Mortality in Victorian Asylums, ca. 1870-1910.

The causes, processes and monitoring of lunatic death, with a focus on post-mortems

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Alois Alzheimer (1864-1915)
(Courtesy of the Alzheimer Disease Society)

The doyen of psychiatric pathologists. His work was and is paradigmatic for all those who strive in this field.
Abstract

This thesis examines causes of death from post-mortem records, an underused resource, in two asylums (the Royal Montrose Asylum and the Berkshire Asylum) in the period 1870-1905 to investigate their consistently high mortality. The Lunacy Commission recommended that asylum post-mortems should be carried out frequently to provide oversight of this mortality but also to encourage pathological research. This study examines the role of the asylum post-mortems in clarifying the causes of mortality while exploring their wider significance in elucidating discourses on theories of insanity.

In a study of more than 600 post-mortem derived causes of death, it was fruitful to look at conditions such as mania, melancholia, dementia, imbecility and epilepsy separately. Death in insanity was more likely to occur in the early weeks of admission, often the first attack, but was not directly linked to overcrowding or malnutrition. Evidence is accrued to show that the severity of the mental illness, and neither suicide nor violence, was the key driver of premature death. Although rare, cases where violence against patients was evident provide a window into a cover up culture. About 60% of insane patients died from infections (40% from tuberculosis). The novel finding of atheroma in vessels, particularly those of the heart, was made in many of the insane, especially frequently in those who died with exhaustion, which accounted for 15% of deaths. Vascular disease was linked to pathology in the brain. The presence of vessel atheroma raises issues about the role of stress and adversity in these patients’ reduced longevity.

This thesis contends that these routine post-mortems served limited clinical or academic purpose and played little part in reducing asylum violence. However, post-mortems helped to reduce opprobrium for staff in cases where patients had choked to death. Other positives included enhancing scientific benchwork and raising the prospects of pathological insights into insanity.
Acknowledgements

I must first thank my supervisors, Dr Jonathan Andrews and Professor Jeremy Boulton. I have been exceedingly fortunate to have been able to carry out this work under the guidance of Jonathan. He first suggested the topic. It was, and is, an exciting one for me, marrying as it does a topic which is of longstanding personal interest and importance with a relatively unexplored area of medical history. Jonathan is a world expert on nineteenth-century asylums and this expertise is set within a deep appreciation of the social history of the era and a detailed understanding of the processes of history. His knowledge is, thankfully for me, encyclopedic but, as if that was not enough, he has been meticulous, detailed, and comprehensive in his supervision and revision of my early, stuttering attempts at historical writing. He has been encouraging throughout suggesting that I should attend meetings, get to know the topic from several vantage points and speak with my own “voice.” I thank him for stimulating me and this encouragement is a big part of my increasing involvement with and growing enjoyment of the history of medicine in general and the history of psychiatry in particular. Jeremy has also been very supportive and his wry comments have contained much wisdom. From my perspective, both my supervisors have shown great patience and fortitude! The Covid pandemic has had a major impact on all our lives. In my case, it truncated my archive explorations but more importantly robbed me of some of the student experience and the vividness of face-to-face interactions. However, my supervisors have done their very best to counter these drawbacks through the auspices of Zoom!

My family also deserve my great gratitude. My wife, Viv, has been wonderfully supportive and also a bastion of sense and perspective. There must have been a simple answer to some of my problems and questions, but she refrained from giving it! I must thank her particularly for putting up with my absences at archives etc. and in particular for her grandchild care duties she then carried out alone. But then, she has been covering my back for a long time... My children have always been bemused by my antics and peccadillos, but they have all been very supportive and encouraging of this latest venture. In a way that I can’t really describe, this work is for them but also for my late parents. My mother whom I adored and my father whom I revered both taught me to love life and appreciate its rich history. I hope all the family like it and that they share my sense of pride.
I thank the staff at the Tayside Archives at Dundee for their heroic efforts in finding me the right material. In particular, I greatly appreciate the help of Dr Jan Merchant, Sharon Kelly and Dr Kenneth Baxter. They were unfailingly helpful, encouraging and amusingly down to earth. Similarly, I’d like to thank Dr Mark Stevens and all the staff at the Berkshire Records Office for their quiet efficiency and understanding.

There are several other people who I must mention. First among these is Michelle Miller, from the University IT Department, whose unfailing patience and amazing expertise saved me from my format woes. And all done with a smile! I had invaluable help from Ian Wheeler, local historian extraordinaire, particularly with his detailed knowledge of Fair Mile Asylum but also his support with images and asylum plans and his introduction to Tony Spackman. Tony very generously sent me several images from his Fair Mile collection. Sandra Fletcher from the History Department has been a great help. She gave me, gives everyone, a feeling that she is on your side. I have also had confidence building from Claire Hilton and Alan Beveridge, two psychiatrists turned medical historians who are as excellent at the latter as they were at the former. My friends Kenny Abernethy and Alan Foulis have both provided help and reassurance.

So, lots of people to thank for me getting to this point. However, all the analysis and most of the ideas, warts and all, are my own. I have worked in, or been closely involved with, psychiatry for 45 years. During that time, many of the nicest and most decent people I have encountered have been psychiatric patients. I have no doubt those epithets apply equally to the patients studied here and this work is dedicated to them.
## Table of Contents

List of Tables ........................................................................................................................................... xi

List of Figures ............................................................................................................................................ xiii

Abbreviations and Glossary ................................................................................................................. xiv

Chapter 1. Introduction ......................................................................................................................... 1

1.1 Overview of research questions and main arguments .......................................................... 1

1.2 Methods, data collection and associated methodological issues ........................................ 12

   1.2.1 General comments ....................................................................................................................... 12

   1.2.2 The asylums studied ....................................................................................................................... 13

   1.2.3 Records examined ........................................................................................................................ 20

   1.2.4 The periods studied ......................................................................................................................... 30

   1.2.5 Sample sizes ................................................................................................................................. 31

   1.2.6 Control populations ....................................................................................................................... 32

1.3 Synopsis and main lines of enquiry ......................................................................................... 33


2.1 Introduction .......................................................................................................................................... 35

   2.1.1 Mortality in insanity and the growth of asylums and their regulation .......................... 37

   2.1.2 The nature of the sample: Patients in the asylum in the Victorian era ....................... 44

   2.1.3 The causes of mortality in asylums ......................................................................................... 53
2.2 Analysis of Causes of Death at the Berkshire Asylum, 1896-1905 ................................................. 60
  2.2.1 Age at death ................................................................................................................................. 66
  2.2.2 Causes of death amongst non-GPI cases ................................................................................. 66
  2.2.3 Methodological issues regarding data on causes of death ..................................................... 76
  2.2.4 Mortality rates in the population compared to the asylum ...................................................... 79
  2.3 Conclusion ..................................................................................................................................... 83

Chapter 3. Asylum Post-mortems in Mania and Melancholia ......................................................... 86
  3.1 Introduction .................................................................................................................................. 86
    3.1.1 Deaths and post-mortems in Montrose Royal and Berkshire Asylums ...................................... 96
  3.2 Findings and commentary ............................................................................................................ 109
    3.2.1 Post-mortem delay ..................................................................................................................... 109
    3.2.2 Rates of post-mortems .............................................................................................................. 110
    3.2.3 Length of admission ................................................................................................................ 112
    3.2.4 External appearance ............................................................................................................... 113
    3.2.5 Ribs .......................................................................................................................................... 114
    3.2.6 Causes of death ........................................................................................................................ 114
    3.2.7 Relationship of bodily state at death to cause of death ........................................................... 124
    3.2.8 Specific features of the post-mortem findings ......................................................................... 126
    3.2.9 Post-mortem findings in individual cases ............................................................................... 138
  3.3 Conclusions ................................................................................................................................... 141

Chapter 4. Death by Exhaustion in Asylums .............................................................................. 146
Appendix B. Causes of death in main clinical subgroups (Mania, Melancholia, Dementia, Epilepsy, Idiocy and Other) across a 10-year period (1896-1905) in the Berkshire Asylum.286

Appendix C. Causes of death from post-mortem reports of those aged 55 or under at death with diagnosis of mania or melancholia in Berkshire and Sunnyside Asylums ......................... 292

Appendix D. Post-mortem reports in cases with melancholia or mania dying at or under the age of 55 in Sunnyside, 1892-1901. ......................................................................................................... 293

Appendix E. Post-mortem reports in cases with melancholia or mania dying at or under the age of 55 in Fair Mile, 1896-1905. ......................................................................................................... 319

Appendix F. Clinical notes of the two cases of sudden death by suicide in Sunnyside. .... 347

Appendix G. The nine cases whose cause of death was changed from exhaustion after post-mortem......................................................................................................................... 348

Appendix H. Two cases which did not fit the pattern of the other 12 cases of senile mania closely........................................................................................................................................ 349

Appendix I. A case of delirious mania. ................................................................................................. 350

Appendix J. Case summary of patient with potential catatonia ....................................................... 351

Appendix K. 11. Collated information from pathological and clinical reports from vascular causes of death in Sunnyside, with condition of mania or melancholia and under 65 years of age at death in study period, 1892-1901. ................................................................. 352
Appendix L. Collated information from pathological and clinical reports of vascular causes of death in Fair Mile, with condition of MA or ME and under 65 years of age at death in study period, 1896-1905.
List of Tables

Table 2-1 Causes of death put into 5 categories ................................................................. 67
Table 3-1 Length of stay in the asylum prior to death in the post-mortem cohorts........... 113
Table 3-2 Recorded causes of death in mania and melancholia cases dying ≤ 55 years of age in those with and without a post-mortem in the two Asylums......................................................... 116
Table 3-3 Recorded causes of death in mania (MA) and melancholia (ME) cases dying ≤ 55 years of age in the two Asylums and age at death .................................................................................................................. 118
Table 3-4 Body habitus at death and cause of death in both Asylums ................................. 125
Table 3-5 Pathological findings in the cause of death sub-categories in post-mortem brains of deaths in mania and melancholia .............................................................................................................. 128
Table 3-6 Pathological findings in the cause of death sub-categories in post-mortem hearts of deaths in mania and melancholia .......................................................................................................... 130
Table 3-7 Pathological findings in brains of patients with and without vessel atheroma .... 131
Table 4-1 Number, gender, age at death and diagnostic group of patients dying of exhaustion between 1872 and 1906 in both asylums .............................................................................................................. 167
Table 4-2 Length of illness, number of attack and apparent precipitants in eight cases of mania who died of exhaustion ................................................................................................................ 174
Table 4-3 Bodily state on admission and at death of exhaustive deaths ............................ 177
Table 4-4 Cardiovascular findings on examination and post-mortem of exhaustive deaths.. 180
Table 5-1 Age, gender distribution and clinical label of cases with vascular cause of death..204
Table 5-2 Length of stay in the asylum prior to death in the vascular cause of death cohort.207
Table 5-3 Body habitus on admission and at death of the 40 patients from both Asylums..207
Table 5-4 Recorded vascular causes of death in mania and melancholia in both Asylums ..209
Table 5-5 Comparison of mania and melancholia cases with vascular cause of death ascription and presence of specific pathological change in the vascular system ........................................... 211

Table 5-6 Pathological findings in post-mortem brains of deaths in mania and melancholia who died with a vascular cause and their association with vascular pathology markers..... 213
List of Figures

Figure 1-1 Engraving of Moulford Asylum.......................................................... 15

Figure 1-2 The dormitory of Male 4, Fair Mile in about 1913 and the day room of Female 7
with the gallery behind, Christmas, 1913 .............................................................. 17

Figure 1-3 Engraving of Montrose Royal Asylum circa 1890 ................................. 19

Figure 1-4 Results file from the post-mortem report of KK, August, 1905. ................. 25

Figure 1-5 The Board of Visitors to the Berkshire Asylum circa 1910 ....................... 28

Figure 2-1 Deaths per annum across a 10 year period 1896-1905 at the Berkshire Asylum.. 61

Figure 3-1 The 1870 plan of Berkshire Asylum ..................................................... 98

Figure 3-2 The renovated mortuary at Berkshire Asylum ..................................... 99

Figure 3-3 Shelves and neck block at Berkshire Asylum mortuary photographed in 2003.... 99

Figure 3-4 Photographs of mortuary at Montrose Royal Asylum taken by author in 2017 .. 101

Figure 3-5 The grave of William Murdoch, amidst unmarked pauper graves............... 103

Figure 3-6 Plaque on wall bordering pauper graves at Cholsey Parish Church............. 104

Figure 3-7 Schematic of a post-mortem record proforma, Fair Mile Asylum .................. 106

Figure 4-1 Deaths from exhaustion in the two asylums in five-year blocks, 1872-1900. ..... 164
# Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
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<tbody>
<tr>
<td>FH +ve</td>
<td>Family History of insanity</td>
</tr>
<tr>
<td>GPI</td>
<td>General Paralysis of the Insane</td>
</tr>
<tr>
<td>LCA</td>
<td>London County Asylum</td>
</tr>
<tr>
<td>LCC</td>
<td>London County Council</td>
</tr>
<tr>
<td>LC</td>
<td>Lunacy Commission</td>
</tr>
<tr>
<td>ME</td>
<td>Melancholia</td>
</tr>
<tr>
<td>MPA</td>
<td>Medico-Psychological Association</td>
</tr>
<tr>
<td>NAD</td>
<td>No Abnormality Detected</td>
</tr>
<tr>
<td>REA</td>
<td>Royal Edinburgh Asylum</td>
</tr>
<tr>
<td>SD</td>
<td>Standard Deviation</td>
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<tr>
<td>SMR</td>
<td>Standardised Mortality Rate</td>
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# Glossary

<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
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<tbody>
<tr>
<td>Atheroma*</td>
<td>Fatty deposits in an artery.</td>
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<tr>
<td>Arteriosclerosis*</td>
<td>Chronic condition of widespread atheroma and secondary changes of thickening, hardening and/or calcification of arterial walls.</td>
</tr>
<tr>
<td>Caseation*</td>
<td>Damaged tissue (usually by tuberculosis) converted into soft cheesy substance.</td>
</tr>
<tr>
<td>Congestion</td>
<td>An excessive accumulation of blood or mucus in an organ.</td>
</tr>
<tr>
<td>Hepatinisation*</td>
<td>Characteristic pathological change found in pneumonia.</td>
</tr>
<tr>
<td>Hyperaemic*</td>
<td>Excess of blood in an organ.</td>
</tr>
<tr>
<td>Hypertension</td>
<td>High blood pressure.</td>
</tr>
<tr>
<td>Ischaemia</td>
<td>Deficient supply of blood to a body part due to an obstruction to the flow of arterial blood.</td>
</tr>
<tr>
<td>Oedema</td>
<td>Abnormal infiltration and accumulation of fluid in tissue or cavity.</td>
</tr>
<tr>
<td>Phthisis</td>
<td>Progressive pulmonary tuberculosis.</td>
</tr>
<tr>
<td>Meninges</td>
<td>Any of the three membranes that envelop the brain and spinal cord.</td>
</tr>
<tr>
<td>Miliary Tuberculosis</td>
<td>Acute tuberculosis in which tubercles are formed in organs, spread by the blood.</td>
</tr>
<tr>
<td>Myocardium*</td>
<td>The muscular layer of the heart wall.</td>
</tr>
<tr>
<td>Sulcus*</td>
<td>Shallow furrow on the surface of the brain separating adjacent convolutions.</td>
</tr>
<tr>
<td>White Matter</td>
<td>Neural tissue of the brain and spinal cord that consists of nerve fibres and their sheaths.</td>
</tr>
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1 Those with an asterisk * are also to be found as footnotes to the relevant text.
Chapter 1. Introduction

1.1 Overview of research questions and main arguments

The study of the history of psychiatry was initially something of a battle between opposing ideologies and ranged from narratives charting the progress of the psychiatric profession to that of psychiatry as a form of sinister social control. However, more recently, as Wallis points out, more nuanced approaches have been undertaken and, for the most part, the history of psychiatry has become a social history, concerned with day to day asylum life coupled with an examination of the experiences of patients and their families with the power dynamics between staff and patients at the forefront. Porter, in 1985, advocated a move away from “physician-centred” accounts of medical history, noting that mental illness was a complex social phenomenon involving family and community as well as sufferers and physicians, pointing out the marginal involvement of doctors in ordinary people’s healthcare and wellbeing. Porter sought to develop a new history of medicine based on the experience of the patient enlisting the ethos and methodologies of “history from below.” Placing the patient experience at the centre of the analysis has been a productive approach. Over the past few decades, there has also been a significant resurgence of more biomedically oriented studies focusing on scientific developments in psychiatry, neurology, and medicine with several historians exploring contemporary scientific practices in psychiatry in depth. This thesis is largely concerned with this latter theme but aspires to place this account of asylum medical practices in the contexts of both the life of rural asylums and the scientific milieu of the time.

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The phenomenon of premature and frequent death in Victorian asylums has been analysed and debated in the secondary literature but gaps in our understanding remain. There has been considerable scholarship on death, dissection, burial and mourning in the Victorian era but, in comparison, historians have less frequently explored how the deceased insane were dealt with in this period, with some notable exceptions. David Wright stresses that three decades of historical scholarship were preoccupied by how individuals got into the institution, and much less interested in how they left and particularly in explanations that were put forward for those whose admissions ended in death rather than discharge. The first and main concern of this thesis is to provide an analysis of the circumstances and causes of deaths in Victorian asylums in an attempt to understand to what extent and in what ways asylum patients’ deaths were related to aspects and impacts of their mental illnesses and/or to their incarceration in the asylum. This thesis also evaluates the extent to which the practice of post-mortems throws light on the causes of death linked to these excess deaths and seeks to elucidate how far the incidence of excess and premature mortality is accounted for by the presence of physical illnesses concurrent to, and/or associated with, insanity. An assessment is also made of the degree to which the asylum environment itself hastened the demise of these unfortunates. McGovern argues that historians must explore the inner history of asylums to discover more about their mortality, one part of which is to investigate causes of death in greater detail as here.

In tackling this question, there are several linked issues and discourses to be addressed. Firstly, the extent to which premature mortality in the asylum was driven by an accumulation of cases likely to die at an early age, for example, those suffering with organic conditions (conditions with a recognised physical basis), like epilepsy and general paralysis of the insane (GPI), has been little

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considered and is largely unknown.9 One way to explore this question is to look at the average age of death in these asylums overall and in sub-groups. Across the two asylums studied, the average age at death was in the mid-50 years, indicating that despite the increases in life-expectancy over the Victorian period for both the normal population (who Cutler and colleagues show had an average age of death in the late sixties for those surviving until at least teenage years) and the mentally ill, the gap in life-expectancy between these groups remained substantial at around 10-15 years. This gap was very similar to that reported by Wright and colleagues who studied this issue in similar settings in Ontario. However, the current study found that this average age at death was made up of three broad components: a) a population with disorders where insanity and/or behavioural disturbance were secondary to underlying predominantly neurological disorders who had an average of death in their 40s, b) an older group, usually denominated as demented but sometimes as senile, who had a much more advanced age at death and c) those with the label of insanity (that is people with mania and melancholia) who had an average age of death in their mid-50s. It is argued here that sufferers of these latter conditions lost 10-15 years of life expectancy compared with the non-insane.10 Since mid-50s is an average age of death for the insane, a substantial number of sufferers died very much earlier than their “allotted span.”

Secondly, some scholars argue that the premature mortality in asylums was a direct consequence of the living conditions prevalent in Victorian asylums and suggest that infections and epidemics, linked to the poor sanitary conditions, malnutrition and overcrowding prevalent in most asylums, were responsible for the frequent early demise of many inmates.11 Here, it is however argued here that asylum mortality is not directly linked to such environmental factors. Evidence is drawn from study data, from comparative modern studies and from the contemporary literature to show that there is no association between the numbers in an asylum and its mortality rate. Nor does the data support the contention that mortality was a consequence of overcrowding or malnutrition themselves, at least not directly. This study confirmed the findings of other, sometimes larger,

studies that death occurred most often in the early months of admission, pointing to the main drivers being the mental illness itself and/or patients’ health status pre-admission and/or the living conditions at home/outside the asylum. Although infections, most notably tuberculosis, took a heavy toll, the findings from this study provide an evidential base for the view put forward by Ernst that the infections that led to the final demise of the insane were conditions which preyed on the vulnerable weakened bodies of those ravaged by florid mental illnesses, what Szreter calls “scavenger diseases.”12 This study also supports the findings of recent scholars that death by suicide was rare in asylums (less than 1% of all deaths) and agrees with the established view that this scarcity was the result of close monitoring of the many who exhibited suicidal ideas.13 My research, involving a combined clinical and pathological approach, also shows convincingly that death involving violence was uncommon, in contradiction to narratives to the contrary.14

The narratives surrounding two specific causes of death in insanity, exhaustion, and vascular causes, which have been speculated about in both the contemporary and modern literature but not studied systematically, are examined. Together these causes of death accounted for over a third of deaths and both showed a predilection for young patients. In both conditions, examination of the post-mortem records proved helpful indicating that, at least for historians, if not for the patients of the time, the performance of a post-mortem was important, perhaps pivotal, in aiding understanding of these particular causes of death and their associations.

