]	*1*	*0*	*0*	*1*
Bismuth[8]	*5*	*0*	*7*	*12*
Chalk	*2*	*0*	*0*	*2*
Iron	*18*	*13*	*9*	*40*
Kino	*1*	*0*	*0*	*1*
Lead[9]	*4*	*1*	*0*	*5*
Logwood	*1*	*0*	*1*	*2*
Zinc[10]	*1*	*0*	*0*	*1*
Unspecified Astringent	*1*	*0*	*0*	*1*
5. ATTENUANTS	7	4	4	15
6. BLEEDING	0	2	0	2
Leeches	*0*	*2*	*0*	*2*
7. CATHARTICS	8	8	10	26
Castor Oil	*0*	*0*	*1*	*1*
Colocynth	*0*	*3*	*0*	*3*
Enema	*1*	*0*	*3*	*4*
Jalap	*2*	*2*	*2*	*6*
Magnesium	*3*	*0*	*0*	*3*
Mayapple[11]	*0*	*0*	*1*	*1*
Potassium Tartrate	*0*	*1*	*0*	*1*
Rhubarb	*2*	*0*	*0*	*2*
Unspecified Cathartic[12]	*0*	*2*	*3*	*5*

Appendix L (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
8. CORROSIVES	25	11	13	49
Acid, Nitric	*7*	*4*	*4*	*15*
Acid, Sulphuric	*12*	*2*	*2*	*16*
Arsenic	*5*	*5*	*5*	*15*
Silver Nitrate, Application	*1*	*0*	*2*	*3*
9. DEMULCENTS	5	3	0	8
Mucilage, Acacia	*5*	*1*	*0*	*6*
Mucilage, Unspecified[13]	*0*	*2*	*0*	*2*
10. DIAPHORETICS	20	23	10	53
Acid, Vegetabilis[14]	*0*	*0*	*1*	*1*
Alcohol[15]	*12*	*15*	*8*	*35*
Antimony, Unguent	*0*	*1*	*0*	*1*
Senega[16]	*8*	*7*	*1*	*16*
11. DIURETICS	8	21	15	44
Broom	*0*	*0*	*3*	*3*
Cantharis[17]	*1*	*0*	*0*	*1*
Digitalis	*2*	*11*	*7*	*20*
Potassium, Bitartrate[18]	*2*	*1*	*0*	*3*
Potassium, Citrate/Nitrate[19]	*1*	*1*	*3*	*5*
Strophanthus[20]	*1*	*4*	*2*	*7*
Unspecified	*1*	*4*	*0*	*5*
12. EMETICS	13	10	1	24
Antimony, Various[21]	*1*	*0*	*0*	*1*
Horseradish	*12*	*10*	*1*	*23*
13. EMOLLIENTS	3	2	0	5
Linseed	*3*	*2*	*0*	*5*
14. EXPECTORANTS	27	13	13	53
Balsam, Tolu	*3*	*1*	*0*	*4*
Benzoin	*4*	*0*	*1*	*5*
Potassium Iodide[22]	*9*	*6*	*11*	*26*
Squill	*10*	*2*	*1*	*13*
Unspecified Expectorant	*1*	*4*	*0*	*5*
15. MERCURY[23]	8	3	8	19
External[24]	*1*	*0*	*2*	*3*
Internal[25]	*7*	*3*	*6*	*16*

Appendix L (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
16. OPIUM[26]	21	8	7	36
Internal, Oral[27]	*19*	*8*	*7*	*34*
Internal, Rectal	*1*	*0*	*0*	*1*
External/Other	*1*	*0*	*0*	*1*
17. REFRIGERANT	2	0	0	2
Borax[28]	*2*	*0*	*0*	*2*
18. SEDATIVES	15	8	11	34
Belladonna[29]	*9*	*1*	*3*	*13*
Hyoscyamus	*1*	*2*	*0*	*3*
Potassium Bromide[30]	*4*	*2*	*8*	*14*
Sulphonal[31]	*1*	*0*	*0*	*1*
Trional[32]	*0*	*2*	*0*	*2*
Unspecified Sedative	*0*	*1*	*0*	*1*
19. STIMULANTS	32	9	11	52
Capsicum	*2*	*0*	*1*	*3*
Cardamom	*3*	*0*	*1*	*4*
Cascarilla	*1*	*0*	*1*	*2*
Cassia	*0*	*0*	*1*	*1*
Easton's Syrup	*7*	*2*	*2*	*11*
Ginger	*1*	*1*	*1*	*3*
Juniper	*0*	*0*	*1*	*1*
Mint[33]	*8*	*0*	*1*	*9*
Mustard, Plaster	*7*	*3*	*2*	*12*
Turpentine	*3*	*3*	*0*	*6*
20. TONICS	62	36	30	128
Calumba[34]	*6*	*1*	*2*	*9*
Cherry, Wild[35]	*0*	*1*	*0*	*1*
Dandelion[36]	*0*	*0*	*1*	*1*
Gentian	*8*	*3*	*2*	*13*
Hops[37]	*0*	*0*	*1*	*1*
Orange[38]	*1*	*6*	*1*	*8*
Phosphorus[39]	*13*	*1*	*3*	*17*
Quassia	*9*	*4*	*6*	*19*
Quinia[40]	*13*	*7*	*8*	*28*
Strychnine[41]	*12*	*13*	*6*	*31*