Historians have varied a great deal in their interpretation of exhaustion as a cause of death in insanity with views ranging from it being a nonspecific cover for a host of acts of commission or omission by asylum staff to it being the outcome of a florid illness to it serving as a synonym for emaciation and wasting. These alternative views are examined. The term exhaustion was utilised commonly in the nineteenth century when ascribing death from several disparate conditions including epilepsy, tuberculosis, apoplexy, and insanity. However, when this cause of death was

applied to those dying with mania or melancholia, exhaustion appears to have become less commonly and more precisely employed as the century progressed. Even though the term death by exhaustion was generally used more precisely in this late Victorian period, this study found that one in five of cases of death by exhaustion had an underlying physical disorder of a sort that was likely to cause death. Thus, most previous historical analyses and conclusions drawn therefrom have been based on samples that were not of pure cases of exhaustion. In the research for this thesis, examining the cases remaining after the removal of those with underlying physical disease, left a group that continued to be heterogeneous. It has been argued that exhaustion was employed as a mitigation for unexplained, probably contentious, asylum deaths but this study maintains that there is little or no evidence to support that assertion. Conversely, it is shown that death by exhaustion was associated with very acute episodes of insanity. The notion put forward by some modern commentators, that, for most of such cases, an acute stressful event or emotional shock precipitated these fatal illnesses was not confirmed. This thesis argues instead that of the two narratives which Smith considers underlay death by exhaustion, her second one, that of acute florid psychotic illnesses which becomes fatal seems to fit with most of the cases described in this series.15

Another explanation put forward by some modern historians is that many patients were admitted to asylums in a feeble state during the process of dying and died there from inanition, which was labelled exhaustion, the asylum acting merely as a hospice. Commentators have linked this idea to the prevalent notions of degeneration and toxaemia held by Victorian alienists. While this notion may have some validity, it is contended that it is not applicable to the majority of cases who died of exhaustion in this study. Emaciation on admission or at death was by no means universal in these patients. Some modern scholars argue that such deaths were cardiac in origin based on the belief that the heart was subject to dramatic influences from the emotions which it could not sustain. Only limited support for this contention was found. However, atheroma (hardened fatty deposits) in major vessels was apparently widespread in these young (even by the standards of the day) cases of fatal exhaustion. It may well be that these vascular deposits are linked to the stress of the insanity and are associated with some patients’ early and rapid demise. Finally, the explication of death by exhaustion in asylums put forward by Margaret Harris and colleagues that the patients who died by exhaustion may simply have given up, a form of death often described in

15 Smith, “Visitation by God,” 110.
nineteenth-century literature in the apparent absence of other plausible candidates, is examined. Adland also suggests that patients who died by exhaustion had a “blind drive for annihilation” or “were determined to die.”16 However, on the contrary, I found patients struggling for survival despite overwhelming odds which were not of their making.

This study shows that vascular abnormalities and heart disease, evidenced by the presence of atheroma in the cerebral and cardiac vessels and degeneration of the heart muscle, were prevalent in the deceased insane. This aspect of asylum mortality has been little discussed or theorised about partly because few have looked at the content of post-mortem reports before. Vascular and heart disease were found amongst those with all sorts of conditions: in those patients with mania and melancholia who died prematurely but even more frequently in those who died with exhaustion. The role of these abnormalities in hastening the demise of these patients is examined. To facilitate this a cohort who had vascular disease as the specific cause of death was scrutinized. This was an opportunity to look at this cause of death in a more isolated way, in purer “culture.” The cohort was, as might be expected in a retrospective enquiry of an asylum population, a bit of a ragbag. Some died of what appears to be rheumatic heart disease and other pathologies that were and are unclear and uncertain. However, the vast majority had atheroma in vessels of the heart and/or brain or the changes of fatty heart. Some died of apoplexy, seemingly related to vessel disease with a potential, but unknown, contribution from undetected high blood pressure. Others became weak or died of syncope. This was a common outcome of those with a degenerative process in the myocardium, known as fatty heart or morbis cordis. The hallmark of this condition was fatty deposits in the heart whose origin, as postulated at the time and subsequently demonstrated, is ischaemia (shortage of blood supply). It is argued that the atheroma prevalent in the insane was associated, in variable fashion and to variable degrees, with the problems of insanity itself. It is further contended that this atheroma was probably an outcome of uncontrollable stress and/or the increased likelihood of infection and/or the deprivation experienced by the insane. Tuberculosis and, to a lesser extent, other infections, were major killers of the insane in asylums but so too, I maintain, was vessel disease. The latter was not as prevalent as severe infections and not as visible to either alienists, the public or historians. It is

argued that vascular disease is an overlooked mechanism that at least in part provides an explanation for the frequent early demise of the asylum insane.

The second broad area of enquiry this thesis addresses is the performance of post-mortems in Victorian asylums - why they happened and what, if anything, they achieved. The numbers of post-mortems carried out, the reasons behind any variations and the background of those who carried out the post-mortem are all issues that have received limited attention in the secondary literature, apart from a few notable exceptions.17 Our understanding of what objectives the day-to-day practice of this procedure sought to fulfil and then actually fulfilled is also incomplete and under-researched. Similarly, knowledge of the relationship between the coroner and medical health care institutions, particularly asylums, is lacking. In the nineteenth century, overlapping and sometimes contradictory legislation, including Coroners Acts, Lunacy laws and Anatomy Acts, meant that the process of recording, storing, examining and burying deceased asylum patients was and is subject to interpretation. Wright and colleagues, examining mental hospitals in Canada between 1841 and 1891, discuss the value of asylum post-mortem records as an historical source and posit that they are a “useful window onto several contentious debates on the history of ‘madness’.” These authors highlight how little work has made use of these records and this thesis seeks to redress that imbalance.18

The background to the burgeoning of post-mortems and facilities for them in asylums is outlined in Chapter 2. This includes the public and professional concern engendered by the continued high mortality rate in asylums for many decades despite the Lunacy Acts of 1845 (England) and Scotland (1857) and concerns, again shared by the public and professionals, regarding problematic care, if not abuse, in these establishments. The English Anatomy Act of 1870 laid down that all deaths in institutions should have a post-mortem. This asylum post-mortem and the sometimes-related process of an inquest (or similar procedure in Scotland) have been seen, for the main part, by historians as a way to check on asylum doctors and staff and thereby prevent or curtail abuse and satisfy and to provide reassurance for the authorities and wider public.19 Post-mortems were mandated as a method to identify and, more crucially, prevent violent deaths and deaths from

non-accidental injury. Pertinently, there was still a perception of a lot of abuse going on in institutions, the majority of which, after all, were publicly funded. Furthermore, a range of other concerning issues - for example the causes of relatively frequently found fractured ribs in the insane - were as yet unresolved. Reports of abuse and scandal lessened across the nineteenth century but whether post-mortem practice had any part to play in this, as suggested by some commentators, is speculative. The current study suggests that, if post-mortems did reduce violence and violent deaths, it was a more theoretical than real effect. Signs of violence were identified in the post-mortems of two cases from Fair Mile asylum, yet no action came from those findings either on the part of the coroner or from the Visitors or the Lunacy Commission (henceforth LC). Zuck contends that “gentlemanly agreements” between asylums and Coroners were commonplace so that this layer of public protection was diminished.20 There may well have been similar unspoken accords between medical officers and attendants so that those performing post-mortems were disinclined to find changes that may have stemmed from abuse or neglect. Thus, evidence was adduced here to show that while the post-mortem itself may have been carried out with care and diligence and an open enquiring mind, the processes for independent scrutiny thereafter seemed to be lacking.

Another role the post-mortem played followed on from individual pieces of legislation, like the 1885 Lunacy Acts Amendment Act (for England and Wales) and the 1857 Lunacy (Scotland) Act, which encouraged meticulous record-keeping that aimed to protect doctors and staff from allegations of negligent treatment.21 Post-mortems acted, in Andrews’ words, as “insurance against lawsuits” and against this yardstick, the routine post-mortem appears to generally have achieved its aim.22 In the data series from two disparate asylums examined here, the post-mortems appeared to have been carried out with attention to detail. The success of this aim was further exemplified by showing that the post-mortem exercise reduced potential opprobrium for the staff in cases of death by choking. However, the question of whether these post-mortem activities had an impact on the asylum care in general or on the fate of individual inmates has been little discussed by commentators. It is unclear how often, if at all, post-mortems carried out

21 Cullen, “Post-mortem in the Victorian Asylum,” 280-96.
in Victorian asylums flagged up important concerns over the care or management of individual inmates, particularly those dying suddenly or violently or by suicide and, if so, what were the consequences of this concern. No examples of this virtuous cycle were observed in this study. However, the asylums examined were well run and tightly regulated in any event. Arguably, the fact that a post-mortem was effectively routine acted as a preventative measure, additional to the other processes (for example the monitoring of restraint) to reduce violence and encourage moral management stratagems that were carried out in both asylums. Post-mortems had an aura about them that they could reveal the truth and this idea was probably held by the attendants and may have given them pause for thought. Thus, some limited evidence that post-mortem played a part in reducing asylum abuse and violence was found but the impact seems to have been small. The improved situation in asylums regarding abuse and violence in the later part of the century more probably followed from general improvements in the standard of care along with other methods of scrutiny.

There is a dearth of historical analysis exploring whether the practice of carrying out frequent post-mortems could or should alter the conclusions drawn from analyses that are based purely on death registers. The opinions of scholars about the causes of death in insanity, most of which have not relied substantially, or at all, on post-mortem findings, are critiqued in what follows and several differences, some major, some nuanced, appear. The issue of whether the recorded cause of death in the post-mortem record and that in the death register were the same or different has also been little studied but it was found that almost invariably they were consistent, a finding that gives succour to those studies relying solely on death registers. The post-mortem report provided a cause of death and details of pathological findings from individual organs and, for the most part, these were consonant with each other. While the causes of death following the post-mortem seemed accurate, they were homogenous, and no unusual findings were made which seems out of step with most post-mortem practice. It appears that the medical officers performing them were happy to pigeonhole cases into accepted and acceptable causes of death. Little is known about the level of expertise, training, and conformity with these procedures of those performing the post-mortem in the asylum and this thesis expands what literature there is. Burney has discussed the issue of who carried out the post-mortem examination and draws a contrast between a specially trained pathologist and the physician who treated the patient at the end of their life. He argues that a pathologist would examine the body objectively and provide a cause of death based purely on the signs on the body, whereas the physician who treated the patient at the end of their life
would approach the post-mortem table with a cause of death already in mind. I conclude that it is difficult not to concur with the broad thrust of Burney’s argument. Another issue which bears on this question is that there was very little difference in the causes of death between those cases who had and those who did not have a post-mortem. On the one hand, this finding could be an indicator that the regular practice of post-mortems was useful and improved cause of death reporting in similar cases who did not have a post-mortem, but this finding may also point to the relative futility of the complex and laborious procedure itself.

However, asylum post-mortems were also mandated by the LC because they believed in the merits of the post-mortem as a knowledge-making exercise, and they held that the objectives behind their performance should be broad in scope. These objectives included establishing more definitively and scientifically patients’ causes of death and via this more thorough monitoring leading to a reduction in asylum mortality rates. Post-mortems also encouraged an enhancement of the acquisition and dissemination of knowledge about asylum mortality, the pathology of insanity, and the causes and patterns of both. It should be recalled that the post-mortems described here were carried out in an era when insights in medicine based wholly or in part on post-mortem findings were occurring with increasing frequency and when pathological research and post-mortem investigation, particularly in Germany and, to a lesser extent, in Britain, into mental illness was becoming more systematized and bearing at least some fruit. In terms of an understanding of the brain basis for insanity, however, little progress was evident in this study where there was a lot of use of vague terms, particularly when describing brain changes. Many historians consider that post-mortem findings reported during this period from individual organs of the insane such as brain and heart were non-specific, observing that many alienists at the time expected the findings of post-mortems in lunatics to be consonant with general theories of insanity, like degeneration, that held sway at the time. In support of this, congestion in the brain was the sole finding in a number of cases. This term was in popular use and was promoted as a key abnormality by some influential practitioners. Arguably, congestion, a term with no clear limits,

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26 William Hammond, Cerebral Hyperæmia: The Result of Mental Strain or Emotional Disturbance (New York: Putnam, 1878).
was found because the search was for something, anything, in the brain of a highly disturbed individual. Hurren argues that the practice of asylum post-mortem post-mortems “seldom established new breakthroughs” and, as far as conditions such as mania and melancholia as opposed to GPI or similar disorders, it is hard to disagree with this conclusion.27 There is no evidence that the findings of the 172 post-mortems in insanity analysed in detail in Chapter 3 were ever part of a publication or similar public output other than a footnote in the LC’s Annual Report. Findings from post-mortems in asylums in this late Victorian period that were of value, albeit limited, were made but they emanated from study of a small number of cases in specialist centres, usually involving a pathological expert. Of course, this negative outcome must be seen in the context that even pathological experts struggled with finding anything consistent in insanity or, later, in “psychosis” as seemingly functional disorders with delusions and hallucinations began to be called, as Beer charts, from the 1920s onwards.28 This situation essentially pertained for the many subsequent decades leading to psychiatry being dubbed “the graveyard of Pathology” by Plum in 1972.29 More recently, using complex refined techniques and a great attention to detail and controls, consistent findings in the brain in psychoses have been found, although modern neuroimaging is seen by many as the more fruitful pursuit than neuropathology. Thus, the massive amount of time and effort spent in asylum post-mortems - for example, in weighing the organs or carefully dissecting and examining the meninges - led to virtually nothing concrete in terms of advances in pathological knowledge or treatment. However, a saving grace is that some of the medical officers and some relatives may have taken positives from the process and felt that at least something was being done. It is also true to say that if no one had looked, no one would have found anything. As Worboys emphasises, albeit in the context of the nascent science of bacteriology, ambiguous results of tests did not necessarily mean that the process of investigation was unproductive and he opines that such activity marked the awareness of “new aetiological and pathological models.”30 Similarly, Wallis observes that asylum doctors performing post-mortems “were using these opportunities for observation and experiment to navigate complex issues of vulnerability, aetiology, and therapeutic possibilities.” Though unsuccessful, “the process of investigation could

27 Hurren, “‘Abnormalities and Deformities’,” 65-77.
nevertheless lead to practical and theoretical developments that were important to contemporary observers.”31 The post-mortems explored in the current study are considered from these vantage points to assess to what extent they fostered these various ideals.

1.2 Methods, data collection and associated methodological issues

1.2.1 General comments

The approach that was taken was to study deaths and post-mortems in asylums semi-quantitively. All deaths and their attributed causes from two asylums across approximate ten-year windows were recorded and studied. Data accrued from post-mortems across these years were studied with particular attention paid to those with forms of what is deemed “functional” insanity. Numbers studied were quite large – approximately 1,000 deaths and several hundred post-mortems - which led to some associations emerging and reasonably firm conclusions. It is important to note that despite the large numbers of cases being examined by someone with medical and psychiatric training, there are still potential pitfalls with selection issues and confirmation bias, where conclusions which fit with one’s preconceptions are drawn. It was felt however that this approach had advantages particularly as it was performed in tandem with other methods. The discourse and narratives surrounding these issues in the contemporary literature and the analysis of them in secondary commentaries were also examined and critiqued. Another approach followed was to study some cases in depth, following their story in detail, while taking care to be aware of the risk of associations being made which are not warranted as they may have occurred by chance - the so called “search after meaning” – i.e., that because a set of circumstances occurred in a patient, it necessarily follows that such circumstances were the cause of the outcome, be it disease or death. An outline of the main methods used is given below together with an account of how the data was extracted accompanied by a critique of these methods.

31 Wallis, Investigating the Body, 182.
1.2.2 The Asylums studied

The criteria for choosing the asylums to be studied was that they had to have good archive material available in the form of comprehensive post-mortem records alongside other records (e.g. registers of admissions and of deaths and discharges, clinical case notes and reports from Physician Superintendents, Visitors and Commissioners). To ascertain suitable sites, an initial scoping exercise was carried out. An internet search revealed three asylums whose archival records fitted these criteria. These were 1. Berkshire Asylum (with records in the Berkshire Records Office, Reading) 2. Royal Edinburgh Asylum (henceforth REA: with records in the library of Edinburgh University) and 3. Montrose Royal Asylum (with records held by NHS Tayside, located at Dundee University). None of these records were digitised, so trips to each were made to examine the records and their level of detail. These investigations revealed that comprehensive and consecutive post-mortem records for all three asylums had been preserved with records dating from approximately the mid nineteenth century until well into the twentieth century. In the earlier parts of the nineteenth century, post-mortem findings tended to appear as short addenda to patients’ case records. As the century progressed, dedicated post-mortem volumes were introduced. Some asylums – such as the REA – introduced separate pathological records in bound volumes as early as the 1850s, but most asylums, and the other two surveyed, implemented this in the later decades of the century. In general, and for the asylums listed above, the post-mortem record became more standardised as the century went on, as institutions were increasingly expected to account for both the clinical treatment of patients and the rigour of their pathological procedures. Such records were monitored and checked, exemplified by each book carrying the signatures of visiting Commissioners. The post-mortem records in use throughout the UK altered over the later part of the century with increasing detail requested but also with the adoption of standardised pre-printed proformas. In later versions of the proformas, at the top of each entry were spaces for the usual particulars of name and time of death, but also Form of Mental Disorder on Admission” and “Form of Mental Disorder at Death.” These details were however missing from the records of the REA. While this information could have been ascertained for each patient from other records, this is a very time-consuming task and this partly led to the REA’s exclusion from further study. I was mindful of what Dr Howden (latterly Physician Superintendent at Montrose) said in 1871 on the topic of doing research on the same pathological records at the REA: “Anyone who has attempted to work up statistical facts from a Pathological Record, must have felt how much time and trouble were wasted in wading through case after case which had no connection.
It was considered that ease of access to clinical descriptors would allow a large survey of causes of death for each clinical label. The results of this work are shown in Chapter 2. Another factor which led to the REA being omitted from further investigation was that it was a teaching hospital well known for its expertise in several disciplines, including that of pathology. Indeed, the REA was the driving force behind the establishment of the Scottish Asylums Laboratory scheme (set up in 1896) whose main purpose was to do pathology research on the brain in insanity. Thus, the REA was a centre of excellence and therefore not fully representative of asylum practice in the UK at that time. Furthermore, it was decided that it would be better to focus on a smaller number of asylums. The remaining two asylums had excellent records and provided interesting, and potentially fruitful, similarities and differences. Both were semi-rural and had, on the whole, good reports from Commissioners but were from different countries with different legislation and their staff and patients had differing backgrounds. A brief account of these two asylums will now be given. An account of how these asylums handled deaths and post-mortems is given in Chapters 2 and 3.

1.2.2.1 Moulsford/Berkshire Lunatic Asylum (aka Fair Mile)

Berkshire’s Court of Quarter Sessions formed a union with the Boroughs of Reading and Newbury to build their own asylum close to the village of Moulsford, in the parish of Cholsey. The asylum was known at first as the Moulsford Asylum. It was designed in typical style by C. H. Howell of Islington (1824–1905), the leading asylum architect of the time, and its main buildings are shown in Figure 1.1. The first patients were admitted in 1870. The asylum was designed to accommodate 285 patients but was almost full to capacity within the first year. The asylum was extended between 1878 and 1880 to accommodate 609 patients. Additional staff cottages were built nearby, and outbuildings such as the chapel and bakery were enlarged and improved. In 1896 the Borough of New Windsor joined the Berkshire Union, and the asylum was expanded again to a capacity of 800. In 1897, its name was changed to the Berkshire Lunatic Asylum. In 1948, when it was incorporated into the National Health Service, the name was changed to Fair Mile Hospital although it had been known locally as this for some time.

Figure 1-1 Engraving of Moulsofd Asylum which appeared in The Builder in 1869 (Berkshire Records Office, D/EX 1678/2/1, photograph Spackman collection, https://forgottenFair Mile.blogspot.com/).

The hospital was virtually a self-sufficient community with its own bakery, laundry, chapel, farms and gardens. It was run by the Medical Superintendent, with the support of Assistant Medical Officers, and nursing and clerical staff. In 1892, William Murdoch (1857-1917) was promoted to the post of Medical Superintendent having been an assistant there since 1881. By all accounts he was industrious, determined and highly respected. The hospital employed a wide range of auxiliary staff including a farm bailiff, gardeners, an engineer, a baker, a shoemaker, seamstresses, laundresses, cooks, and housemaids. Most of the staff lived in nearby cottages, and in addition to wages they received various emoluments. These could include several cooked meals, uniforms, board and lodgings, or an allowance of farm produce to take home. Staff were expected to work from 6am to 8pm, six days a week, and from 6am to 6pm on Sundays. Murdoch was thought of highly by the staff, partly linked to him granting them better allowances and leave and working hard to improve their living conditions. Despite perennial problems with recruiting sufficient personnel with the required skills, discipline and tenacity for asylum nursing, morale is said to have been high.33

Murdoch ran a tight ship and, like his predecessors and many of his peers, was involved in every aspect of asylum life from patient care to ordering materials for the buildings and the adjoining farm. Murdoch was a strong proponent of the doctrine of moral management and was energetic in finding and encouraging occupation for the patients, most notably on the farm (for the males) and in the laundry (for the females). Both male and female patients worked cleaning the wards and dayrooms. Murdoch was also adamant that patients should receive as good food as was available and get ample amounts of fresh air and exercise. Accommodation was in strictly sex segregated dormitories to the left and right of the central blocks. Each ward had a single corridor running its whole length, with sleeping accommodation on one side. These corridors were used as day rooms and enabled the staff to continually observe the patients (Figure 1.2). This common feature of asylum design was partly a practical consideration. It was also influenced by Jeremy Bentham’s concept of the panopticon: the idea that continual surveillance not only optimised the good order of the institution but could also induce people to survey themselves and therefore learn to behave in accordance with social norms.  

However, Edginton argues that the design of asylums of that period is less related to the panopticon concept and more about using “space as an interactive component in the process of healing insanity” and that “the intention of the design was to aid in the cure; the outcome was in the everyday life of the patients.” Edginton thus makes the case that the design of these asylums was in keeping with the tenets and delivery of moral management. The layout of the Berkshire dormitories, and the asylum more generally, conforms to that principle.

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Figure 1-2 The dormitory of Male 4, Fair Mile in about 1913 (above). The day room of Female 7 with the gallery behind, Christmas, 1913 (below). (Both Spackman collection).

1.2.2.2 Montrose Royal Asylum (aka Sunnyside)

Montrose in the eighteenth century was a relatively pleasant and prosperous settlement on Scotland’s east coast. The asylum was founded in 1781 as the Montrose Lunatic Asylum, Infirmary and Dispensary, the first public mental health institution in Scotland. It obtained a Royal Charter in 1810. It owed its existence largely to the efforts of Susan Carnegie, a local philanthropist. Carnegie developed many schemes to benefit the poor or mad unemployed working-class people of Montrose. In order to implement these schemes, she had to learn to navigate political complexities. Religious and civic governance were closely linked in the eighteenth century; Montrosians were under the control of both the Kirk Session, the Church of Scotland’s governing body, and the Town Council. Like other women of her era, Carnegie allied with men to help
promote her campaigns for social reform. When Carnegie was 36, she drafted a proposal for a building to house those with mental illness who up until then either wandered unprotected or were incarcerated in the Tolbooth. Her aim was to provide an environment in which patients could receive the appropriate care and treatment to allow them to return to society, an enlightened attitude at this time. In 1834, the Governors of the asylum, carrying out the wishes of Mrs Carnegie, appointed William A. F. Browne as Medical Superintendent. Browne was a bright, inquisitive, and ambitious man. While earning a medical degree from the University of Edinburgh, he learned about phrenology which inspired him to explore psychiatry. He travelled to the continent to study with Esquirol. Browne came to Montrose in 1834 when he was only 29 to take up his first post as a Resident Medical Superintendent. He had only been there for three years when he published a series of lectures (his “fighting book” according to Keddie, past Physician Superintendent and historian of the asylum) that launched his career and became pivotal within asylum science: What Asylums Were, Are, and Ought to Be. In these lectures, Browne recommended that patients be treated with respect and kindness and be provided with opportunities to engage in worthwhile activities, a moral therapy approach that had been advocated by Carnegie and, most famously, by the Tukes at the York Retreat. In 1858, a new improved asylum was completed three miles north of Montrose which contained accommodation for 300 paupers as well as for the more affluent classes and it became known locally as Sunnyside Asylum. By this time, Browne had become the first Commissioner in Lunacy for Scotland and was recognized as a leading Scottish alienist. Browne returned to the Montrose Asylum as one of the three statutory Commissioners in Lunacy. Browne’s reports contained both wholesome praise and pungent criticisms. He noted with approval the number of patients engaged in different occupations, and the busy social programme as means of distraction and

diversion. But at the same time, Browne deplored the overcrowded state of the galleries. The Physician Superintendent during much of the period of this study was Dr James Howden. Howden was interested in, but critical of, developments in drug treatments for excitement. All accounts suggest the asylum during his tenure ran well and the use of mechanical or chemical restraint was noticeably low. The asylum’s patient numbers had grown to 670 by 1900 leading to further building. The site was officially closed in late 2011 and patients were sent to a new £20 million build at Stracathro Hospital - the Susan Carnegie Centre. Sunnyside was open for 230 years and was the oldest psychiatric hospital in Scotland.

1.2.2.3 Private wings

Both asylums had private wings. The one at Sunnyside was in a particularly large and grand building and is the right-hand building in Figure 1.3 below. Numbers in these parts of the asylum varied but were generally in the low tens or single figures. These patients had separate admission, discharge and death registers and the numbers of these events were recorded separately in the reports of the Physician Superintendent. Very few, if any, of these clients had post-mortems. On this basis, it was decided not to examine any of the records pertaining to private patients in either asylum and so all the information in this thesis relates to pauper patients.

![Figure 1-3 Engraving of Montrose Royal Asylum circa 1890 (Friends of Sunnyside Hospital, https://www.urbandonedteam.com/reports/sunnyside-royal-hospital).](https://www.urbandonedteam.com/reports/sunnyside-royal-hospital)
1.2.3 Records examined

1.2.3.1 Case Notes

Case notes have been relatively neglected as a resource by British scholars. This has been partly due to issues in accessing such large amounts of material, a problem which is being partly overcome by increasing archival cataloguing and digitisation. Gayle Davis also argues that clinical records are a rich but neglected source among historians of medicine, although both she and Andrews stress that historians must be aware of changes in the ways that case notes have been constructed over time. A related issue is that the case history is only part of a wider narrative and belongs to the clinical and professional world of the practitioner. As Hess and Mendelsohn have argued, the case history is usually contextualised within a codified narrative framework that involves a process of selection, formatting and indexing, part of a tradition of “ordering the world on and through paper.” Berkenkotter traces the dimensions of shifts in case histories over three centuries using asylum records (from the REA) and her exploration of the medical narrative highlights how the generalisations implicit in case histories have implications for how we understand medical knowledge.

Case notes were extensively examined in this thesis and were found sequentially organised by date of admission to the asylum and by gender. For some of the sampling frames described below, all case notes that were available were examined but for some others, the case note sampling was limited by availability and time. Availability in both archives was generally good and an estimate is that about 95-98% of relevant case notes could be found. Several case reports had gaps and some parts of them were unintelligible. The absence of certain notes and documentation is a drawback but a relatively minor one as there remained a substantial amount of material from which to develop an overall picture. Case note books were very numerous. They were written sequentially so in cases where the admission was long, details of a case could be spread over several, sometimes many, case report volumes. A volume often only covered a year or part of a year and

40 A complete list of sources used in this work is to be found in the Bibliography.
44 Carol Berkenkotter, Patient Tales: Case Histories and the Uses of Narrative in Psychiatry (Columbia, SC: University of South Carolina Press, 2008), 4-14.
therefore examining just one patient in detail required very many volumes. Inevitably, this practical difficulty led to it only being possible to scan for key detail and therefore the accounts of some patients extracted and analysed in this thesis represent only partial summaries. In both asylums, the handwriting was very variable and the identity of the writer mostly unclear. However, it was usually possible to obtain clear information from around the times of admission and that leading up to death which were the most germane periods for this research. The case history also included reports from the order that led to the admission as well as an account of the mental and physical state on admission. This information was relevant for the study and so these aspects were noted in some detail. There was often little of significant import in the case note account of the course of illness in the asylum and the limited material salient to pathology and mortality was often buried in a plethora of mundane entries, so this part of a patient’s journey was often only scanned quickly.

Asylum case notes were multi-authored and reflect the contributions of a number of voices, with the voice of the patient the least well represented. They were written from a particular standpoint, a “set” as it known in psychology, some of which may be discernible and some which may be not. For example, in the case of Fair Mile, there is evidence that Murdoch, Medical Superintendent from 1892 to 1918 (and therefore for the majority of the study period) subscribed to then fashionable idea of “degeneration”. The doctrine of degeneration, that physical stigmata and deviant behaviour, if and when inherited by subsequent generations, led to a “retrograde movement” (in the words of Henry Maudsley, the theory’s most influential alienist proponent) towards “savage progenitors.” This inversion of evolutionary theory appeared to provide an explanation for the putative increase of insanity in the Victorian era and for the poor outcome of many patients, and was used to both explicate and justify the growth of asylums themselves. Like many of his colleagues in the asylum sector, Murdoch was convinced that insanity would get more frequent unless halted by eugenic and custodially quarantining measures. It is also clear that he was a strict authoritarian, and it is therefore probable that his views pervaded the language used by his staff in writing case notes. In addition, the information I have reported is unavoidably

selective and itself governed by my set and preconceptions. Extracting information from case notes is not a neutral undertaking. Another researcher, with a different “set”, may have selected different material for comment. Swartz expands on this point and discusses ways in which asylum records might “either mislead, or fictionalize histories of these institutions.” 48

For the purposes of this thesis, it was deemed inappropriate to retrospectively diagnose any case. The arguments for and (much greater) arguments against this practice are set out in detail in Chapter 4. To briefly anticipate and summarise, many scholars feel it is not valid and potentially anachronistic to diagnose specific patients with labels we now use as past clinical descriptions are conditioned by stigmas, concerns, questions, and ideologies often divergent from than those produced in modern clinical contexts. This is particularly the case when a new derived diagnosis is based on what an often-unknown writer, in a largely unknown situation, wrote in a case book some time in the nineteenth century. If this were to be done it would amount effectively to diagnosing a patient without having seen them in person. The label applied in the case notes and in various registers was used at face value. For the bulk of this thesis, the most germane labels are those of mania and of melancholia. The historical discourses regarding these two labels and some of the issues raised by, and problems encountered with, them will be discussed in Chapter 2. The diagnostic label given to patients was usually to be found within the case notes. It was also found, almost invariably in the same words, in the admission register, in the register of deaths and in the post-mortem report. This label was copied verbatim into the results spreadsheet. However, there was a lot of variability in the terms used. For example, in the case of mania, one might find some cases labelled as just that, others as acute mania, others again as chronic mania. Taking the example of mania further, some cases were labelled as acute delusional mania, some as acute delusional insanity. Dealing with each of these labels as separate entities has value but it has the effect of creating a myriad of categories. So, for both mania and melancholia (a term which also often had several descriptors around it) these variants have been grouped together to create an overriding category. The scholarship that justifies this decision is discussed in the relevant chapters. Nuances may have been missed and there is a risk (slight in my view) of category errors. As always, there is a need for further research on this topic and efforts at replication. A related issue is that in the death register and in the post-mortem report, the mental state at admission and at death was given separately. Typical examples might include acute mania at admission but

chronic mania at death or acute mania on admission but dementia at death. The latter label has been noted but this work has not analysed this data, and the issues thereby raised further at this time. There may be a wealth of information contained in these ascriptions of the later life of patients, but they were far too numerous to categorize.

1.2.3.2 Admission Registers

Both archives contained books giving brief outline details of admission. In some Victorian asylums these records contain much useful information, some utilising proformas, potentially of considerable use to the historian as described by Andrews.49 However, in these two asylums only bare demographic detail and a telegraphmatic account of factors which led to the admission were recorded. The length of the episode prior to admission and which attack this was, were noted from this register. Main details of the circumstances of admission, including reports from those certifying the patient, were summarised from case notes.

1.2.3.3 Post-mortem Reports

Pathological records from both asylums were available in sequential bound volumes which contained chronologically ordered post-mortem reports. There were five volumes of such reports in the Berkshire archives and six in the Dundee archive covering the period from approximately the mid-nineteenth century to the 1920s. At the beginning of each volume there was an alphabetical index of patients who had received a post-mortem. The reports were inspected each year by the LC and a short, signed statement of its approval could be seen on the inside of the front cover.

Generally, each post-mortem was on one page, and all were handwritten although from about 1870 they were written onto printed proformas. A representation of a printed proforma is shown in Chapter 3 and examples are to be found in Appendix A. In a few cases in both asylums’ records there were line drawings of findings, usually illustrating changes on the surface of the brain, but there was no pattern to this activity which appeared ad hoc and sporadic. None of the cases described in detail here was accompanied by an illustration. Occasionally (and somewhat alarmingly), unspecified smudges could be seen on post-mortem reports: pages may have been

stained with blood or damaged by preservatives, making them what Wallis calls “unusually resonant objects.”

The format employed by these asylums was a printed proforma which contained fields for entering specific information. While this more standard form of notetaking may have made the material more “user-friendly” for the historian, the standardisation of record keeping may have influenced the content of the notes themselves. As Hess and Mendelsohn suggest, the formatting of patient histories so that information is divided into “elements”, while allowing easier access and making them comparable and combinable, then and now, may result in a structure that constrains valid analysis. Gitelman similarly argues that an ambition of bureaucracy is to rid itself of all emotions and subjective judgements; an ambition, she opines, which is epitomized by blank pre-printed forms which, in this analysis, become prescriptive. While historians have to be wary of ideas gleaned from such sources, there is no alternative but to use them, at a practical level. The key details of the post-mortem report were noted directly into a spreadsheet on a laptop. An example of my derived result from Fair Mile is shown below in Figure 1.4.

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51 Hess and Mendelsohn, “Case and Series,” 287.
<table>
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<th>No. 94 K******** K**** (PM 212)</th>
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</thead>
<tbody>
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<td>Governess</td>
</tr>
<tr>
<td>PM performed</td>
<td>Lawson</td>
</tr>
<tr>
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<td>Single, Farringdon. Transferred from Bethlem Hospital</td>
</tr>
<tr>
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<td>Heredity and climacteric</td>
</tr>
<tr>
<td>Bodily condition</td>
<td>2&lt;sup&gt;nd&lt;/sup&gt; Class, thin and feeble</td>
</tr>
<tr>
<td>Duration of attack</td>
<td>14 months</td>
</tr>
<tr>
<td>Attack</td>
<td>First</td>
</tr>
<tr>
<td>Date of death</td>
<td>18&lt;sup&gt;th&lt;/sup&gt; Aug 1905</td>
</tr>
<tr>
<td>Date of admission</td>
<td>5&lt;sup&gt;th&lt;/sup&gt; Jan 1898</td>
</tr>
<tr>
<td>Age at death</td>
<td>53</td>
</tr>
<tr>
<td>Date of PM, PM delay</td>
<td>19th Aug 1905, 24 hours</td>
</tr>
<tr>
<td>Cause of death</td>
<td>Heart disease</td>
</tr>
<tr>
<td>Mental condition</td>
<td>Acute then Chronic mania</td>
</tr>
<tr>
<td>External appearance</td>
<td>Somewhat emaciated with no injuries etc.</td>
</tr>
<tr>
<td>Head</td>
<td>Brain soft and gyri are wasted. Sulci gaping. Grey matter wasted and white matter oedematous. Atheroma of basal arteries.</td>
</tr>
<tr>
<td>Abdomen</td>
<td>NAD</td>
</tr>
<tr>
<td>Heart</td>
<td>Weight (oz)</td>
</tr>
<tr>
<td>R lung</td>
<td>6</td>
</tr>
<tr>
<td>L lung</td>
<td>18</td>
</tr>
<tr>
<td>Liver</td>
<td>20</td>
</tr>
<tr>
<td>Cerebrum</td>
<td>26</td>
</tr>
</tbody>
</table>

Various points about this results file are noteworthy. They were, by necessity, written in a telegraphic style. Only what were deemed to be key, positive findings were noted in the results.

Figure 1-4 Results file from the post-mortem report of KK, August, 1905.
sheet, so that, unless noted, a structure not mentioned in the results sheet had been reported to be normal in the post-mortem record. Exceptions to this were the usually quite long descriptions of the skull and its meninges (dura mater, pia and arachnoid processes). An *ex-cathedra* decision was made to exclude these (many and voluminous) findings from the results on the grounds that it was deemed unlikely to be a fruitful use of time. Similarly, there were often descriptions of the surrounding layers of the heart and lungs, and sometimes complex measurements of hearts valves, that have been excluded from further enquiry. Positive findings in the abdomen were a rare occurrence. In the case example shown in Figure 1.4, no abnormalities were detected, and this has been recorded with the abbreviation NAD. Finally, it can be seen that organ weights were measured at post-mortem in the vast majority of cases, and these were faithfully noted in my results sheet but, as yet, no analysis has been undertaken of these measurements.

This methodology is perhaps open to the criticism of it being subjective and selective. The strictures noted above with reference to case note evaluation and the dangers of confirmation bias apply equally to the extraction of data from post-mortem reports, arguably even more so. However, this is the first time, to my knowledge, that anyone has tried to quantify information from these records, and they give one of the few available windows into both the process of asylum post-mortems and their outcomes in that era. Inevitably there must be selection of findings deemed noteworthy. Perhaps the best way to justify this methodology and mitigate anticipated criticism is to say that this project could be considered as a scoping one, useful for identifying issues for further, more focussed enquiry. Historians have identified a number of related issues in the examination of post-mortem reports. For example, Mooney, in his study of deaths in London, observes that deaths in institutions in the nineteenth century were characterised by limited diagnostic “depth” and accompanied by a loss of the “patient narrative.”

A further stricture about post-mortem reports relating to their underlying ideologies is highlighted by Wallis who warns that when consulting post-mortem records from this, or indeed any period, that “it is crucial to bear in mind that anatomical evidence often played a part in supporting scientific racism and eugenically-inflected conceptions of disability.”

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54 Wallis, “Post-mortem Records,” 63.
It was noted above, under the heading case histories, that differing versions or variants of what might be considered to be the same condition were lumped together and that only the admission mental state label was considered at this juncture. These procedures were also carried out when it came to relate cause of death to diagnostic label. The latter also showed variants, all of which were noted in the original data sheet, but a similar aggregation process took place to create broader categories. In many cases this was straightforward. For example, it seems plausible that there was little significant difference between “Melancholia with exhaustion” as a cause of death compared with “Exhaustion from melancholia,” or “phthisis” compared with “miliary tuberculosis.” However, some causes of death were more complicated but were still amalgamated into a broad classification - these instances have been individually discussed in the relevant chapters.

1.2.3.4 Death Registers

Both asylums had registers of deaths. In the case of Sunnyside, there were separate volumes containing information of each sequential death, including demographic detail, whether a post-mortem was performed (although this data was not always accurate) and the cause of death. In the case of the Berkshire Asylum, deaths were reported in the Register of Discharges, Removals and Deaths which, like Sunnyside, were chronologically ordered and contained similar material. From the Berkshire records, information about deaths was also obtained from the General Statement books (1896-1902, 1902-1909). These books included monthly mortality statistics and comments about them by the Medical Superintendent in his report to the Visitors.

1.2.3.5 Medical Superintendents’, Annual and Chaplains’ Reports, General Statement Books (Berkshire/Fair Mile) and Minute Books (Sunnyside)

Both asylums have an extensive written heritage in the archives, much of it contained in the above volumes. The Commissioners in Lunacy on both sides of the border produced rules about record keeping and recording information was not seen as ephemeral but as an integral part of the life of a Victorian asylum. In Berkshire, responsibility for running the asylums was devolved to the magistrates who appointed a Committee of Visitors for whom the Medical Superintendent produced a monthly report. The photograph below is the only available one of a Committee of Visitors and was taken in about 1910 (Figure 1.5). The dignified personage in top hat and spats is Mr J. T. Morland, long-serving Clerk to the Visitors. The other seated gentleman might be either the Medical Superintendent, Dr Murdoch, or the committee’s chairman, Mr Martin. Murdoch did
not always condescend to attend the monthly meetings. Since the superintendent was accountable to the Visitors in nearly all matters - and highly dependent on their support and permission where any expenditure of public funds was required - his absences may not have been entirely helpful.\textsuperscript{55}

![Image of the Board of Visitors to the Berkshire Asylum circa 1910 (Spackman collection).]

Figure 1-5 The Board of Visitors to the Berkshire Asylum circa 1910 (Spackman collection).

The monthly reports of the Medical Superintendent included accounts of admissions, discharges, and deaths but also a short commentary about issues related to these events. Also included in his report were details of outbreaks of infections and any untoward incidents. The report also served as an opportunity for the Superintendent to make comments. These often took the form of observations on the nature of admissions and the state of the asylum, usually concentrating on the inappropriateness of the former and the consequent pressure on the latter. Murdoch was critical that “asylums are looked upon as nursing homes for the demented and aged, and as houses of detention for idiots and imbeciles” and considered that there were other establishments better suited to the care and treatment of these conditions.\textsuperscript{56} It should be emphasised that asylum superintendents like Murdoch had little control over admissions. Admission to the asylum reflected a relationship between several local officials of differing

\textsuperscript{55} Wheeler, Fair Mile Hospital, 28.

\textsuperscript{56} Murdoch quoted in Mark Stevens, Life in the Victorian Asylum: The World of Nineteenth Century Mental Health Care (Barnsley: Pen and Sword, 2014), 143.
professions and required certification by the local magistrates. Although the Magistrates controlled admission to asylums, it was the Board of Guardians (ad hoc authorities that administered the Poor Law) that had to justify the resultant charges to the ratepayers. Each of these groups had their own attitudes to, and tensions with, each other and to central bodies. The Medical Superintendent’s report was his best, probably only, chance to express his views. Unfortunately, minutes of the Visitors meetings are missing for the period under review. The Medical Superintendent’s report to the Visitors is to be found in the General Statement Book which also contains a myriad of other reports from the asylum administrator, including accounts of the fabric of the asylum and its farm and all aspects of asylum income and expenditure. Despite the unavailability of the Visitors’ reports, we know that they were generally positive and supportive of the staff as they are alluded to as such in the asylum’s Annual Reports. Annual Reports contained a long report from the Physician Superintendent which captured all the above reports’ details and summarised them into various headings. There was some critical commentary from Dr Murdoch in the Annual Reports, but it was noticeably less strident than that seen in the equivalent report to the Visitors. There was then a report from the visiting Commissioners who also appended their signatures. Their comments were generally anodyne and supportive and any negative critique or rebuke to the asylum was expressed in the most gentlemanly of terms.

Some of the chaplains left quickly, often after a year or less. However, some of the chaplains threw themselves into asylum life with gusto. The Revd F. T. S. Dyer (Chaplain 1896-1910), played in the band, worked in the library, and succeeded in getting the Visitors’ Committee to give him an annual allowance for the purchase of new library books. Each month, the Chaplain gave an account of services held and of funerals officiated. There are some details from the latter activity which are alluded to in subsequent chapters but, for the most part, these reports were dry and repetitive and contained little of interest. The only excitement was the report of the dismissal of a holiday relief Chaplain for frequent drunkenness in August 1898.