Appendix L (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
21. INDETERMINATE[42]	68	29	23	120
Acid	*5*	*2*	*4*	*11*
Aromatic, Powder	*2*	*0*	*0*	*2*
Botanical	*23*	*11*	*8*	*42*
Chemical	*11*	*8*	*1*	*20*
Enema[43]	*3*	*0*	*3*	*6*
Patent Formulations[44]	*12*	*5*	*5*	*22*
Pill	*1*	*0*	*0*	*1*
Potassium	*1*	*1*	*0*	*2*
Powder	*1*	*0*	*0*	*1*
Saline[45]	*1*	*2*	*2*	*5*
Water, Cold[46]	*2*	*0*	*0*	*2*
Other	*4*	*0*	*0*	*4*
22. UNCLASSIFIED[47]	192	89	96	377
Aconite	*1*	*1*	*0*	*2*
Antifebrin[48]	*5*	*0*	*2*	*7*
Applications	—	—	—	—
Bath, Tepid[49]	*0*	*1*	*1*	*2*
Blister[50]	*1*	*1*	*5*	*7*
Cold[51]	*2*	*3*	*1*	*6*
Colloidion, Flexible	*1*	*0*	*0*	*1*
Gargle[52]	*0*	*2*	*0*	*2*
Oil, Olive	*2*	*1*	*1*	*4*
Plaster, Various[53]	*7*	*9*	*5*	*21*
Other	*3*	*2*	*1*	*6*
Behavioral[54]	*14*	*10*	*9*	*33*
Catechu	*2*	*0*	*0*	*2*
Chloral[55]	*2*	*1*	*3*	*6*
Chlorodyne[56]	*1*	*3*	*0*	*4*
Chloroform	*25*	*17*	*7*	*49*
Cod Liver Oil[57]	*19*	*2*	*0*	*21*
Creosote[58]	*16*	*0*	*2*	*18*
Croton Oil	*3*	*0*	*1*	*4*
Diet, Special[59]	*51*	*16*	*25*	*98*
Ergot	*7*	*1*	*0*	*8*
Head Shaved	*0*	*0*	*3*	*3*
Inhalation[60]	*5*	*1*	*2*	*8*
Iodine	*6*	*4*	*0*	*10*

Appendix L (*continued*)

Treatments	Diagnosis Groups: TB-Related	Non-TB Pulmonary	Other	TOTAL
22. UNCLASSIFIED, CONT.				
Pepsin	*0*	*0*	*4*	*4*
Salicylate[61]	*2*	*4*	*5*	*11*
Surgical Intervention[62]	*17*	*9*	*17*	*43*
Vinegar	*0*	*1*	*0*	*1*
Water Bed	*0*	*0*	*2*	*2*
TOTAL	589	334	335	1258

[1] Categories two through eighteen follow Cullen's (1789) treatments classification, with the exceptions of bleeding, mercury, and opium. Where drugs fell into multiple categories, I estimated the appropriate category from context.

[2] Includes potassium bicarbonate.

[3] Includes two simply noting "soda" among the patients' prescriptions.

[4] Includes alkaline lotion.

[5] Includes ammonium acetate, bromide, carbonate (smelling salts), chloride, citrate, sulfate, and aromatic ammonia. Cullen (1789) also classified ammonia alone as a diaphoretic, while Bartholow (1879) also classified ammonium carbonate as a stimulant and expectorant (173 – 76).

[6] Includes moschi and musk enema.

[7] Cullen (1789) also classifies alum as a refrigerant.

[8] Classification of bismuth as an astringent is based on its use today in antidiarrheals such as Pepto-Bismol.