In the Sunnyside archive, the only source of additional information was the Minute Books. There were two of these available, covering the periods 1887-95 and 1895-1903. These books were

58 Wheeler, Fair Mile Hospital, 28.
sequential minutes of monthly meetings of senior staff and Visitors of the Montrose Royal Lunatic Asylum infirmary and Dispensary. The latter consisted of 20 people and was chaired by Provost Mitchell and included 5 Reverends. Within the minutes there were reports on admissions, discharges, and deaths. Details of untoward incidents, including the use of restraint and seclusion, were given alongside extensive accounts of expenditure. The Minute books also contained Annual reports to and from the Scottish Commissioners in Lunacy.

These archives have the benefit of being contemporaneous accounts of issues relevant to the questions asked in this work and insight into the way these issues were perceived by both clinicians and administrators. However, these materials, from both asylums, were voluminous and very detailed. Most of the information contained in them was not germane to this project. Data extraction from these archives was thus selective. While most of the Annual Reports and Chaplain’s Reports were consulted and relevant information noted, in both asylums, the information from the year 1895 (chosen at random) was looked at in more detail. However, in reality, relatively few notes were made. It should be noted that it was hard to follow up details of incidents of potential malpractice because of the voluminous nature of these reports.

1.2.4 The periods studied

As mentioned above, the information recorded in asylum post-mortem reports grew and, by the late nineteenth century, most asylums, including the ones studied here, had dedicated post-mortem record volumes containing pre-printed proformas. As Wallis says, pre-printed forms specified what was to be examined and commented upon but do not necessarily lead to greater detail: “The observations and rich language of earlier free-form books could be lost as pathologists completed the record with one- or two-word responses.” Notwithstanding this caveat, Wallis’s conclusion that “Forms are helpful... for the researcher who is interested in a particular body part or condition, being easy to skim at speed” is a very accurate reflection and one germane for this particular study. 60

These considerations led to the conclusion that the best period to look at for the bulk of this work was the very last part of the nineteenth century and the early twentieth century. This was also an interesting period as academic research on insanity and on its pathology was active, but at the

60 Wallis, “Post-mortem Records,” 60.
same time older ideas of insanity were prevalent in the public and asylum staff. An approximate ten-year period was deemed adequate to collect sufficient data to reduce sampling errors. In the Sunnyside records a period starting in 1892 (when a new volume with a slightly enlarged post-mortem proforma started) to 1901 (thereafter the pathological records were sparse in number for about two years for reasons that are unclear) seemed to capture sufficient data with only a short (unexplained) three-month gap in the records. In Fair Mile’s case, data collection was extended for two months further than ten years as, by chance, that allowed the collection of 100 of the target cases for the research described in Chapters 3-5. The period selected was from 8 November 1895 (the start of a new and slightly enhanced pathological record book) to 8 January 1906 (122 months). Thus, the study periods were different between the two asylums though there was an overlap of seven years (1895-1901 inclusive) where data from both establishments was collected.

The data collected in the above periods were the basis of the survey of all causes of death in the Berkshire Asylum laid out in Chapter 2 and the causes of death at post-mortem in those patients with mania and melancholia who died under 55 years of age and who had a post-mortem discussed in Chapter 3. For Chapter 4 which looked at deaths by exhaustion, the sample was expanded from the above dates to be able to have both a larger number of cases to study but also an opportunity to examine temporal trends in the use of this label. Since analysis of the post-mortem record was pivotal to this research, the years chosen for enquiry reflected the availability of post-mortem records. For the Berkshire Asylum, the years from 1872 (when post-mortems first became frequently performed) to 1906 (see discussion above for the rationale for this date) were chosen and for Sunnyside from 1872 (for the same reason that this date was chosen for Berkshire) to 1902 (when post-mortems ceased to be performed for two years). For similar reasons, the sample for Chapter 5 was expanded by studying all those dying with such a cause of death in these periods at or under the age of 65.

1.2.5 Sample sizes

Since the questions asked in this thesis were numerous and often exploratory, there was no way the number of cases to be identified could be stated in advance. The numbers were not pre-specified but emerged heuristically as the study progressed, largely based on what was assessed as adequate sample numbers in the main groups and, where possible, the subgroups. Furthermore, no attempts at statistical evaluation of the data were performed other than the simplest arithmetic comparison within a group. It was deemed vital not just to examine death
register and post-mortem reports, but to look into case notes to gain more understanding of the nature of the patients’ problems and their setting. Similarly, it was important not just to record post-mortem data but to investigate what was said about this activity in accounts and reports of the hospital and to understand the context of the post-mortem activity in the setting of asylum life. However, examining case-notes and looking through hospital reports is laborious, and this led to constraints on the research. For example, it was not practical to get a gender balance in the sample that reflected the reality at the time.

### 1.2.6 Control populations

Existing scholarship on the pathology and mortality of insanity often lacks comparative information from control populations. Attempts to overcome this difficulty are described herein where comparisons to information from the “normal” population are made but, in each case, caveats are stressed. For example, life expectancy data from outside the asylum is based on people with a very different age structure from that within it. In particular, the impact of the still high infant mortality skews such comparisons and attempts to overcome this are fraught with difficulty. These strictures also apply to attempts to compare particular causes of death, for example that of tuberculosis, between the community and the asylum. Attempts were made to find a hospital with adequate post-mortem data so that certain pathological findings and causes of death in the asylum could be compared with that in the community, but it proved impossible to identify a suitable setting for such work. Post-mortem records tend to be available from teaching hospitals or centres of excellence, neither of which match the semi-rural setting of the asylums studied. Another issue is that it proved impossible to compare the outcomes of pauper lunatics in the asylum and the fate of such cases not in the asylum, be they in the workhouse or in the community. This was due to the absence of any extant data in these latter settings.

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61 In late 2021, the Wellcome funded St George’s Hospital, London, Post Mortem archive project was launched ([https://archives.sgul.ac.uk/postmortem](https://archives.sgul.ac.uk/postmortem)). Scans of the post-mortem reports and clinical histories dating from about 1840 to 1920 are freely available on the internet. This unique continuous record of post-mortem practice at a hospital may have provided me with a suitable control population but came too late in the process.
1.3 Synopsis and Main lines of Enquiry

This thesis asks the question of the degree to which analysis of causes of death focusing on those deaths that were followed by a post-mortem illuminates the issues surrounding mortality in Victorian asylums. Overall, this examination of cause of death in insanity provides a nuanced account of asylum mortality in late Victorian and early Edwardian institutions with some novel findings. The two succeeding chapters of this thesis focus on this question by examining all the causes of death in one of the asylums (Chapter 2) and by examining the detail of post-mortem reports carried out in younger patients with insanity in both asylums (Chapter 3). Many of the novel findings of this thesis relate to those young sufferers who died of exhaustion and those who died of heart disease and detailed findings and commentary on these topics are to be found in Chapters 4 and 5 respectively. This research seeks to establish whether another, previously little discussed, underlying factor was impacting on these patients and their longevity, pathology in the vascular system. Only a very small number of alienists at the time, or subsequent historians of the topic, have considered or discussed the pertinence of vessel disease. The relevant histography is laid out in Chapters 4 and 5.

The second set of questions this thesis addresses is an analysis of the objectives behind the vast numbers of post-mortem in asylums in the late Victorian and early Edwardian periods and an examination of which, if any, of these objectives were met. As set out in Chapter 3, overall, the findings of this enquiry into asylum post-mortems reveals that they were carried out on such a large scale because they were mandated and that they served only minor direct clinical or academic purpose. There may have been a rationale for post-mortems earlier in the nineteenth century, but it is questionable whether they continued to serve much purpose in these later periods. Once initiated, one surmises that the Commissioners found it hard to reflect and put limits on the process or could it be they were afraid to even contemplate that? Andrews and Hervey emphasize that the Scottish and English Commissioners respectively were imbued with an essential conservatism, an attachment to red tape and legalism and, even more importantly, “strongly prejudiced towards a self-justifying view” – all factors that mitigated against them opting out of a practice once established.\(^{62}\) One of the objectives of post-mortem practice was to throw

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light on the question of why insane people died frequently and, overall, at a young age and
Chapters 3-5 assess the extent to which that objective was fulfilled. This leaves the final question
of whether the material from a post-mortem series like this provides insights for a medical
historian. I contend that it does and provide a range of evidence to justify this conclusion,
particularly in Chapters 4 and 5.

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*trade... of lunacy, they ‘cannot interfere’-they say,* The Scottish Lunacy Commissioners and Lunacy Reform in
Chapter 2. Mortality and Causes of Death in Victorian Asylums

2.1 Introduction

This chapter has three main purposes. The first is to delineate and evaluate the past five decades of scholarship on dying and death in late Victorian and early Edwardian asylums, concentrating on studies primarily devoted to the period 1844-1914. The second is to present and evaluate the results of research overviewing causes of death in the Berkshire Asylum for the period 1895-1905. The final aim is to set the scene for the categories of patients which will be examined in detail in later chapters.

Recent asylum studies have moved on from debates about insanity as deviance and asylums and alienists as agents of social control or, conversely, tributes to nineteenth-century humanitarian developments for the insane and have instead concentrated on experiences and practices. In this chapter, practices relating to asylum deaths are discussed. An assessment is made of prevailing patterns and causes of mortality in asylums. In these times, the identification of causes of death was seen as a necessary and vital preliminary to understanding and eradicating disease as death was seen as definitive.\footnote{John Eyler, \textit{Victorian Social Medicine: The Ideas and Methods of William Farr} (Baltimore: John Hopkins University Press, 1979).} David Wright and colleagues describe deaths in asylums as a “useful window onto several contentious debates on the history of “madness”.” The main focus here is on one of the questions raised by these authors, “what were the patients dying of, if they were not dying of lunacy itself?”\footnote{Wright, Jacklin and Themeles, “Dying to Get Out of the Asylum,” 599-600.}

There has been a considerable body of work investigating the demography of death in insanity. In England and Wales, data on deaths were collected regularly before this was made compulsory by the 1874 Births and Deaths Registration Act. The LC — the body responsible, from 1845, for overseeing the English asylum system — required dates and causes of death as part of the legal documentation for all patients admitted to registered mental hospitals, so there is a fairly
complete data set for scholars to examine, albeit one containing only limited demographic details. Work by a range of scholars has shown that death in the asylum was frequent and often premature compared with the general population. This large and premature mortality showed a predilection for men, for migrants, for the malnourished and for those suffering from mania. There is a plethora of evidence from both primary sources and secondary analyses that many of the excess deaths occurred early in admission. However, the causes of these deaths remain uncertain, mired as they are in categories subject to many vagaries of time, place and the individual recording it. Building on pioneering work in this field by Luckin, Hardy observes that registered causes of death often bear only an approximation of the truth and that this was particularly the case during the period before standardised systems were put in place.

This chapter documents the causes of death of a large number of asylum patients across a 10-year period in the semi-rural Berkshire Asylum. The vast majority of deaths had a post-mortem examination. The post-mortem was part of the asylum system, but its practice, findings and outcomes are seriously understudied. The issues surrounding asylum post-mortems are examined in detail in Chapter 3 but here the cause of death stated in the post-mortem report and/or the Register of Deaths is the topic of enquiry. Historians acknowledge that uncovering what lay behind the different mortality rates among asylum patients during these decades is a formidable task, further hampered by the limited descriptions of the physical condition of patients on admission and thereafter. By the end of the century, however, there was the beginning of a more centralised and standardised system of records; this, coupled with the development a specialist psychiatric medical press and debates in the profession about asylum mortality, aids an improved, albeit still limited, examination of the topic. Analysis of the data accrued along with a detailed review of comparable data in the literature should lead to an increased understanding of the issues.

2.1.1 Mortality in insanity and the growth of Asylums and their regulation

The importance of lunatic deaths in prompting lunacy reform and shifts in the nature and focus of lunacy provision became evident in the later part of the eighteenth century. Public and professional interest in the topic was fuelled by the apparently scandalous deaths of patients in asylums, such as York Asylum, and also private madhouses, such as Hoxton, particularly when the institutions were shown to have been culpable and neglectful. The controversies which ensued strongly influenced public debates and reformist responses. The widely held view that better regulation and monitoring of care were the ways to counteract the high asylum mortality is described by several historians. The much-publicized smaller mortality at the Retreat at York with its focus on moral treatment fed strongly into this narrative. From 1792 to 1845, the average death rate per annum at the Retreat was 4.7%. Similar comparatively low rates were seen in a few establishments such as Bethlehem Royal, whereas much higher ones (as high as 30%) were seen in other asylums particularly those with inadequate diets, poor hygiene and regimes characterised by frequent use of restraint. Historians have come to the view that some of these differences were due to differing admission policies and local demographic variations and the consequent case-mix, but at the time it was widely believed that, since there were major differences in mortality between patients with the same condition, many deaths were avoidable. The parlous condition of lunatics attracted much public attention and this prompted Committees of the House of Commons to investigate the management of the asylums and other settings where insane were kept (such as licenced houses) in 1807, 1815-16, and 1827. Many concerns were raised but there was little improvement.

These enquires, however, created concern and emphasised the need for more thorough and accurate information. The work of William Farr was an important part of subsequent developments. Farr was appointed as Compiler of Abstracts in the Registrar-General’s office and

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was promoted to the post of Chief Statistician in 1838.\textsuperscript{73} Farr’s 1841 report on asylum mortality provoked significant public concern. It was one of the key influences in driving the Lunacy Act of 1845, leading, \textit{inter alia}, to the requirement for asylum Superintendents to publish annual reports which included figures on mortality.\textsuperscript{74} Farr suggested that the mortality rate in asylums in England and Wales was at least three times that of the general population. Farr observed that the highest rate of mortality in the asylums was amongst patients between 35 and 40 years of age and amongst males. He also demonstrated that the annual mortality of both male and female paupers in licensed houses was twice as great (at 21% per annum) as the average mortality of lunatics in asylums. Farr demonstrated that there was no information available on the mortality rate of lunatics in the community or workhouses.\textsuperscript{75} This still pertains. A number of scholars have examined health and mortality statistics in workhouses but the lack of written records on lunatic inmates in workhouses and the absence of registers from which accidents or death could be ascertained (as were required in asylums) continues to hamper useful comparison.\textsuperscript{76} Farr also demonstrated that the annual rate of mortality was greater in the acute rather than the chronic stage of insanity and that it was higher in the early period of admission. Thus, at the hospitals of Bethlem and St. Luke the annual mortality among the class called “curables” (acute cases - mania, monomania, or melancholia) was 11% but only 6% among “incurables” (chronic cases - incoherence, imbecility, or dementia).

There is a great deal of evidence which shows that the high rates of mortality compared to that of the general population continued on into late Victorian asylums, especially amongst recent admissions. Anna Shepherd records a cumulative death rate at Brookwood Asylum, Surrey of about 30% (32% in males) between the years 1867 to 1885, endorsing Wright’s comparable findings at Buckinghamshire County Asylum.\textsuperscript{77} Farr’s protégé and successor, Noel Humphrey analysed data compiled for the 1890 LC report and found similar patterns of mortality (including

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\end{flushleft}
early age of death and excess in males) as those described by Farr nearly 50 years earlier. Whilst stressing that Farr had “very considerably understated the effect of insanity upon English mortality,” he recognised that the case mix was different from the time that Farr reported. Humphrey estimated that in the 1850s only about 60% of the insane were under treatment in asylums, hospitals or licensed houses whereas by the 1890s the proportion of the insane located in these institutions had increased to 86%. Mortality rates remained at the same level in the later part of the nineteenth century, but this headline figure is, to some degree, misleading. One problem is that these mortality rates were calculated as the number of annual deaths divided by the asylum population, but this calculation takes no account of throughput and the variance that a high or low turnover would bring. This problem means that comparisons between settings or groups have limitations. Humphrey also produced evidence to show the increasing average age of patients in asylums and pointed out that this effect would of itself naturally increase the death rate. While the mortality rate was still high in lunatics (estimated in the 25-55 age group to be six times the rate that prevailed among the same ages of the general population), the continued high overall figure was also related to deaths in a more elderly group with dementia syndromes.

Public concern still ran high on this issue. An LSE pamphlet, published in 1890, captured press comment on the LC data and described the continuing high mortality as “Frightful” highlighting that, despite the increased provision of care and its improvements, the problem of premature mortality in the asylum was unrelenting.

Some argued that it was not late admission but admission itself that was the problem. At the time there was a debate about the comparative benefits of supervised care. Most leading contemporary medico-psychological authorities sought to explain higher and early mortality patterns in asylums by pointing to wider community and demographic factors, though a few others saw the asylum environment and the effects of confinement itself as to blame. Opinions on this plainly vary. The phenomenon of death soon after admission, exemplified by Farr in 1841, frustrated Medical Superintendents throughout the period. In 1863, John Charles Bucknill

80 Anon, “Public Opinion on Private Lunatic Asylums, Giving an Account of the Frightful Mortality in them, Disclosed by the Report of the Commissioners in Lunacy,” LSE Pamphlets, 1890, 1-14. The tenor of this detailed article can be summed up by the following quote from it “If we accept the statement often made by those who have studied the subject, that numbers of sane persons are got into these places by relatives for the express purpose of being got rid of, the enormous death rate is easily explained,” Ibid, 3.
(Physician Superintendent at Exminster, first editor of the *Journal of Mental Science*) was reported by Miller as saying that “the mortality in the Essex Asylum has been materially increased by deaths ensuing shortly after admission,” further stressing that “delays in sending patients to asylums… are the natural results of…. a sordid but mistaken economy, which is… multiplying the sum of human misery, and doubling the mortality of our hospitals for the insane.”\(^{81}\) On the other hand, Harriet Sturdy reviews evidence to show that the avoidance of admission by the practice of boarding-out was a better approach for several reasons, including lower mortality. She points out that, in the 1870s and 1880s, the average asylum mortality rate in Scotland was 8.2% but among Scottish lunatics who were boarded out only 5.6% and even lower when the placement was in private houses.\(^{82}\) She acknowledges, however, that the lower mortality rates in the community samples may well have been due to less ill patients being so placed.

By the later decades of the century, it became apparent that the problem of high asylum mortality could not be fully explained by long admissions and older patients. The data pointed the other way. In 1877, Chapman (Medical Superintendent of the Hereford County and City Asylum) confirmed the observation that the excess of deaths was in the younger male patients at an early stage of admission to the asylum, particularly the first year when nearly one-third of male and over one-sixth of female patients died. These rates fell to 12% during the second year of residence until at the end of five years “the sources of excessive mortality appear to be nearly exhausted.”\(^{83}\) In 1894, Chapman further showed that the mortality of males aged between 40 and 50 was six times greater than the general population.\(^{84}\) Similar findings were reported by Arthur Mitchell, a Commissioner in Lunacy for Scotland, later knighted, who, in his study of 3800 deaths in Scottish asylums in 1879, emphasised that the excess death rates were in the younger age groups.\(^{85}\) In 1905, Robert Jones, Superintendent of the London County Asylum (henceforth LCA) at Claybury, reported on a large study that confirmed many of the findings of previous small studies. Of the 96,846 insane persons who had been received into the London asylums since the year 1831 (the


first year in which any records were made) up to 1904, there were 32,258 deaths, a proportion of 33%. He showed that across this large data base that the majority of inmates who died did so within the first year whereas only a minority died after the fifth year of residence. He suggested that the comparative mortality returns in the Registrar-General’s report and those of the LC showed that the death rate in asylums in 1902 was between six and seven times higher than among the general population, even after allowances were made for infant mortality, childhood infections and accidents. The average age of males admitted during 1903 into LCC asylums was 42 years. Jones calculated that the normal expectation of life at this age, based upon actuarial tables, was 24 years (i.e. to 66 years of age) but the average age at death of 721 males dying during 1903 in the asylums of London was 51 years, a gap of 15 years of life.\(^{86}\) Renvoize and Beveridge find that about a third of the deaths in Yorkshire county asylums were under the age of 40 though they also put this in context by commenting that about 40% of asylum admissions at this time were aged 20-40, mirroring the Victorian population which was particularly youthful, with four out of five being under the age of 45 in 1871.\(^{87}\) Careful analysis is thus required yet the tendency of the insane for death at a younger age than predicted by life tables for the general population has been the subject of enquiry of only a few modern commentators.

Melling and Forsythe show that cure rates were the initial standard used to compare asylums but both Leonard Smith and Cathy Smith note that this changed to death rates in the mid-nineteenth century.\(^{88}\) In 1855, Thurnam argued that asylum mortality was a “much less fallacious standard of comparison” than recovery and had the character of a reliable “test.”\(^{89}\) The belief that mortality rates were a good metric to judge the reputation of an asylum became widely held. Medical superintendents, concerned that the raw mortality data was being used to judge them and their asylum, railed against this way of comparing asylums.\(^{90}\) Eyler gives a detailed account of the arguments that raged across the nineteenth century on this topic, pointing out that the arguments

\(^{90}\) Smith, “‘Welcome Release,’” 122.
of both proponents and detractors of this metric had both strengths and weaknesses. Asylums were funded by ratepayers, these figures were of considerable public interest. Annual reports produced by each institution and national statistics were compiled, published, and commented on in journals and in local and national newspapers. Having been used as an argument for reform, the published mortality rates ensured that the death of lunatics remained in the public domain. Cathy Smith describes how statistics on mortality rates became one of the key factors by which asylums came to be seen by the public as failing and how these mortality rates became part of an increasingly negative prognosis for the insane. These conclusions contrasted with the ambitions of those who had earlier advocated the building of public county asylums. As Smith observes, mortality statistics “served the critics of asylums better than their earlier advocates.” A low death rate was seen as an indicator of a healthy environment and effective management, whereas a high death rate was seen as indicating deficiencies in buildings, treatment and management practices which promoted adverse perceptions of individual asylums and the asylum movement in general. Similar issues and trends were linked to death by suicide albeit on a smaller scale. Shepherd and Wright, for example, stress how suicide became linked with Victorian concerns with “bad deaths,” a kind of sore thumb sticking out against “bourgeois values” (such as humanitarianism and progressivist utilitarianism) which permeated the middle classes. Suicide prevention became another bench-mark for judging an asylum’s achievements. A low suicide rate demonstrated the skills of medical superintendents and the staff and the quality of their care whereas even minor comparative increases in suicide frequency prompted uncomfortable questions and unfavourable comparisons. Another consequence of the continued high mortality of asylum inmates during the nineteenth century was its negative impact on how asylums were perceived by those in government and those who championed the asylum movement. The amount of legislative activity relating to asylums from the 1840s to the 1860s suggested parliamentary enthusiasm for such activity, but that period was followed by what Bartlett describes as an “equally loud... legislative silence.” Bartlett relates the reduction in legislation at least in part to the limited success of the asylum

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92 Smith, “Visitation by God,” 111.  
93 Shepherd and Wright, “Madness, Suicide and the Victorian Asylum,” 178.  
movement, manifested by a continued high mortality rate, along with asylums’ growing cost. However, Jones disagrees with this formulation suggesting empowering the LC was seen by politicians as the best way forward.\textsuperscript{95} Whichever was the predominant factor, Smith concludes that since “the numbers of lunatics dying remained little changed after the widespread establishment of county asylums, the medical profession and the asylum movement were left fundamentally exposed and publicly accountable.”\textsuperscript{96} She wryly notes that since asylums were meant to restore physical and mental health, the deaths of disproportionate numbers of their inmates constituted “something of an inconvenience” for their claims to efficacy and that this had implications for the legitimacy of asylumdom and the status of institutional psychiatry.

In a paradoxical way, however, frequent deaths strengthened the position of doctors within asylums, as the involvement of asylum physicians with death was seen as a crucial and widening aspect of their professional role. Ray contends that the frequent occurrence of death in asylums fostered the development of an aspect of doctors’ professional practice which was to conduct post-mortems. He concludes that “The asylum death-rate did not undermine the medical and sick-role construction of the asylum inmate, but in some respects re-enforced it.”\textsuperscript{97} Smith, rather archly, posits that a substantial death rate in the asylums was partly in the interests of the asylum doctors as “It enabled them to retain sufficient capacity in the system to provide for the constant streams of people requiring admission.”\textsuperscript{98} This was, in fact, the practical reality though there is no evidence of the excess deaths being acts of commission. In fact, the medical profession had to account and provide persuasive mitigations for the continued high death rate in asylums, even when the admission of moribund patients to them was beyond their control.

By the later decades of the century, an increasing emphasis on scrutiny, monitoring and central oversight were the hallmarks of the authority’s response to concerns about asylums, including their continuing high mortality. Mellett describes how all deaths in asylums had to be reported to the local Coroner and to the LC. Institutions were named and thereby shamed.\textsuperscript{99} The emergence

\textsuperscript{96} Smith, “‘Visitation by God’,” 111.
\textsuperscript{98} Smith, “Welcome Release,” 122.
\textsuperscript{99} Mellett, “Bureaucracy and Mental Illness,” 221-50.
and effects of this new central oversight of asylums has been evaluated, but historians have not closely examined the Commission’s monitoring of asylum mortality. However, the work of Hervey and Andrews respectively examining the work of the English and Scottish Commissioners shows that the LC could and would intervene when an asylum’s mortality rate was particularly high (though in those cases there were other markers of a poor quality of care).\textsuperscript{100} Whether the specific scrutiny of mortality figures from the majority of asylums had much impact is, however, open to question. The recording of incidents in the asylum including deaths became part of what Arieno calls the increasing “centralised bureaucracy” of Victorian times.\textsuperscript{101} Medical superintendents were obliged to complete more and more paperwork on the treatment of patients, including the number of times mechanical restraint was used. The monitoring of attendants' behaviour also became an increasing focus during the period with some dismissed for neglect and mistreatment.\textsuperscript{102} Medical superintendents were thus placed in a difficult position. An increasing patient population and high patient/staff ratios made the regulation and control of violent and dangerous inmates difficult, yet mechanical restraint, one of their main means of preventing self-harm and aggression, was all but forbidden.\textsuperscript{103} The unremitting high death rate, often of young people, served to heighten both the risks and the tensions.

2.1.2 The nature of the sample: Patients in the Asylum in the Victorian era

This section analyses the demographic profile of admissions to Victorian asylums, and more specifically that of particular groups of patients relevant to this thesis. In doing so, it seeks to provide answers to the questions: what were the factors that led to an individual being admitted to the asylum and did these factors have an impact on the mortality within asylums? The continued high mortality occurred in parallel with the large increase in the numbers of those committed to the asylum across the period. The nineteenth century saw a marked rise in the proportion of the population officially recognized as insane. Walton estimates that during the period 1807-55 the rate of those admitted per 10,000 of the population increased more than sevenfold, from 2.3 to 16.5. The rate of growth eased off in the second half of the century but still

\textsuperscript{100} Hervey, “A Slavish Bowing Down,” 98-131; Andrews, “‘They're in the Trade’.”
\textsuperscript{102} Smith, “Behind Closed Doors,” 301-27.
rose further to 29.3 by 1890. This mirrored, but much outstripped, the rise in the population.\textsuperscript{104} Before the legislation in the earlier part of the nineteenth century which led to the growth of asylums, many families kept their insane relatives at home and tried to cope with them, sometimes in secret. Once the family felt no longer able to do this, there were a number of options: private care in the home, union workhouses, private madhouses, charitable hospitals and county asylums. Few could afford the private madhouses and hospitals and even fewer the costs of private care in the home. Most of the increase in asylum inmates arose from those who had to fall back on the Poor Law for maintenance in a crisis of this kind.\textsuperscript{105} A lunatic was termed a pauper if the money for his or her maintenance came in whole or in part from public funds. The differential in admission rates between the classes was dramatic. Walton finds that up to 1844, more than 80% of the insane were paupers but that over the next two decades the number of pauper lunatics nearly doubled, while private patients increased by only a quarter.\textsuperscript{106} While the term pauper lunatic is routinely imbued with a range of negative meanings and constructions as to social condition and status, data on the occupational and residential backgrounds of patients on admission is often at odds with this image. In a range of asylum studies, the vast majority of inmates were found to have been in work before admission, as, for instance, amply demonstrated in Tobia’s study of the Bristol Asylum.\textsuperscript{107} Arieno found that about 60% of patients were from the “labouring group” and about 35% from the “trades” group.\textsuperscript{108} Those such as vagrants and the long term unemployed made up less than 5% of admissions.

The factors underlying the increase in asylums and asylum dwellers have been much debated, sometimes vigorously.\textsuperscript{109} These putative reasons will be examined with a focus on the nature of admissions and whether high mortality in the asylum was related to the particular type of people admitted. Firstly, was the high mortality related to the increase in transfers from workhouses of particularly vulnerable patients? There is convincing evidence that numbers of transfers from

\textsuperscript{105} Peter Bartlett and David Wright, \textit{Outside the Walls of the Asylum: The History of Care in the Community 1750-2000} (London: A. & C. Black, 1999).
\textsuperscript{108} Arieno, \textit{Victorian Lunatics}, 80.
workhouses to asylums rose across the Victorian period but although the medically unwell and frail, and therefore those more likely to die, were moved, this does not appear to be the main reason behind most transfers. Myers shows that the ratio of the insane in the workhouse to those in asylums fell steadily from 1.16 in 1842 to 0.2 in 1910. Early in this period, it was recommended that most pauper lunatics should be admitted to asylums as there was great criticism of the overcrowded conditions under which lunatics were kept in workhouses, often in attics or basements with no facilities for exercise. In practice however, many lunatics, particularly those who were quiet and easily managed, remained in the workhouse, perhaps, as Bartlett suggests, as some preferred that option. Ellis describes how the introduction of the four-shilling grant in the mid-1870s has been put forward as a factor promoting greater transfer of patients from workhouse to asylum and how commentators suggest that this marked a pivotal point at which the asylum’s “curative pretensions” came under a significant amount of pressure. However, from his study of two County Asylums, Ellis indicates that there is little evidence the grant was responsible for changes in either the size or composition of the asylum population. By the end of the century, even the LC were reconciled to the accommodation of “feeble-minded, imbecile, idiot, chronic psychotic and demented” paupers in workhouses but continued to hold the view that the acutely insane should be admitted to asylums. Dangerousness, attempted suicide and the threat of suicide were important criteria in the decision of workhouse officials to seek admission of patients to an asylum. Carpenter observes that while workhouses sent acute cases which were thought to be curable to an asylum, they appeared to have resisted sending people who were chronically ill to the asylum unless clearly a risk to themselves or others. Thus, although case mixes changed somewhat because of increased transfers from workhouses, it does not seem likely that this factor specifically accounted much for the continued high asylum mortality.

111 Hodgkinson, “Provision for Pauper Lunatics,” 146.
114 Myers, “Workhouse or Asylum,” 576.
Secondly, while asylum admissions from the community also rose markedly across the Victorian period, there is little specific evidence that any change in criteria for admission was responsible for the subsequent mortality of a significant number of the insane. Scull and others claim that alienists were the prime movers behind the surge in admissions and its principal beneficiary.\(^{116}\) Wright however persuasively challenges that notion, countering that while medical men were indeed the beneficiaries, that stemmed from “social forces beyond their control” and in practice asylum doctors complained bitterly at the lack of medical input to the process. Admission to the asylum involved no specialised medical expertise, input, or selection. Wright describes medical superintendents as “peripheral agents” in the confinement of the insane and this view is supported by work done on Devon asylums by Melling’s group.\(^{117}\) Scull initially argued that asylums were “convenient places for inconvenient people.”\(^{118}\) However, other scholars have compellingly challenged this viewpoint and agree that the majority of the insane admitted to asylums were violent to self/others, destructive and radically behaviourally disordered, or, as Walton termed them (arguably, equally problematically) “impossible people.”\(^{119}\) Beveridge also convincingly contests the claim that the asylum was used to incarcerate society’s dissidents and discontents and instead finds that the make-up of the patient population of the REA “reflected that of the general population of Edinburgh.”\(^{120}\)

Scull later adapted his view to “the asylum provided a convenient and culturally legitimate alternative to coping with intolerable (my italics) individuals within the family.” Scull suggests that asylum admission was likely to be most attractive to poor families with fewer resources for coping with dependent and economically unproductive relatives.\(^{121}\) Wright agrees that economic pressure and the shift towards the nuclear family with industrialization led to an eroding of wider

\(^{116}\) Andrew Scull, “Psychiatry and Social Control in the Nineteenth and Twentieth Centuries,” *History of Psychiatry* 2, no. 6 (1991): 149-69.


\(^{118}\) Scull, The Most Solitary of Afflictions, 135, 246.


ties of kinship and community and to increased admissions as the family’s “pragmatic response.” This chimes in with the work of Melling who stresses the impact of factors like urbanisation, industrialisation and migration in increasing asylum admissions. However, research by the Andersons showed that industrialisation actually increased the size and complexity of the urban family, due to housing shortages and kin seeking out relatives on their move to cities. Moreover, data from Tobia’s study of over 5,000 admissions to Bristol Lunatic Asylum in the second half of the nineteenth century convincingly concludes that the “stereotype of the impoverished pauper admitted to the asylum did exist but these patients were certainly in a minority.” Thus, the social factors behind the growth in asylum numbers are myriad and complex and their net effect remains unclear. However, factors like deprivation and displacement are associated with early death and therefore these trends may, in part, explain the continued high death rate of asylums.

Some historians, incline to the view, a broadly correct one in my opinion, based on scrutiny of studies illustrating the apparent severity of the insanity of most of those admitted, that the vast majority of admissions were necessary for the relief of suffering of the patient, their families and the community and not necessarily the result of economic pressures. Smith, for example, comments that, while the asylum might have been “part of a broader master plan to rehabilitate deviants into adopting bourgeois values,” in practice it was the “humanitarian goals that were... more effectively met.” Several historians point out that, rather than the family committing uneconomic family members, lack of family was more often the driver of admissions. For example, Irish patients admitted to the asylums in Liverpool tended to be isolated and without contact with kin. Walton finds that, in industrial Lancashire, tighter kinship groups and communities with stable resources were those with fewer admissions and that families often delayed involving

123 Melling and Forsythe, Insanity, Institutions and Society, 10.
127 Smith, “Family, Community and the Victorian Asylum,” 122.
authorities, in some cases because of shame or guilt. Likewise, detailed research by Melling, Adair and Forsythe in large asylums in Devon identifies the absence of marital partners and a peripheral position in the kinship household as the features likely to be associated with asylum admission. Thus, Scull’s original proposition is likely to be only partly true and the situation more complex. It is noteworthy, however, that social isolation seems to be a clear factor behind the growth in admissions and, as discussed in later chapters, is known to be a stressor associated with proneness to mortality.

Hare argues that the growth of asylums was related to an increase in the incidence of insanity and bases this assertion on an increase in the number of first admissions. However, in my view and that of others, he makes a large number of questionable assumptions while so doing. Beveridge, for example, in his commentary on cases admitted to the Royal Edinburgh Asylum (REA), is one of the many unconvinced and Scull wrote a vigorous riposte. Scull contends that asylums reached capacity because “families were encouraged to place their mentally imbalanced relatives where they could receive professional care and treatment.” Another factor is that patients themselves may have pushed for asylum admission because of economic necessity. Bartlett underlines how a high proportion of admitted patients were very deprived and to them “a life of farm work, sewing, quoits on the lawn and dances every week coupled with three square meals a day must have appeared almost idyllic.” Shepherd emphasises that the therapeutic environment of work, pastimes and entertainment continued in pauper asylums even as numbers grew. Bartlett also hints at an “implied alliance...between relatively able paupers actively pursuing admission and the asylum staff.” Scull’s and Bartlett’s views are predicated on the notion that the increase in admissions was largely due to the addition of lots of milder cases. However, Hare argues (and provides back up data) that there is no evidence for cases being milder on admission. Several studies which have investigated case notes and applied retrospective classification to them have shown that asylum patients, in the main, had major mental problems. For example, most patients admitted to the Yorkshire asylums were deemed to be severely disturbed mentally and similar

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134 Shepherd, Institutionalizing the Insane, 127-32.
findings were reported in studies of Ticehurst, Denbigh, Fife and Edinburgh asylums.\textsuperscript{135} However, the perils of retrospective diagnoses are many, based as they are on the assumption that the nature of insanity is unchanging.\textsuperscript{136} Turner argues that the process has some validity but more scholars argue that diagnosis of mental illness is culture-bound and that those who attempt to retrospectively diagnose patients who are long dead utilise data that is often compromised and conditioned by the oft flawed paradigms of a specific time.\textsuperscript{137} Furthermore, the difficulties of retrospective labelling are magnified when one considers that the Victorian period was an era when epilepsy and insanity were often confused and when concepts such as moral insanity and masturbatory insanity were applied to populations. So, in the end, a clear answer to the size of the impact of these various factors on asylum numbers and/or asylum mortality continues to be elusive.

What seems less contestable and more generally accepted in modern scholarship is that while the growth in the number of asylum inmates partly arose from a widening of the definition of insanity, a widening of the certificatory criteria for asylum admission generally had the greater impact.\textsuperscript{138} Asylums moved substantially beyond the earlier preoccupations with the dangerous insane to embrace much broader categories of mental derangement, including those manifesting delusional disorders (without a history of violence). Legislation and perhaps financial incentives also placed more emphasis on those unable to take care of themselves.\textsuperscript{139} Scull argues that the decision of central government to grant each parish a four shilling subsidy for each lunatic confined in the asylum acted as a financial incentive to the growth of numbers in the asylum.\textsuperscript{140} Sturdy and Parry-Jones point to the grant’s effects in reducing the numbers of those boarded with their relatives


\textsuperscript{140} Andrew Scull, \textit{The Most Solitary of Afflictions}, 370.
and conclude that it had a negative impact on the curative nature of asylums.\textsuperscript{141} Broader categories of patients such as those with epilepsy and paralysis (who were largely excluded from admission to eighteenth-century lunatic hospitals), those with alcohol and drug related conditions, with neurological afflictions and with learning disabilities became larger proportions of the institutionalised population by 1900 than they had been in 1800. In addition, in the later part of the nineteenth and early twentieth century there were a growing number of older patients within the asylum. The renowned REA Medical Superintendent and Insanity Lecturer Thomas Clouston complained in his 1904 Annual Report that the rise in mortality between 1873 and 1906 was due to an increasingly aged and frail group of patients being admitted.\textsuperscript{142} Prime reasons for their admission included inability to cope in their household, as well as lack of familial enthusiasm to maintain them at home any longer. In some instances, older inmates had encountered abuse by their relatives, as well as themselves inflicting violence on their family members.\textsuperscript{143} In addition, studies of a number of individual English pauper asylums document a general decline in recovery rates during the latter part of the century. Scull observes that the recovery rate fell markedly between 1870 and 1890, paralleling similar findings at Devon asylums.\textsuperscript{144} Hare identifies a general fall in recovery rates for asylums in England and Wales but considers that it began only in the 1890s, a period in which Tobia also finds recovery rates fell to an all-time low.\textsuperscript{145} The consequent large numbers of chronic cases of these types made asylums seem to several commentators to be places of containment more than hospitals - in Hodgkinson’s words “more like refuges for incurables instead of hospitals for the treatment of disease.”\textsuperscript{146} The inclusion of these groups and their effects on asylum case-mix are likely to have had a substantial impact on the frequency and demography of asylum mortality.

Finally, while admissions do not seem to have been selected on the basis of physical morbidity per se, there is general agreement between historians on the insane’s poor physical health both at

\begin{itemize}
  \item Allan Beveridge, “Madness in Victorian Edinburgh,” 152.
  \item Scull, \textit{Museums of Madness}, 269-76; Melling and Forsythe, \textit{The Politics of Madness}, 178-183.
\end{itemize}
admission and during chronic incarceration. For example, in the mid-1870s, 25% of all cases in one Scottish district asylum were either “epileptic or syphilitic,” tuberculosis was found in 20-30%, while heart and lung disease was commonplace. Several studies show that arrival in poor condition, with poor prospects of recovery was especially seen in men. Melling and colleagues demonstrate that the circumstances of admission had a large bearing on death rates with a particularly high death rate in males in the first days, weeks or months of admission. Women admitted to Devon asylums in a healthy physical state often survived for long periods before they died but those who died within the first six months of admission were much more likely to be described as feeble and in poor health on admission. Those admitted to asylums in Liverpool who were of Irish extraction (many had migrated post-famines) show this phenomenon particularly clearly, with a high burden of ill-health and emaciation from destitution and extreme hardship. They were described as “ravaged by the effects of bad living-problems such as marked social isolation, erratic lives and intemperance.” These patients also had high levels of physical exhaustion which was related to factors such as “tramping in search of work... and lack of food” exacerbated by mental disorder, particularly mania. Mania was diagnosed in 20% of non-Irish patients in Rainhill Asylum but, in contrast, over half of its Irish patients were diagnosed with mania. These patients were also excessively disturbed, unruly and volatile and their death rate in the first few months of their admission approached and sometimes exceeded 50%. Cox and colleagues comment that asylum doctors tended to fuse “poverty and urban living with ideas of degeneration” sometimes leading to stereotyping of a racial nature, as in the case of the Irish. Scull insists that the increasing focus on degeneration theory in asylums in the late nineteenth century, and its eugenic undertones, became itself “an unimpeachable argument” for asylum growth and “mass segregation.”

In summary, frequent deaths of relatively young people, particularly males, in the months after admission to an asylum is a major feature of this literature. The evidence points to the high mortality and reduced longevity not simply being directly caused by poverty and inter-current

147 Doody, Beveridge and Johnstone, “Poor and Mad,” 887-97.
150 Andrew Scull, “Museums of Madness Revisited” in The Insanity of Place/the Place of Insanity (London: Routledge, 2006), 83.
physical illness but rather multifactorial and related to complex factors in the case mix which changed as the century progressed.

2.1.3 The causes of mortality in Asylums

Two main scholarly narratives have underpinned our understanding of the causes of premature deaths in asylums: i) deaths ascribed primarily to those admitted after being unwell for a significant period, typically accompanied by physical ill-health and marked weight loss, whose condition was further worsened by asylum conditions; and ii) deaths ascribed to those whose insanity was acute and rapidly advancing, so that death appeared to be direct consequence of their mental disorder. The former was numerically greater than the latter, but Smith charts the increasing emphasis by alienists on the importance of the latter group, highlighting how those with severe manias and melancholias were at the greatest risk of untimely demise.\(^{151}\)

In strong support of the first narrative was Scull, who contends that people were committed to the asylum to be nursed to their death and that asylums had become “the resting place for the broken-down and physically decrepit,” a view shared by a number of contemporary alienists and historians.\(^{152}\) However, the phenomenon of death soon after admission was also seen in a private asylum where there was much less physical ill health on admission but still a relatively early death of males, particularly the husbands. Melling and Forsythe speculate that husbands were considered more valuable to the household and were more likely to be cared for at home for longer, until they reached a more advanced stage of illness.\(^{153}\) Melling and colleagues also examined the reports of the physical examination of those admitted to Exminster during 1880-82 and found that men were on the whole in good or fair health but nonetheless the death rate in males was high and their death was often premature.\(^{154}\) The phenomenon of physically frail, disturbed patients being admitted and dying soon after lessened as the century progressed, as


\(^{152}\) Scull, *Museums of Madness*, 115; Jones, “Prognosis in Mental Diseases,” 1579; Doody, Beveridge and Johnstone, “Poor and Mad,” 887-97; Shepherd, *Institutionalizing the Insane*, 140.


strikingly shown by Tobia in his detailed study of the Bristol Asylum.\textsuperscript{155} Yet death rates, particularly premature deaths, remained elevated at much the same rate.