[9] Includes a lead acetate application.

[10] Includes all topical uses of zinc; I classified zinc used internally as an emetic.

[11] Classification following Butler (1908, 494–95).

[12] Includes black draught, saline purgative, and unspecified aperient/purgative.

[13] Includes mucilage enema.

[14] Includes citric acid.

[15] Cullen (1789) also classifies alcohol as a sedative.

[16] Cullen (1789) also classifies senega as a diuretic.

[17] Includes epispasticus.

[18] Includes potus imperialis, comprising cream of tartar, lemon peel, sugar, and water.

[19] Potassium nitrate is also classified as an antacid; potassium citrate is used as a diuretic today.

[20] I inferred the classification of strophanthus from its similar action to digitalis according to Butler (1908, 257).

[21] Includes antimony powder, antimony wine, and tartarated antimony.

[22] I inferred the classification of potassium iodide from modern pharmaceutical applications.

[23] Mercury is classified by Cullen (1789) as a cathartic, corrosive, diaphoretic, emetic, errhina (nasal decongestant), menagoga (menstruation agent), and sialogoga (salivation agent).

[24] Includes Scott's dressing, a mercury-containing application.

[25] Includes one case in which calomel was prescribed as a laxative and one case in which I inferred the "Grey powder" prescribed to be a mercurial preparation.

[26] Opium is classified by Cullen (1789) as an antiseptic and diaphoretic, while poppy is classified as a sedative.

[27] Includes nepenthe and codeia.

[28] Includes boracic ear wash.

[29] Includes belladonna prescribed as an application and atropine.

[30] Potassium bromide was not classified by Cullen (1789), but was used widely as a sedative during this time period. Occurrences include sodium bromide and bromidia.

[31] Classification from Butler (1908, 168).

[32] Classification from Butler (1908, 170).

[33] Includes inhalation and intralaryngeal injection of menthol.

[34] Classification from *The Edinburgh New Dispensatory* (Duncan 1830, 393).

[35] Classification from Butler (1908, 452).

[36] Classification from *The British Pharmaceutical Codex* (Pharmaceutical Society of Great Britain 1907, 1149).

[37] Includes lupulin application.

[38] Classified as a tonic, but most often used as a flavoring agent per *King's American Dispensatory* (Felter and Lloyd 1909, 311).

[39] Classification from Butler (1908, 386). Includes Parrish's Syrup and Fellowe's Syrup, patent preparations containing phosphorus.

[40] Includes cinchon.

[41] Classification is based on Cullen's (1789) placement of St. Ignatius' bean as a tonic. Includes one case for which I assumed the "nux" prescribed to be nux vomica.

[42] Drugs in this category are classified in multiple categories that I was unable to resolve from context, or were not specific or legible enough to allow classification.

[43] Includes meat suppository, nutrient, peptonized milk, and whisky.

[44] Includes Angier's Petroleum Emulsion, Buchan's Infusion, Carlsbad Salts, Carrick's peptenoids, Colman's Wine, Grey's and/or Guy's Pill, and Hamilton's Pills.

[45] Includes salts.

[46] Includes ice to suck.

[47] Cullen (1789) does not classify the drugs in this category.

[48] Includes exalgine, per Butler (1908, 366–70).

[49] Includes hot bath and vapor bath.

[50] Includes mesotan and methyl salicylate (oil of wintergreen).

[51] Includes applications of ice.

[52] Includes mouth washed out and mouthwash.

[53] Includes chest strapping, fomentation, heat application, jacket, and splint.

[54] Includes bed rest, kept awake, and recommendations to go to the country.

[55] Includes butyl chloral hydrate and chloral hydrate.

[56] Chlorodyne was a patent formulation comprising chloroform, cannabis and laudanum.

[57] Includes Scott's Emulsion, a cod liver oil formulation still available today.

[58] Includes guiacol.

[59] Includes prescriptions for arrowroot, barley water, beef tea, Brand's Essence, chemical food, cold fluid, lime water, liquor carnis, malt, malt extract, milk, peptonized milk, small meals, soda water, soup, and Valentine's or Wyatt's meat juice, as well as diabetic, farinaceous, fluid, light, light/low ordinary diets and those comprising specifically detailed menu items.

[60] Includes amyl nitrate, nebulizer "comp n'o 1", oxygen, pine, and illegible inhalations.

[61] Includes aspirin and salicylic acid.

[62] Includes catheter, light treatment, patient between sandbags, and stomach wash/lavage, as well as other surgical interventions.