A number of historians have questioned whether the cause of the insanity ascribed at admission has some bearing on subsequent mortality, but this enquiry is complicated by the fact that, while such attributions to admissions were commonplace, the process was for the large part arbitrary. Ray observes that there was a “keenness to emphasise moral causes” of insanity as opposed to heredity or physical causes which the family also supported because of the stigma of lunacy.\textsuperscript{156} Jacyna argues that this separation was artificial and did not reflect the true picture; clearly this was the case in relation to death.\textsuperscript{157} In Ray’s study of the Lancaster Asylum, a case with the ascription of moral causes was just as likely to die prematurely and early as those without such a label. Another potential factor that has been considered to explain the early demise of asylum patients is the psychological impact of insanity and of admission but again the picture is far from clear-cut. Once admitted, the insane were not treated uniformly. Each asylum had its own set of rules and evolved its own traditions and culture. There was no “hegemonic model of treatment,” although some scholars assert that the LC legitimised a medical model of insanity in which medical superintendents and their staff exercised considerable control over the residence and treatment of patients.\textsuperscript{158} Mellett and others argue that the Commissioners’ medicalization of insanity became particularly prominent in in the face of growing "therapeutic nihilism" ("the sombre years of neo-Darwinian pessimism and eugenics doctrines" that Adair and colleagues describe) and that this model particularly applied to pauper patients, who thereby had a reduced sense of agency.\textsuperscript{159} Incarceration brought with it the stress of not being in control of one’s destiny, though a minority of scholars have utilised sources which challenge the “notion of powerless and inarticulate pauper families,” as does Shepherd reviewing patients’ letters at Brookwood asylum.\textsuperscript{160} Cramner proposes a number of behavioural reasons why morbidity and mortality were high in those committed to

\textsuperscript{155} Tobia, “The Patients of the Bristol Lunatic Asylum,” 128.
\textsuperscript{156} Ray, “Models of Madness,” 229-64; Smith, “‘Visitation by God’,” 107.
\textsuperscript{158} Melling and Forsythe, The Politics of Madness, 6.
\textsuperscript{160} Shepherd, Institutionalizing the Insane, 11.
the asylum and puts emphasis on how the insane “can be self-neglectful, unconcerned about chills or food.”\textsuperscript{161} The historical data on asylum mortality has been considered by Sims who advances a number of psychological theories, including evidence for the importance of negative life events such as loss (e.g. bereavement) on mortality.\textsuperscript{162} These interesting lines of enquiry have not been the subject of extensive or comparative study however and so it is difficult to discern whether the excess mortality, particularly in younger patients, was driven primarily by psychological, behavioural, biological or social factors. Given the methodological strictures which abound in this area, the difficulties for this type of analysis are likely to continue.

The phenomenon of premature death in the insane early in the admission of males raises the question of intemperance as a potential associated factor into focus. There has been a longstanding debate about the relationship between intemperance and insanity but there is surprisingly little work on the role alcohol abuse (in its various forms) might have had to play in the excess death in people admitted to asylums. Thompson documents a connection between alcohol abuse and neurosyphilis but this is not explored further.\textsuperscript{163} Porter shows how cases of insanity were attributed to “drink and intoxication” at Bethlem Royal in the early part of the nineteenth century, and the idea of a close and common relationship came to be held particularly strongly in Scotland in the later parts of the century.\textsuperscript{164} In 1874, David Yellowlees, later Physician Superintendent of Glasgow Royal Asylum, opined that “It is surely within the truth to assert that half the existing cases of insanity are due directly or indirectly to this social curse.”\textsuperscript{165} Halliday stresses the range of evidence during the 1880s-early 1900s showing that, in almost a third of those received at two large Glasgow pauper asylums (Gartloch and Woodilee), the cause of the insanity was linked directly to alcohol, while alcohol also accounted for many relapses and readmission to chartered asylums, including Crichton Royal.\textsuperscript{166} However, doubts about this relationship continue to surface and most commentators agree these latter figures are unlikely

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\textsuperscript{165} David Yellowlees, “Insanity and Intemperance,” \textit{British Medical Journal} 666 (1873): 394.
\textsuperscript{166} Emma Catherine Halliday, “Themes in Scottish Asylum Culture: The Hospitalisation of the Scottish Asylum 1880-1914” (PhD diss., University of Stirling, 2003), 144-45.
\end{flushleft}
rates and numbers. Halliday observes how leading late Victorian alienists, like Thomas Clouston and Hamilton C. Marr, were averse to asylums serving as receptacles for chronic alcoholism, and that it was often poorhouses instead which served as holding bays for such cases. Arguably, Victorians were over-diagnosing insanity as a condition of toxic alcohol induced pathology. Indeed, the late Victorian period had reduced rates of intemperance compared with earlier eras, partly due to influential temperance and inebriate reformatory campaigns. In 1876, Farr stated that the deaths from alcohol were rapidly deceasing, and that they were very few in number - less than 1,500 persons in England. Mott, a pathologist for the LCA, stated that while alcohol had been asserted as a cause of or an association with insanity on admission for between 15 and 30% of admissions to London asylums, he was surprised to report that out of 1200 post-mortems at Claybury Asylum, only 2% died with any evidence of liver cirrhosis and so came to doubt the high rate of attribution of alcohol as a cause of insanity. What is clear from the foregoing is that there is little or no direct evidence for alcohol abuse being having a strong link to asylum mortality and that more research is needed on this particular topic.

Suicide did not account for the youthful mortality. Despite the frequency of suicidal thoughts and behaviour of admitted patients, Shepherd and Wright summarise evidence to show that the total mortality for suicide in UK asylums never exceeded 20 in number per year compared with the approximately 1,500 suicides per year in the community. Examination of data from the REA confirms the low frequency of death by suicide, with it accounting for less than 1% of all deaths there. Although suicidal ideation was recorded in a third of the patients on admission to the REA, none of these succeeded in killing themselves while in-patients. Some historians attribute this to the close observation and protection afforded to the suicidal patient by the asylum attendants, although most scholars are more circumspect in supporting the proposition that the asylum prevented many deaths by suicide. Shepherd and Wright conclude that in fact relatively few patients were “actively suicidal,” if this was defined as “patients who have made serious

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167 Shepherd, *Institutionalizing the Insane*, 124-5; Edward Hare,” The Two Manias,” 83.
172 Shepherd and Wright, “Madness, Suicide and the Victorian Asylum,” 177.
attempts on their lives, and [were] likely to repeat them.” The extent to which preventive measures were successful depends on how many individuals would have killed themselves were it not for the intervention of the admission and the surveillance of the staff and there is a lot of uncertainty around this.

An important question is whether admission to the asylum itself hastened patients’ demise and there is a significant discourse centred on the environmental factors of asylum life which might exhibit potential associations with death. Although insanity outside the asylum in different, or non-professional, settings has been studied, there is relatively little useful comparative data on mortality of the insane in these settings. Emetics and purgatives, and cold or warm baths were commonly used for mania. It has been postulated that these techniques, which historians have commonly termed “heroic” (if vigorously used), or simply “depletive,” could further weaken the patient’s constitution and increase the chances of a fatal outcome. More definitively, a few case reports describe deaths occurring during or shortly after mechanical restraint. However, the use of emetics and purges had diminished markedly by the time of this study and had largely been replaced by use of chemical treatments (whose potential impact is discussed in the next chapter). Similarly, the non-restraint movement along with closer monitoring of restraint and greater use of seclusion at the time of study diminished the effect of restraint as an important factor in asylum mortality.

Asylums were overcrowded and therefore potential breeding grounds for infectious diseases and some scholars have seen these factors as of direct importance in the high asylum mortality. Deaths from tuberculosis and pneumonia were common and, particularly in the earlier part of the Victorian era, crowded unsanitary institutions could encourage epidemics of these infections and other highly infectious disorders like dysentery and sometimes, even more fatally, cholera. Leonard Smith describes the proneness of asylum inmates to infectious diseases but, such deaths became less frequent as conditions at least partially improved. As Ernst underlines, these infections particularly, though not exclusively, affect those already suffering from a fragile state of

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175 Shepherd and Wright, “Madness, Suicide and the Victorian Asylum,” 177.
health on account of other illnesses, including insanity.\textsuperscript{179} It is often not clear if those who succumbed to these infections in asylums developed them on top of other medical conditions and/or if they were the immediate causes of death. A number of scholars also make the point that there are difficulties in ascertaining if these infectious diseases were contracted inside the asylum, or if they were already present on admission.\textsuperscript{180} Renvoize and Beveridge provide evidence to support the contention that high patient: staff ratios and poor environmental conditions contributed to the increased death rate.\textsuperscript{181} A similar picture was reported from Exminster Asylum in Devon, where the “inexorable pressure on its accommodation and staff” with high ratios of patients to staff were associated with periods when death rates peaked.\textsuperscript{182} Associations between overcrowding, high use of restraint and high mortality rate were found in records of the Govan Asylum in the 1880s.\textsuperscript{183} However, as summarised by Wright and colleagues, there is overall little direct evidence that there is an association between the total numbers in an asylum (or the extent of overcrowding within it) and the mortality rate.\textsuperscript{184}

Another potentially negative impact of asylums upon patients’ lifespans was abuse of inmates by staff. Low calibre of staff was seen by many at the time to be a major factor contributing to the failure of the Victorian asylum.\textsuperscript{185} Melling and Forsythe, and Smith document the frequent complaints of violent treatment and, as Smith emphasises, the threat of violence or punishment was often used as a coercive force.\textsuperscript{186} However, Russell suggests that the temptation to scapegoat the attendants for the failures of the asylum was great as it provided an easy means of deflecting blame from more highly placed quarters.\textsuperscript{187} Many complaints appear to have been rejected on enquiry. Of particular concern during this period was the number of fractures, particularly of ribs, found at post mortem. Asylums were accused of wilful mistreatment on this basis but further study, summarised by Wallis, showed that the insane were peculiarly prone to bone disease.\textsuperscript{188} Alienists of the period had come to a similar viewpoint. George Jonathan Hearder, the Medical

\begin{footnotesize}
\textsuperscript{179} Ernst, “The Limits of Comparison,” 404-18.
\textsuperscript{180} Wright, Jacklin and Themeles. “Dying to Get Out of the Asylum,” 606-08.
\textsuperscript{181} Renvoize and Beveridge, “Mental Illness and the Late Victorians,” 19-28.
\textsuperscript{182} Forsythe, Melling and Adair, “The New Poor Law and the County Pauper Lunatic Asylum,” 335-55
\textsuperscript{184} Wright, Jacklin and Themeles, “Dying to Get Out of the Asylum,” 599–600.
\textsuperscript{185} Smith, “Behind Closed Doors,” 301-327.
\textsuperscript{186} Melling and Forsythe, The Politics of Madness, 183.
\end{footnotesize}
Superintendent at the Joint Counties' Lunatic Asylum, Carmarthen, argued that the fracturing of ribs could have happened at any stage in life and that they were only discovered during a post-mortem. Hearder was convinced that asylum officers were subject to “unjust odium” and that the high numbers of patients who did not suffer fractured bones was indicative of the “very great care” they received.\textsuperscript{189} On this theme, superintendents often referred to the returning diaspora of discharged cases, as evidence that they had been fairly treated. Lee-Ann Monk points to evidence suggesting that asylum culture saw some forms of violence, for example in reprisal or self-defence against a violent patient, as legitimate, and that inmates could make the distinction between rough treatment and cruelty.\textsuperscript{190} At the time, however, the emphasis on fractured ribs undoubtedly added to the brutish portrait of the asylum attendant and the notion that violence was potentially a key component of asylum mortality. Evidence for and against this hypothesis is sought in this thesis.\textsuperscript{191}

There is general acceptance that a key factor in asylum mortality was the high prevalence of physical disorders among the insane. Many scholars point out that insanity made its sufferers vulnerable to other diseases, often serious, echoing Andrews’ account of the alienist Pritchard, who, many years earlier, had “affirmed the dangerous susceptibility of the insane to congestive diseases of the bowels, liver, heart and intestine” and lunatics’ “peculiar propensity to various disorders profoundly deleterious to organic life.”\textsuperscript{192} Another reason for the conjunction of fatal medical disorders and insanity was that there were some physical disorders in which mental disturbance was part of the clinical picture e.g. epilepsy and general paralysis of the insane (GPI). These conditions led to a range, variously attributed, of between 15 and 25% of all deaths. Furthermore, inmates were not immune to suffering the physical diseases of the general population - many died from heart conditions, strokes, cancers, or other conditions that would account for deaths in the community. Deaths from cardiovascular disease and stroke were common in each of the three York asylums even in relatively young patients aged 20 to 40 years.\textsuperscript{193} The average age at death of asylum patients rose across the period mirroring the

\textsuperscript{189} Robert Ellis, “A Field of Practise or a Mere House of Detention? : The Asylum and its Integration, with Special Reference to the County Asylums of Yorkshire, c. 1844-1888” (PhD diss, University of Huddersfield, 2001), 248.


\textsuperscript{191} David Wright, “The Dregs of Society?” 5-19.


\textsuperscript{193} Renvoize and Beveridge, “Mental Illness and the Late Victorians,” 19-28.
increased longevity of the general population but lagged behind it by approximately 15 years throughout. What lay behind this prematurity of asylum death is a major part of this study.

Cathy Smith observes that “death from insanity mattered in the nineteenth century” and that “death in numbers posed further questions.” In the 1860s death could still be attributed to the intervention of the Almighty, evidenced by some patients having their cause of death recorded as “Visitation by God.” In the later stages of the century, a materialist conception of death predominated but, even then, despite the growth in autopsy science, metaphysical explanations were sometimes suggested. Diagnoses of death remained “vague and imprecise” even in the late nineteenth century. Numerous scholars note that deaths were often casually attributed to “general decay,” “exhaustion after mania” or “debility after melancholia,” reflecting the uncertainty in ascribing the cause of death and the limitations of pathological investigation. Smith suggests this imprecision was deliberate to excuse the medical profession from direct culpability in the deaths of their patients. All these observations beg questions which this chapter seeks to answer with a detailed study of Berkshire Asylum mortality data obtained from the Register of Deaths. The procedures surrounding deaths, burials and post-mortems (and the detail of their reports) which took place here and at Sunnyside are discussed in the next chapter and set in the context of the burgeoning of dead-houses (mortuaries) and post-mortems across Britain.

2.2 Analysis of Causes of Death at the Berkshire Asylum, 1896-1905

Deaths in the Berkshire Asylum were recorded in the Register of Deaths and Discharges. Information about numbers in the asylum and deaths was also obtained from the General Statement Books (1896 –1902, 1902 - 1909) and the medical superintendent’s Reports to the

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197 Smith, “‘Visitation by God’,” 109.
Visitors which included monthly mortality statistics. There were 613 pauper deaths in the Berkshire Asylum across the ten years from 1896 to 1905, the annual distribution of which is shown in Figure 1. (The number of private patients’ deaths is very small and these cases have not been included).

Figure 2-1 Deaths per annum across a ten-year period 1896-1905 at the Berkshire Asylum (blue columns) and numbers of post-mortems per annum (red columns). Figures are obtained from the Pathological Records.

The number of patients in the Berkshire Asylum rose gradually but inexorably from 580 in 1896 to 720 in 1905, much to the chagrin of the medical superintendent and the Board of Visitors. Although there was an active building programme at this time, the increased accommodation was not available until the early part of the twentieth century, leading to reports of substantial overcrowding from 1899 to 1902. This may have played a part in the higher annual death rate (deaths per total number of inpatients) in those years, peaking at 13% in 1900, compared with the lower figures at the beginning (8% in 1896) and end (9% in 1905) of the period. The latter figures are in keeping with the average 8.2% reported by the LC for 1890 for all county and borough asylums and Hare’s summary of mortality in insanity across all asylums in this period of about 10% per annum.\footnote{198} The increased death rates in the middle years were attributed locally to concerns over the quality of the water supply and sewage disposal. There was a slight but definite increase in deaths from infections such as dysentery and enteric fever in those years, conditions which may have been linked to the problems with sanitation. A similar pattern of deaths from gastrointestinal diseases was observed.

infection linked to water supply problems was seen at the Glenside Asylum Bristol during the same period.\textsuperscript{199}

Figure 1 shows that the post-mortem rate during this period was high. The rate reached 100% for several of the years and was never lower than 88%. These figures are higher than have been reported for many asylums. This may have reflected the rigorous control exerted by William Murdoch, the medical superintendent who was a stickler for regulations, but there was also significant external pressure from the LC and corporate psychiatric quarters, such as the Medico-Psychological Association (MPA) and its journal, to maximise asylum post-mortem rates.\textsuperscript{200} Another factor boosting the enhanced rate at Fair Mile was the practice that consent of relatives for a post-mortem was tacitly assumed (see Section 3.1.1.1 for commentary on this issue and examples of this practice in the current study).

Across the ten-year period there were 613 deaths. The Register was scrutinised to glean the limited information contained therein: name, age at death, mental condition on admission and at death, cause of death and whether a post-mortem was performed. Examination of the Register and the post-mortem reports showed that it seems likely that the Register was completed after the post-mortem report had been written as the same words are used in both. Of the 613 cases, there was no clinical label given either at admission or at death for five cases and these have been excluded from the further analysis.\textsuperscript{201} The remaining 608 cases fell into six broad clinical categories:- i) Mania (which included common cases of acute, chronic and senile mania as well as more rarely recorded recurrent mania and delusional insanity); ii) Melancholia (including acute and senile melancholia, rarely with stupor); iii) Dementia (usually just dementia but occasionally secondary dementia and rarely primary dementia); iv) Idiocy (sometimes labelled as imbecility, occasionally with mania or dementia); v) Epilepsy (occasionally with mania) and vi) General Paralysis of the Insane (GPI).

The cause of death in insanity (which was comprised of mania and melancholia) is the main focus of this work and so the development of the concepts of these conditions across the nineteenth century and the theoretical principles on which these labels were based is briefly traced. Berrios

\textsuperscript{200} Wheeler, \textit{Fair Mile Hospital}, 21; Andrews, "Death and the Dead-house," 11.
\textsuperscript{201} There was nothing remarkable about the causes of death for these cases.
shows how both conditions were redefined as disorders of affect (mood) rather than of intellect or cognition but that in both cases aberrant behaviour was the cornerstone of diagnosis in practice. Mania was the most common label covering a wide range of overactive and/or psychotic manifestations. Melancholia was the second most common form of mental disease diagnosed in British asylums, and, by the latter part of the century, it was also one of the more standardised and homogenous psychiatric diagnoses in terms of its symptom picture. A few decades earlier, however, the nosological status of melancholia in Britain had been much less clear, its symptomatology diverse and inconsistently overlapping with other conditions like monomania and moral insanity. Thus, while the term melancholia has been used to denote a form of illness or madness since antiquity, the biomedical model of melancholia that emerged in the mid-nineteenth century was historically new and conceptually different from any earlier meanings of the term. The influential alienist Clouston was one of the first and most powerful proponents of the clinical integrity of melancholia.

In the Berkshire sample, the death rate averaged 9% per annum. The percentage of deaths in each of the groupings was Mania (27%), Melancholia (12%), Dementia (28%), GPI (12%), Idiocy (12%), Epilepsy (10%). Melling and Forsythe are seemingly the only modern scholars who have comparable data. In their large study of Exminster Asylum between 1845 and 1914, a sample of 4000 residents (out of a total of 15000 admissions over that period) was studied in detail. The death rate was much higher at 37%. Deaths were grouped into four broad diagnostic groups. 40% of the deaths were in the Mania group, 20% in the Melancholia group, 32% in the Dementia group and 8% in the GPI group. It appears that idiots or epileptics have been excluded from this sample and, if this exclusion is performed on the current sample, a broadly similar profile of deaths is found. There is, however, likely to be a lot of spurious accuracy in both these sets of figures as both their methodologies are very broad brush. Caution is also necessary in performing any comparison between populations as institutional mortality figures adjusted for age, gender and

patient intake are not widely available. Most of the available asylum figures are crude mortality rates (henceforth, CMR), rather than age-adjusted, standardized mortality rates (henceforth, SMR). SMRs are considered to be essential for comparisons over time and place. Tunstall-Pedoe indicates that while the CMR reflects the real number of deaths at a particular time, without standardisation for age, the CMR is always misleading.\(^{206}\) This phenomenon can be shown using asylum data. Farr made an attempt to examine mortality in asylums using the average age of patients, comparing that with mortality in a similarly aged group in the community.\(^ {207}\) This method showed that mortality in the asylums was about 5 times higher than that in the general population. However, a recent study on deaths in Hatton Asylum, Worcester calculates the number of years each patient who died was resident in the asylum during 5-year age periods. This data was then used to calculate a SMR using census data-derived age and sex-specific death risks from the local population for comparison, and this showed a SMR of 2.6. The method used by Farr was also carried out on the Hatton data which showed an SMR more than twice as high (at 5.4), suggesting that, without more precise age stratification, Farr’s method greatly over-estimated the true mortality rate in asylums.\(^{208}\) Thus, in the absence of age stratified data from both asylums and the local population from which the asylum patients were drawn, comparisons between asylums, with the general population and over time, are likely to be misleading. However, the proportions of death in diagnostic categories within an asylum over a shortish period may provide some useful information.