APPENDIX M

COMPARISON OF STATISTICAL SIGNIFICANCE ACROSS SYMPTOM CATEGORIES, PERIODS, AND INSTITUTIONS

Significance for Fisher exact tests of differences in signs and symptoms among diagnosis categories. Significance at the level of $p < 0.05$ is denoted "+", near-significance ($p < 0.10$) "±", and non-significance ($p \geq 0.10$) "–". See tables 3.3, 4.3, 6.2, 6.3, and 7.2 for exact P-values and appendices B through F for complete listings of signs and symptoms comprising each category.

	GRI								RIE	
	1794 – 1820		1821 – 1840		1841 – 1880		1881 – 1905		1841 – 1880	
	Pulm	Other	Pulm	Other	Pulm	Other	Pulm	Other	Pulm	Other
1. Appearance and Comportment	–	+	+	+	+	+	+	+	+	+
2. Circulatory	–	+	+	–	+	+	+	+	–	+
3. Dermatological	–	+	±	+	+	+	+	+	+	+
4. Digestive	–	–	+	+	+	+	–	+	–	+
5. General	–	–	–	–	–	±	–	+	–	–
6. Genitourinary	–	–	–	–	+	+	–	+	+	+
7. History, Medical	+[1]	+[1]	+[1]	+[1]	+	+	+	+	±	+
8. History, Personal					+	–	+	–	+	+
9. Mouth and Throat	–	–	–	±	–	–	–	–	–	+
10. Pain	–	+	–	+	+	+	+	+	–	+
11. Neurological	N/A		–	–	–	–	+	+	–	–
12. Respiratory, General	±[2]	±[2]	+	–	+	–	+	+	+	+
13. Respiratory, Auscultation/Percussion			+	–	+	–	+	±	+	+
14. Respiratory, Chest Form/Sensation			–	–	+	–	–	±	+	+
15. Miscellaneous	–	+	–	+	–	+	+	+	+	+

[1] I recorded medical and personal history under a single category prior to 1841.

[2] I recorded all respiratory signs and symptoms under a single category prior to 1821

APPENDIX N

COMPARISON OF STATISTICAL SIGNIFICANCE ACROSS TREATMENT CATEGORIES, PERIODS, AND INSTITUTIONS

Summary of significance for two-tailed Fisher exact tests of differences in treatments recorded in cases diagnosed as TB-related disorders vs. non-TB pulmonary disorders ("Pulm") and TB-related disorders vs. non-TB, non-pulmonary disorders ("Other"). Significance at the level of $p < 0.05$ is denoted "+", near-significance ($p < 0.10$) "±", and non-significance ($p \geq 0.10$) "–". The period 1794 – 1820 at GRI is not included here, as treatments were not classifiable by the same scheme as later periods: see table 3.4 for the period 1794 – 1820. See tables 4.5, 6.7, 6.8, and 7.9 for exact P-values and appendices G through K for complete listings of treatments comprising each category.

	GRI						RIE	
	1821 – 1840		1841 – 1880		1881 – 1905		1841 – 1880	
	Pulm	Other	Pulm	Other	Pulm	Other	Pulm	Other
1. Anodyne	–	–	N/A		N/A		N/A	
2. Antacid	–	–	–	–	–	–	–	–
3. Antispasmodic	–	–	–	+	–	+	–	–
4. Astringent	–	–	–	–	–	–	–	±
5. Attenuant	N/A		N/A		N/A		–	–
6. Bleeding	–	–	–	–	N/A		–	–
7. Cathartic	–	–	–	–	+	±	–	–
8. Corrosive	–	–	–	–	–	–	–	+
9. Demulcent	–	–	–	–	–	–	–	–
10. Diaphoretic	–	–	+	–	–	±	+	–
11. Diuretic	–	–	–	±	–	–	–	–
12. Emetic	+	–	–	–	–	–	+	–
13. Emollient	N/A		N/A		N/A		N/A	
14. Expectorant	–	–	–	–	±	+	–	–
15. Mercury	–	±	–	–	–	–	–	–
16. Opium	–	–	–	–	–	–	–	–
17. Refrigerant	N/A		N/A		N/A		N/A	
18. Sedative	–	–	–	–	+	±	–	+
19. Stimulant	–	–	+	+	–	–	+	±
20. Tonic	–	–	–	–	+	–	±	–
21. Indeterminate	–	–	–	±	–	–	–	–
22. Unclassified	–	–	+	+	+	+	+	+