Of the 608 cases, the cause of death for 71 was GPI, an almost invariably fatal form of neurosyphilis. Although the possibility of syphilis had been suggested as a cause of this syndrome as early as 1857, this was not generally accepted and the disease was thought to be multi-causal and related largely to the destructive influences of the urban environment and, in particular, to excesses of alcohol, tobacco and sex, until pathological and bacteriological research in the later part of the nineteenth century.\(^ {209}\) The vast majority of the GPI deaths here had GPI (or an equivalent like general paresis) as the mental state on admission and at death. In three cases, GPI


\(^{209}\) Davis, “The Most Deadly Disease of Asylumdom,” 270.
was not the condition ascribed on admission - one of the cases was admitted as acute mania and two were labelled as dementia. This indicates that these three cases were mislabelled in life and the true condition only identified after death, a low rate of this misdiagnosis compared with earlier in the Victorian period. However, there may have been other neurological conditions manifesting as insanity mislabelled during life and indeed at post-mortem. Melling and Forsythe in their study of Devon asylums in the 1880s conclude that “there is good reason to believe that the term [GPI] was widely used ... to encompass a wide range of disorders rather than restricted to symptoms of syphilitic infection.”210 Davis observes that in some cases the diagnosis could not formally be given until “pathological proof” in the brain was uncovered at post-mortem.211

However, by the time of the deaths in the current study, there was widespread knowledge of the clear and pathognomonic signs of GPI at post-mortem and these markers were found in all cases ascribed as GPI. In 1902, Charles Mercier (Lecturer on Neurology and Insanity, Westminster Hospital Medical School) stated that “there is no other malady in which the same combination of pathological changes is found,” which suggests that, although there may have been a few misattributions in this post-mortem series in cases not clinically labelled as GPI, they were not common.212

The average age at death of this predominantly male group (the male to female ratio was 4.8:1) of GPI sufferers was 41 years with a range from 27 to 60 years old which conforms to much of the data in Davis’s classic paper. The average age of the GPI deaths at the REA was 42 and male GPI admissions outnumbered females by a ratio of 4.1:1. The frequency of death from GPI found in the current sample (11%) also conforms to the pattern reported by Davis, who showed that GPI accounted for between 20 and 28% of asylum deaths in the larger urban institutions, 12 to 20% of mortality in the smaller urban institutions (Berkshire Asylum served several middle sized towns as well as a rural population) and below 10% in the small, rural hospitals in Scotland. The mode of death for cases of GPI was stated for a few as pneumonia or exhaustion, usually as a secondary cause of death, but the mode of death of the GPI sufferers is not further discussed here and these cases have been excluded from the analysis that follows.

211 Davis, “The Most Deadly Disease of Asylumdom,” 266-73.
2.2.1 Age at death

The average age at death in the Berkshire Asylum over the ten-year period of this study was 56 years, with the GPI cases excluded. In all groups there was a considerable range and variance, as detailed in Appendix B (which documents the ages and causes of death of the six diagnostic subgroups). This overall age at death is driven by the younger average age at death of the idiocy group (39 years) and the epilepsy group (45 years). Some of the secondary literature on the topic of asylum deaths ignores the presence of the above groups and discusses premature deaths in insanity as if the whole population of the asylum were suffering from insanity, eschewing recognition that asylums served as places of refuge for those cases of idiocy, epilepsy, or GPI not manageable or managed in the community.

The age at death of the groups more conventionally considered as insane, those with mania and melancholia, was 58 and 57 respectively. As denoted in Appendix B, the mania and melancholia groups contained a number of people labelled as senile mania (n=24) or senile melancholia (n= 5). The age range of those so labelled was quite narrow (62 to 85) and the average age of death of this group was 72, distinct from the mania not so labelled and more akin to those labelled as dementia. There is an argument that senile cases were qualitatively different from those with acute mania/melancholia and that the differences were not just a matter of age. This point is discussed in more detail in the following chapter where case-notes have also been examined. If those suffering from the senile forms of mania and melancholia are excluded, the average age of death in mania falls to 55 and in melancholia to 56. This average age of death was used to select a younger insane population for more detailed analysis in Chapter 3. The average age at death of the 169 people labelled as suffering from dementia was 74 years. However, this average covered a huge age range from 22 to 88, with a quarter of cases under the age of 60. Some but by no means all of these younger cases were labelled as having “secondary dementia” (see Appendix B where these cases are denoted).

2.2.2 Causes of death amongst non-GPI cases

The causes of death for the 537 cases remaining after the 71 cases of GPI are excluded were placed into 5 broad categories. The detail of this method is shown in Appendix C. The frequency of these broad cause of death groupings in each clinical subgroup is shown in Table 2.1.
<table>
<thead>
<tr>
<th>GROUP</th>
<th>Total number of deaths</th>
<th>CAUSE OF DEATH IN REGISTER OF DEATHS (BERKSHIRE ASYLUM)</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Exhaustion n (%)</td>
<td>Brain Conditions (e.g., Senile decay/Cerebral softening/Cerebral atrophy) n (%)</td>
<td>Vascular disease (e.g., Morbis cordis/Heart disease/Heart failure, CVA) n (%)</td>
<td>Infection n (%)</td>
<td>Other n (%)</td>
</tr>
<tr>
<td>Mania</td>
<td>165</td>
<td>26 (16)</td>
<td>39 (24)</td>
<td>36 (22)</td>
<td>54 (33)</td>
<td>10 (6)</td>
</tr>
<tr>
<td>Melancholia</td>
<td>70</td>
<td>7 (10)</td>
<td>16 (21)</td>
<td>17 (26)</td>
<td>25 (36)</td>
<td>5 (7)</td>
</tr>
<tr>
<td>Dementia</td>
<td>169</td>
<td>1 (0)</td>
<td>88 (53)</td>
<td>21 (12)</td>
<td>52 (31)</td>
<td>7 (4)</td>
</tr>
<tr>
<td>Idiocy</td>
<td>72</td>
<td>3 (4)</td>
<td>12 (17)</td>
<td>7 (10)</td>
<td>34 (47)</td>
<td>16 (22)</td>
</tr>
<tr>
<td>Epilepsy</td>
<td>61</td>
<td>11 (18)</td>
<td>9 (15)</td>
<td>9 (15)</td>
<td>16 (26)</td>
<td>16 (26)</td>
</tr>
<tr>
<td>Total (% of all deaths)</td>
<td>537</td>
<td>48 (9)</td>
<td>164 (30)</td>
<td>90 (17)</td>
<td>181 (34)</td>
<td>54 (10)</td>
</tr>
</tbody>
</table>

Table 2-1 Causes of death put into 5 categories (exhaustion, brain conditions, vascular diseases, infections, and others). Causes of death in the Register were usually short descriptors and usually only one cause of death was stated. For the purposes of the analysis here, in the relatively rare instances when two causes were stated (e.g. heart disease and pneumonia), only the first cause of death has been used.

Exhaustion was a fairly common cause of death comprising 9% of cases overall. It was rarely a cause of death in dementia or in idiocy but was a particularly common cause of death in mania (16%) and melancholia (10%). In epilepsy, exhaustion was ascribed even more frequently (18%) as the cause of death. In several cases “exhaustion from epilepsy” was stated as the cause of death. There were six epilepsy cases whose cause of death was ascribed as just epilepsy and six as “status epilepticus.” All these latter cases are put in the “Other” category in Table 1. It is likely the cases labelled as dying from epilepsy died during an uncontrollable fit and those with “status epilepticus” died after a prolonged series of fits. It is therefore unclear what “exhaustion from epilepsy” actually means. Exhaustion as a cause of death in insanity was seen generally in younger subjects (the average age at death from exhaustion for both mania and melancholics was 54 years). Death from exhaustion during mania had been observed by several authorities of the period including Burrows in 1828, Pritchard in 1835 and Bell in 1848. Smith gives details of

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213 This is even more marked when senile mania and melancholia cases are excluded (see Chapter 4).
several manic patients who were subjected to mechanical restraint and died soon afterwards in an exhausted state but none of the cases described here had been subject to restraint. However, in other respects the figures shown here are consistent with the literature of the time. In 1895, Farquharson (Assistant Medical Superintendent, Counties Asylum, Carlisle) observed that 20% of melancholics died during their first admission from general exhaustion.\textsuperscript{215} In a study by Harris and colleagues of melancholic deaths in a cohort of patients admitted to North Wales Asylum at Denbigh between 1875 and 1924, tuberculosis was the commonest cause of death, but exhaustion was the next most common.\textsuperscript{216} Exhaustion as a cause of death is discussed in much greater detail in Chapter 4.

30\% of cases in my Berkshire sample were given a cause of death as “Senile Decay” or “Cerebral Softening” or “Cerebral Atrophy” or, in a small number, usually the very elderly, “Senility.” For the purposes of the broad-brush analysis presented here these causes of death are lumped together. It is not at all clear whether these labels truly represent the proximal cause of death rather than what the clinical picture and the established practice of the day determined. This category dominated the deaths in the dementia group, accounting for 53\% of the causes of death, with senile decay by far the commonest individual cause of death listed. This type of cause of death was also not uncommon in mania or in melancholia, accounting for approximately a fifth of causes of death, with cerebral softening the cause of death most commonly ascribed. It is noteworthy that the insane dying with this label were, overall, with few exceptions, much older - their average age of death was 71 years compared to 49 years in the group without such causes of death. The striking difference between the insane groups with and without these ascriptions may well suggest that, in the uncertain world of ascribing a cause of death, the patient’s more advanced age made those carrying out this task more likely to look for these specific changes in the brain and perhaps predispose them to accept them uncritically. Andrews comments that relatively few alienists doubted that mental disease could “exist... without a morbid change in... the brain.”\textsuperscript{217} The influential alienist W. A. F. Browne, for example, asserted in 1837 that the “prevailing opinion at

Contradistinguished from any Ordinary Observed or Described Combination of Symptoms as to Render it Probable that it may be Overlooked and Hitherto Unrecorded Malady,” \textit{American Journal of Insanity} 6 (1849): 97-127.
\textsuperscript{216} Harris \textit{et al}, “The Morbidity and Mortality linked to Melancholia,” 3-14.
present is, that no cases do occur where no pathological condition can be observed” and that those recorded as showing no pathological condition “owe this feature to the negligence or ignorance of the narrator.” Barfoot, however, stresses that this view was not universally held.

Were the asylum medical staff more likely to attribute a brain condition as cause of death in the elderly insane because some apparent pathology therein was all they could find at post-mortem and its use added an apparently scientific veneer? The use of these terms, particularly that of senile decay, fitted in with the accepted narrative of the time that old age itself caused inevitable changes, a narrative that was not to shift for a further 70 years or so. These issues are useful reminders that attributing cause of death was not transhistorically neutral but was conditioned by often transient psychiatric paradigms and was, arguably, more art than science. Diagnosing the causes of death amongst the insane was highly subjective and coloured by prevalent models of the day, as well as being influenced by concerns about how others, including official lunacy watchdogs, might interpret apparent imprecision or anomalies in reports.

Vascular disease was a cause of death for about a sixth of the 537 deaths, but this proportion rose to about a quarter in the mania and melancholia groups combined. Despite the higher age at death in dementia, vascular disease was not a particularly common cause of death in this group. In the mania group, a vascular death was found in a relatively young population; the average age of death from vascular causes in the group with senile mania excluded was 57 years but was higher in the melancholia group at 62 years (even after the small number ascribed to senile melancholia were excluded from the calculation). In 1901, the Medical Superintendent of Enniscorthy District Asylum in County Wexford, Thomas Drapes provided evidence from asylum post-mortems in Ireland that deaths from heart disease were twice as common as that of the general population. However, without details of the age structure of such deaths in these populations (i.e. calculating a SMR) such comparisons are avoided in the current study. The histographical trends in the labelling of deaths as vascular will be discussed in more detail in following chapters.

Deaths by infection were common and accounted for a third of all causes of death. This figure was fairly stable across all the clinical groupings, apart from idiocy, where infections accounted for nearly half of the deaths. Historians have observed that this latter group were particularly susceptible to death by infection. Epidemics, such as those described by Wright at Earlswood Asylum for Idiots and Imbeciles in the early 1860s, had a major impact upon institutional death rates in those so diagnostically labelled.221 The circumstances in which the sufferers of “imbecility” were kept within some ordinary, overcrowded pauper and county asylums may have been even more compromised than those for other patients; these were often the first patients to be transferred to other asylums and workhouses in makeshift or more permanent solutions for overcrowding.222 The conditions of imbeciles at the Berkshire Asylum are not clear from perusal of Minute Books, accounts of the Visitors or Commissioners in Lunacy, or other available sources, but one suspects this group were a low-profile, undesired and neglected group generally. William Murdoch, the Medical Superintendent, was outspoken in his criticism of “idiots and imbeciles… being foisted on his undertaking.”223 These observations suggest that the quality of care of these benighted individuals was probably poor even by the prevailing standards of the asylum, with overcrowding and inattention to diet and sanitation, factors apt to increase the likelihood of severe and fatal infections.

There was a wide range of infections at asylums like Berkshire but there was no evidence of gastro-intestinal epidemics like those that had ravaged asylums in the earlier part of the century when, as Smith notes, asylum deaths from dysentery were frequent as were outbreaks of cholera.224 Similarly low incidence of epidemics pertained within other contemporaneous, loosely comparable international contexts. Wright comments, for example, that while the annual rates of mortality in asylums in Ontario varied, the variation did not correspond to well-documented infectious diseases outbreaks.225 Wright’s analysis shows that deaths from epidemic infectious diseases (typhoid or cholera, for example) were infrequent and about 1% of deaths in the late Victorian period. Little “waves” of gastrointestinal infections were evident in the current study but

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222 See e.g. Cherry, *Mental Health Care in Modern England*, 84-85.
disorders like dysentery, enteric fever and “colitis” accounted for just over 4% of asylum deaths, comparable to the Ontario figure.

The main infective causes of death at Berkshire were pneumonia and tuberculosis, which in their various forms accounted respectively for 13% and 15% of all deaths across the period. Pneumonia was a common cause of death in the elderly with melancholia or dementia but less common in the young with mania. Tuberculosis was a particularly common cause of death in those labelled as idiots and this may relate to the adverse conditions in the asylum for such people. Another notable group dying with tuberculosis was young patients with dementia. Dementia was a term applied, inter alia, to patients suffering from acquired brain injury and/or cognitive impairments, which begs the question of whether the fatal tuberculosis ante- or post-dated admission to the asylum. A similar set of issues applies to deaths from tuberculosis in patients with mania and melancholia. In the current study, one in seven of the mania and melancholia group combined died of phthisis and their mean age at death was 38. Tuberculosis was endemic in Victorian asylums but there was much debate about the exact frequency of it as a cause of death. In 1899, London physician, Francis Crookshank pointed out that the true, as distinguished from the "official," phthisis death-rate would be much higher as the official returns underestimated actual phthisis mortality in asylums, which he argued could only be obtained from post-mortem records, as is the case, for the most part, here. Thus, in one county asylum in which the reported proportion of deaths due to phthisis had been consistently about 11%, advanced phthisis was found in 20% when post-mortem reports were examined.\(^\text{226}\) Another issue, first highlighted in 1905 by the LCA Superintendent Jones in his report on post-mortem examinations by the asylum pathologist, was whether finding tubercular signs at post-mortem necessarily equates to tuberculosis being the cause of death. Jones’s survey revealed the presence of active tubercular pathology in 21% of the male deaths and 27% of the female deaths. However, inactive tubercular changes were found in about a third of cases in which other causes of death were apparent; in other words they were co-incidental findings.\(^\text{227}\) This poses the question of whether old infections with the tubercle bacillus can leave scars which have at least the potential to lead to the cause of death.

\(^{226}\) Francis Crookshank, “The Frequency, Causation, Prevention, and Treatment of Phthisis Pulmonalis in Asylums for the Insane; Essay for which was Awarded the Bronze Medal of the Medico-Psychological Association, 1899,” *Journal of Mental Science* 45 (1899): 657-83.

\(^{227}\) Jones, “Prognosis in Mental Diseases,” 1578-83.
death being attributed to tuberculosis inappropriately. These issues led, in part, to a degree of uncertainty of how common death from tuberculosis was in asylums in the late Victorian period. Drapes produced a report to show that the mortality rate from tuberculosis around the turn of the century in asylums was between five and a half and ten times greater than deaths from this disease amongst the general population. However, data from the 75th Annual Report of the General Register Office, summarised by Hardy, provided evidence that deaths from phthisis in asylums did not make any great addition to local death rates while Crookshank believed that asylum TB deaths were only twice those of the community. The age profile of the populations being compared may be the reason behind these disparities. Drapes showed that the death rate in young adults in the community was identical to younger adult patients in the asylums. The increase in deaths from tuberculosis in the asylum occurred in those over 40. This fits with an average age of death from phthisis of 38 in the current study as this is much older than deaths from pulmonary tuberculosis in the community which was centred between the ages of 15 and 35.

Both Drapes and Crookshank, pre-eminent authorities on this topic at the time, came firmly to the view that the excess of tubercular deaths in older persons in asylums was a consequence of them having acquired tuberculosis during their stay in the asylum and they were emphatically supported in this conclusion by commentary in the British Medical Journal. A 1902 review of the Drapes’s report conclusively blamed asylum conditions, asking “Will anybody doubt that this excess mortality is due to overcrowding?” and called for all “large asylums [to be] properly divided into separate wards and pavilions, [with] improved ventilation.” Others, however, were less convinced, suggesting that the case was not wholly proven, since the extreme overcrowding of some cities, including Glasgow, did not translate to unusually high urban tuberculosis mortality. Whether or not overcrowding was the key issue leading to tuberculosis being acquired during a stay in the asylum cannot be conclusively judged. The Berkshire Asylum staff were aware of this debate and there was an active policy to isolate those thought to be suffering from tuberculosis and to manage as many potential sufferers as possible in better aired spaces. Asylums had been

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228 See Chapter 3 for how this issue was dealt with in the current study.
229 Drapes, “Phthisis and Insanity,” 669.
230 Hardy, “‘Death Is the Cure of All Diseases’,” 481; Crookshank, “The Frequency, Causation, Prevention, and Treatment of Phthisis Pulmonalis in Asylums,” 657-83.
encouraged to have outside but covered verandas where beds or chairs could be wheeled out and with sufficient space for the patients to be nursed. This recommendation became widely practiced, including at Fair Mile, and the norm by the early twentieth century. Notwithstanding these efforts, tuberculosis remained common. This may be a consequence that some, maybe many, were carrying active tubercular bacilli and/or suffering from acute tuberculosis on admission. In addition, the condition was highly contagious, particularly amongst those debilitated by their mental condition and, in all likelihood, the straightened circumstances that pertained before admission. Some leading figures of the day came to the view that active tuberculosis could also cause, or at least be intimately linked to, the abnormal mental state that led to admission. Prominent amongst these was Clouston who denominated such cases under the diagnostic term “phthisical insanity.” In his classic paper on the topic, he described how the mental symptoms preceded the pulmonary in most cases (also observing, as found here, a predilection for secondary tuberculosis in those with dementia, those he deemed “trophically lowest”). However, he additionally observed that in about a quarter of cases the “two diseases had arisen so very nearly together,” and concluded the predisposing cause to be “a heredity to both insanity and phthisis.” The historiography surrounding the debates concerning these questions will be discussed in the next chapter, which examines the clinical and pathological details of the insane sample in finer detail.

There seem to be two populations within the dementia group, a younger group in which tuberculous infection led to death and an older group where the cause of death was most commonly a brain condition. Dementia was a widely used diagnosis in asylums in the earlier decades of the nineteenth century usually, but not invariably, to denote an insane person with acquired memory deficits. Berrios charts how the concept of dementia changed significantly over the course of the nineteenth century with the “cognitive paradigm” becoming dominant by the century’s end. The term dementia, to denote largely a predominantly elderly population with memory deficits as a core feature, became utilised more frequently across the last decade of the

nineteenth century and was standard usage by the early 1900s. The sample described here was on the cusp of these various trends. There is a need for further study of the associations of the diagnosis of dementia in the Victorian period, although it is likely to be hampered by shifting use of terms. In some asylums in the later part of the century, the term senile mania was used to designate those more elderly people with cognitive problems, but its use was patchy. Yorston and Haw describe how senile mania was a commonly used term in one Oxford asylum (the Warneford) but rare at the other (the Littlemore).\(^\text{237}\) As identified above and shown in Appendix B, there were a group of people who died with senile mania and senile melancholia in the current sample, all over 60 and most well into their seventies. One suspects, but cannot prove, that this group were more akin to elderly dementia patients (in the more modern sense) rather than the younger manics or melancholics.

About one in ten of deaths was placed in the “Other” category. This category contained small numbers of a wide range of fatal conditions. First in this category are those five in whom no cause of death was given. It is fruitless to speculate as to why this was, and no further work was done on these cases. The rate of death from cancer was low at 2% of all deaths, probably reflecting the relative youth of the asylum population. Drapes, in a systematic study in 1901 utilising post-mortem reports, indicated that deaths in the asylum from cancer were 50% less than the general population and the rate he showed in his survey is compatible with that seen in the current study.\(^\text{238}\) However, it is unwise to say more than that since to do the comparison properly requires details on the age structure of the asylum compared with that for the general population and such an endeavour is outside the scope of this work. There were a small number of deaths which could be thought of as “surgical” i.e. deaths from volvulus (twisting of the bowels) and perforations with subsequent peritonitis. It is probable that an operation would have been life-saving for such patients and likely that operation would have been an available option had they not had the misfortune to be in asylum, but a further exploration of the health inequality issues raised is beyond the scope of this thesis. A major contribution to the “Other” category was from deaths from epilepsy, which accounted for 3% of all deaths and no doubt reflects the dangerousness of fitting, particularly in an era with little in the way of effective treatments. There were four cases of idiocy deemed to have died from marasmus, a form of severe initiation brought on by starvation.

\(^{238}\) Drapes, “Phthisis and Insanity,” 667-78.
These distressing deaths were almost certainly, given the client group in question, likely to have been self-starvation linked to profound intellectual disability.

There was one death aged 33 from self-suffocation with a blanket which was therefore suicide. Thus, out of 608 Fair Mile deaths, over a ten-year period, only one death was suicide. The phenomenon is so rare in this sample for the effect on overall mortality to be negligible. This fits in well with what other commentators have written on this subject. Shepherd and Wright show that rates of asylum deaths listed as suicide between 1861-1881 varied between 0.29% and 0.45% and similar figures were found by Wright in Ontario asylums.239 Beveridge demonstrates that this low rate was despite the fact that many patients were described as suicidal in admission records.240 However, Wright points out that some Victorian commentators believed that the "true" rate of suicidality was closer to 5% of asylum admissions than the higher rate reflected in the asylum admission registers. He speculates that one reason for this discrepancy was that in the negotiations between workhouse authorities and magistrates, Poor Law officials sometimes exaggerated the danger of suicide or dangerousness in order to access beds.241 Notwithstanding this, part of this success in limiting suicides in asylums had to do with the emergence of more refined preventive measures. Earlier in the nineteenth century, restraint was commonly used, but, in the wake of the lunacy reform movement and the public’s interest in this topic, superintendents were obliged to employ other means to prevent suicides within their institutions. Strict surveillance and the frequent use of sedatives became more commonly employed. Clouston emphasised the difficulty of detecting the high-risk inmate and stressed the importance of vigilance to prevent suicidal patients from achieving their goal, an emphasis very much in evidence at the Berkshire Asylum during the period under scrutiny.242 The Fair Mile Asylum Minute Books contained numerous exhortations to staff to comply with safety procedures, including a detailed account of the procedure for the staff’s use of scissors. Success for this strategy was apparent. The carrying out of these difficult tasks fell to the attendants and created conflicts between the interests of care and cure on the one hand, and secure containment on the other. Smith describes how staff of imposing size and build were commonly recruited in asylums, while concurrently and

241 Shepherd and Wright, “Madness, Suicide and the Victorian Asylum,” 194.
242 Clouston, Clinical Lectures on Mental Disease, 81-105.
somewhat contradictorily they were enjoined to deal with their distressed charges in a kind and mild manner.²⁴³

Comparison with historians’ accounts of other asylums shows a broadly similar picture to that found here. At Colney Hatch, brain disease was the commonest cause of death, followed by tuberculosis.²⁴⁴ At the North Riding Asylum in York, general paralysis, brain disease and senile decay were the main causes of death while at Rainhill Asylum, general paralysis and tuberculosis were the most important.²⁴⁵ Malcolm finds that infection was a major killer at St. Patrick’s Hospital in Dublin, while Finnane, in his study of Irish asylums, finds that there was an increased mortality amongst the pauper as compared to the private patients, due to their poorer physical health.²⁴⁶ Mortality patterns delineated in this chapter serve to provide further substantiation but also some areas of significant variation in relation to established conclusions in the extant scholarship. It is both valuable and reassuring that this current comprehensive study, which unlike many others includes a high frequency of post-mortem findings informing the cause of death ascription, largely confirms several other smaller studies carried out on a range of asylums.

2.2.3 Methodological Issues Regarding Data on Causes of Death

A key issue in assessing the implications of these findings, is the extent to which reliable interpretation can be placed on causes of death. This question will be examined by critically assessing the contribution of other commentators to this topic in general, before turning to the particular problems encountered in ascribing causes of death in insanity.

Carter observes that physicians registering death in this era had “beliefs and goals different from those that now direct medical research” and further cautions that “superficial similarity of language” can lead to a false assumption that causes of death identified in this period “can be

readily and meaningfully compared with those we identify today.” He shows that words or phrases (e.g. “mumps” or “dropsy”) may have had very different meanings and contexts in reports from the time. Woodward observes that during this period symptoms were not seen as markers of disease but rather as the disease itself and Hardy comments that this issue applies particularly strongly to data before the production of more reliable and consistent cause of death statistics from the late 1860s. For example, it was only then, as Luckin shows, that typhoid and typhus were reliably differentiated. Hardy comments that this type of problem decreased in subsequent decades whilst acknowledging that there is always an “inbuilt margin of error”. She describes how evolving diagnostic methods and changing fashions in terminology caused shifts in certification practices which, in turn, can lead to misleading conclusions.

Classificatory systems have a long and winding history. In the mid-nineteenth century, William Farr, statistician for the GRO, developed a classification for causes of death into five groups, namely: (1) epidemic; (2) constitutional (general); (3) local (by anatomical site); (4) developmental; (5) the result of violence. To discuss this and other systems, the first International Sanitary conference took place in Paris in 1851 and this was followed by the first International Statistical conference in Brussels in 1853. Statistical and medical perspectives clashed at these and subsequent meetings, but Farr was influential and by 1864 his observation-based system was imposed. While Alter and Carmichael point out that this classification had no grounding in underlying pathology and “little concern for aetiological or diagnostic subtleties”, nonetheless they consider it “an improvement on the even more unsatisfactory, rather random, situation prior to this.” How much it was adopted in practice is unclear, although, in the last two decades of the century, this forerunner of the International Classification of Disease (ICD) was at least in some use. In the last decades of the nineteenth century, the Royal College of Physicians began a concerted attempt to standardize English medical diagnostic terminology by issuing an approved Nomenclature of Diseases. In addition to the improved standardisation that these developments brought, there were further improvements in the death registration system by the introduction of

249 Luckin, “Death and Survival in the City,” 55.
a confidential enquiry system by Dr William Ogle (a successor to Farr). Clinicians were contacted if a certificate’s wording was unclear, and this process not only clarified those certificates but acted as a training exercise. By the end of Victoria’s reign, the Registrar-General was able to announce a very substantial improvement in the quality of death certificates. Reid and colleagues point out, however, that improvements in certification did not necessarily translate into improved statistics, as another key variable was how clerks interpreted the often complicated and sometimes confused data on the certificate. Reid remarks that “Clerks were almost certainly given instructions on how to deal with tricky cases” but notes that these instructions have not survived.251

Analysing the causes of deaths in asylums is subject to all these strictures amplified by the lack of standardisation in diagnosis and uncertainty related to the heterogeneity of the asylum population.252 The causes of death ascribed in records show a lot of vagaries dependent as they were on individuals within asylums, their background, training and whims. For example, it is hard to interpret the data produced by James G. Davey (Medical Superintendent and proprietor of the Northwood Asylum near Bristol) who, in 1855, examined modes of death in Hanwell Asylum and indicated that about 40% were from “apoplexy or epilepsy,” 15% from “pulmonary consumption” and 20% from a gradual decline and exhaustion of the “vital powers.”253 In 1863, Bucknill drew attention to the many deaths that were ascribed to exhaustion in one institution, but what appeared to him to be similar cases in other institutions were ascribed as “prostration,” “general debility” or “general decay.” Bucknill emphasised that these terms had “such wide and indefinite application, as to be almost without meaning,” pleading for such deaths to be labelled “death by syncope” and to be related to the underlying illness e.g. mania or melancholia.254 However, his plea (despite the “support of the Registrar – General”) seems to have fallen on deaf ears. Several commentators highlight variable, individualistic and sometimes quixotic causes of death ascribed

in asylum records particularly in the earlier parts of the century. The extent to which the performance of post-mortems aided accuracy in this regard will be debated in the next chapter.

2.2.4 Mortality rates in the population compared to the Asylum

There was an increase in longevity in the population over the second half of the nineteenth century, but it was not linear and headline figures mask many effects and influences. Life expectancy at birth rose by 10 years across the totality of Victoria’s reign but the rise began only from the late 1860s onwards. Crude death rates remained static between 1841 and 1871 and infant mortality began to decline only at the very end of the monarch’s reign. The factors underlying the reduction in mortality have been analysed by McKeown and his associates with a focus on causes of death. McKeown considers that most of the reduction was attributable to a reduction in infections as non-infective conditions only contributed about 8% to the decline in mortality. However, as Woodward and others highlight these conclusions may be illusory because of errors in certification. McKeown expresses the view that medicine had very little part to play in the reduction in mortality. For example, he indicates that deaths from tuberculosis fell even before the identification of the tubercle bacillus in 1882 by Koch. McKeown hypothesised that the reductions in infective diseases were due to improved nutrition leading to greater resistance to infections and greater ability to fight them. Woodward, however, argues that the evidence for this assertion is lacking. He cites the lack of evidence for a major shift in food consumption across the period (despite falls in food prices) and highlights the lack of change in children’s’ weights and heights. British anthropometric data has confirmed that late eighteenth-century improvements in height attainment (generally seen as a good proxy for nutritional status) were curtailed and even reversed during the middle and latter part of the nineteenth century. The reduction in mortality was found across all social groups and it is argued that a nutrition-based effect on mortality would have shown a class effect. Moreover, as Szreter comments, during the whole of the mid-Victorian

period, when the British economy experienced sustained economic growth, mortality rates failed to improve at all.\textsuperscript{259}

There are further divergences in scholarly opinion on this issue. Clayton and Rowbotham argue that the Victorian diet was very healthy. They argue that this was related to improved output from agriculture, a better transport network, notably the railways, and a political climate that ensured cheaper food. Although the average calorie count per day was high through high carbohydrate (e.g. bread, potatoes) intake, this was matched by the high calorie output per day of the labouring class and the heavy housework of females. They point out that the working-class diet was rich in fruit and vegetables with overall low alcohol and tobacco intake. Some scholars also argue that food may have been worse in this period than before as industrialization of the food industry also brought with it food adulteration but Clayton and Rowbotham maintain that some adulterations brought health benefits (citing, as an example, the watering down of beer).\textsuperscript{260} Clayton and Rowbotham point out that death from cancer and degenerative disease was relatively low in incidence during this period and posit forcefully that this was due to the “healthy diet” of Victorian Britain.\textsuperscript{261} However, most authorities would convincingly argue these conditions were then relatively uncommon because of the relative youth of the society at that time.

Industrialization had a negative impact on population longevity - an effect which according to Szreter concentrated particularly among the families of the “disempowered, displaced migrants” who provided a large part of the workforces in the fast-growing industrial towns and cities.\textsuperscript{262} This effect may have been related to issues with the quality of the water supply and sewerage in overcrowded slums despite the importance of sanitation being well-understood following Chadwick and Simon’s work. However, Szreter argues that industrialization impacted more on health because of the intensely disruptive nature of this change and that such disruptions caused social deprivation leading to disease and death for the most unfortunate and marginalized individuals. Szreter claims that deaths from tuberculosis are a particular hallmark of these effects describing it as “a scavenger disease” causing death once an individual was in a “weakened state.” It is

\textsuperscript{259} Simon Szreter, “Industrialization and Health,” \textit{British Medical Bulletin} 69 (2004): 75-86.
\textsuperscript{261} Clayton and Rowbotham, "How the Mid-Victorians Worked, Ate and Died." 1246.
tempting, perhaps too tempting, to draw parallels between Szreter’s ideas about the links between industrialization and mortality and those between insanity and its mortality. Links are however evident. For example, migrants were known to have a high mortality rate in asylums and asylum patients can readily be categorized as disempowered.263 Marked economic deprivation was not evident in asylum patients of this era prior to their illness but becoming insane at this time led to considerable social deprivation, however it is defined.264

Despite divergences in scholarly opinion, there does seem to be consensus that changes in nutrition, alongside better housing, a reduction in the worst ravages of industrialization and the smaller families of the late Victorian period, combined to be a major influence on mortality reduction. Furthermore, developments in preventive health provision, in social services and in infrastructure (particularly the advent of domiciliary sewering and water supply) are also linked to a reduction in urban death-rates as local authorities’ expenditure increased in the later decades of the century. From the 1870s onwards, urban life expectancies began to climb above the levels of the early nineteenth century, pushing the national average up too, as Britain by this time was predominantly an urban society.265 Another factor impinging on the reduced mortality over Victoria’s reign is the reduction in what Anderson terms “crisis mortality.”266 In the eighteenth century and, to a lesser extent, in the earlier part of the nineteenth century, deaths from major epidemics were frequent and their impact on the population often substantial. The effects of war and food shortages often compounded the effects of the epidemics. However, during the period under review, only smaller more localized outbreaks of infections remained. The fall in deaths from epidemics was most noticeable in the young and was also evident in the asylums.

While life-expectancy of the population rose during this period, so too did the life expectancy of those with severe mental illness in asylums. However, the latter continued to lag behind that of the general population. Wright showed this effect in his study of asylums in Ontario and demonstrates a gap of between 10 and 15 years in survival between asylums and a broadly matched community sample, strikingly similar to that reported by Jones in his study of deaths in

London asylums. 267 Data extracted from constructed life charts has shown that, for those surviving childhood and into adolescence, someone born in the mid-Victorian period could expect to live into their late 60s on average. 268 The average age of death in the asylum insane in this study was 55, a difference which exhibits the same mortality gap of 10 - 15 years. These comparisons have, of course, to be treated with caution. The age of death in the normal population is subject to many caveats related to problems with census data and problems with civil registration of deaths as Higgs amongst others has shown. 269 The average age at death in the asylum is a potentially misleading number, made up as it is of deaths in disparate conditions – the final figure therefore depends on the makeup of the sample at that juncture and locality. A further limitation inherent in this comparison is that it is not comparing like with like, as the numbers are generated so very differently. Notwithstanding these caveats, asylums were places where those at risk of premature death were collected. It is notable that premature death was found in those suffering from insanity, that is the conditions of mania and melancholia, not just in those with the more neurological conditions like epilepsy and GPI and the more organic disorders like idiocy which not only had high death rates but also were linked strongly to premature mortality. In addition, though not contributing to the mortality gap, the increased longevity of the population was starting to have an influence on the continued high death rates in asylums. Many elderly, often frail, patients were admitted and died of problems like senile decay or cerebral atrophy or softening, sometimes, as we have seen, just labelled as “senility.” Many such patients were admitted, in Clouston’s words, “on the brink of death.” 270

Premature death in insanity contributed to the mortality gap, particularly when it is borne in mind that the average age for death was 55 years and just under a half of the group died at a younger age than this average. These observations led, in part, to the strategy adopted in the following chapter of studying those deaths at or under the age of 55. This enriched sample will be examined in detail, with a survey of both the clinical notes and the post-mortem findings, so as to fulfil one

268 Cutler, Deaton, Lleras-Muney, “The Determinants of Mortality,” 97-120.
of the main aims of this thesis, to more thoroughly investigate and determine the main factors associated with premature death in insanity in Victorian asylums.

2.3 Conclusion

This chapter has examined all causes of death across a ten-year period in an asylum with good records and a very high post-mortem rate. An almost complete post-mortem record (about 97% of all deaths had a post-mortem) was achieved. The reasons behind this high rate are not completely certain but it did not seem to be related to the trade in cadavers described by Hurren. The high post-mortem rate can be seen as a strength in terms of providing more extensive data on which to base cause of death diagnosis, reflecting widespread pressure by the LC, the MPA and other lunacy authorities on maximising post-mortem numbers and the opportunities for pathological research in asylums. The data set needs to be analysed with care, given the doubts over the reliability and meaning of causes of death discussed above. Supplementing death register data with information from post-mortem, case note and other asylum reports, which Andrews argues can all be “innately jaundiced,” also has limitations (although whether this applies to post-mortem reports has not really been studied). It is worth noting that the reason 3% of deaths at Berkshire Asylum did not have a post-mortem are unclear. This study lacks a control group, and this hampers comparison and SMRs, which would allow more valid interpretations, cannot be easily calculated. However, a potential strength of this study is that it represents one of the few times that causes of death have been examined by someone with a medical training. McCarthy argues for collaboration in asylum studies and suggests inter-disciplinary research on this topic is wanting. Luckin remarks, however, that researchers whose primary training has been medical may tend to oversimplify findings in social and economic history. These issues are aired further in Chapter 6.

Much of the optimism of the early to mid-19th century that, if only the insane could be admitted to asylums early enough, many more would be cured had significantly diminished by the end of

271 Hurren, “‘Abnormalities and Deformities’,” 65-77
the period. Asylum mortality figures were a stark testament to that. The figures however may have contributed to an awakening of social conscience and the generally more humane treatment of lunatics which developed in the later part of the nineteenth century. The now oft repeated historical narrative is of an early enthusiasm for asylumhood and the techniques of moral management giving way to the asylum as a large, custodial institution in the latter part of the century. However, as Lanzoni said, easy generalizations about the nineteenth-century asylum are hard to come by and the same applies to asylum mortality particularly in the light of the methodological issues highlighted in this chapter. Both the numerator and the denominator which went into making up the mortality rate were subject to shifts of practice, non-medical constraints and numerous confounds and influences. Although headline mortality changed little from the 1840s through to the 1910s, there were important changes which have been discerned in wider demographic and local/regional admission patterns, which partly followed on from changes in service delivery and practice, and from the impact of public health measures. Despite the increases in life-expectancy over the period which applied to both the normal population and the mentally ill, the gap in life-expectancy between these groups remained substantial and substantially the same.

The current findings have added important observations on the scholarship regarding asylum mortality. The average age of death in the asylum was in the mid-50s. It was shown that this was made up broadly of three components. Firstly, a population made up of those with disorders where insanity and/or behavioural disturbance were secondary to an underlying predominantly neurological disorder such as GPI, epilepsy or idiocy who have a young average of death in their 40s. Next, the Berkshire Asylum deaths in the late Victorian/early Edwardian era comprised a significant older group, usually called dementia but sometimes senile, who had a much more advanced age at death. The final core group making up the bulk of the remainder of the asylum’s mortalities comprised those whose primary problem was deemed to be insanity and who had an average age of death in their mid-50s. On average, this latter group’s age of death was 10-15 years earlier than the community population and a substantial number died very much earlier. This study has also identified two causes of death, exhaustion and vascular causes, which together account for over a third of deaths and show a predilection for younger patients. In both of these

274 Hodgkinson, “Provision for Pauper Lunatics,” 138-54.
sets of conditions it conceivable that the post-mortem may be helpful, perhaps pivotal, in assessing the cause of death and its associations. These are the topics for Chapters 4 and 5.

Most commentators agree that despite the improved understanding recent scholarship on this topic has attained there is an urgent need for more detailed local, national, and comparative studies to fill the continuing gaps in our knowledge of asylum mortality. McGovern studied mortality in asylums in the USA and points out that the statistics for mortality changed little from the 1830s through to the 1920s, despite many attitudinal differences, and changes in psychiatric theory and practice over the course of the century. McGovern argues that historians must explore the inner history of asylums to discover more about this mortality, one part of which is for further evidential underpinning and enquiry into individual causes of death in greater detail as follows in the next chapter.276
Chapter 3. Asylum Post-mortems in Mania and Melancholia

3.1 Introduction

The main question asked in this chapter is what the functions of post-mortems in asylums in the later parts of the nineteenth century were. I also enquire whether the numerous post-mortems performed in Victorian asylums contributed significantly to understanding of causes of death in the insane and to the elucidation of the causes of insanity. In addition, to what extent did post-mortems function primarily as a complicated and controversial bureaucratic exercise to satisfy watchdogs like the LC, and to allay the anxieties of the wider public about the safety and utility of asylum practice? Little has been written on the topic of post-mortems and pathological anatomical research in asylums in Britain or Europe before the last two decades. More recently, the scholarship of Andrews, Cullen, Hurren and Wallis has provided substantial case studies to reduce the gaps in our knowledge of modern British developments in this area, though Hurren’s work has thus far focused primarily on death and post-mortems outside asylum contexts.277 Topp and Engstrom have also furnished instructive papers delineating key features of the emergence of asylum post-mortems, mortuaries and pathological laboratory research in the nineteenth and twentieth centuries but the great majority of their work focusses on Vienna and Imperial Germany.278 Wallis argues that one reason the topic of the clinical and pathological investigation of the body has been a “relatively neglected” aspect of asylum history links to Cooter’s notion that historians have assumed that bodies “were imposed upon by rational minds—manipulated to political ends” rather than containing clues to their underlying disease.279 This chapter focusses on post-mortem findings from patients with mania and melancholia who died at an age younger than the average for those conditions.


A range of scholarship has explored the history of pathological and neurological research, in which post-mortems has been shown to have figured significantly from a wide variety of clinical and hospital settings. Physicians began to perform more post-mortem examinations from the mid-nineteenth century onwards. Risse emphasises the influence of the Paris Medical School, where post-mortem work, its “crowning diadem” according to Foucault, led to new connections between disease states and structural changes in promoting this trend.\textsuperscript{280} Gal plots the early developments of British pathology and the growth in both post-mortem numbers and facilities across the century along with the production of increasing numbers of books, lectures, and essays on the topic.\textsuperscript{281} Jacyna and Schlich argue that pathological anatomy became the cornerstone of medical science in the late nineteenth century, particularly when it was allied with the growing use of microscopy and histology.\textsuperscript{282} William Osler, a Canadian pathologist, was particularly influential on both sides of the Atlantic in this context. His credo that: “To investigate the causes of death, to examine carefully the condition of organs... and to apply such knowledge to the prevention and treatment of disease, is one of the highest objects of the physician” became an aspiration for many doctors and medical institutions.\textsuperscript{283} By the latter part of the century, pathological research was routinely performed by clinicians more widely than ever before and pathology made up a large part of the medical undergraduate curriculum and of day-to-day hospital practice, although Jacyna shows that these developments were met with cynicism and downright hostility from some quarters. Rudolf Virchow (1821–1902) practiced in Berlin and was one of the doyens of pathological anatomy in the late nineteenth century. Sturdy observes that Virchow’s refocusing of pathology from gross anatomical lesions to cellular processes introduced a further dimension into pathology in the late nineteenth century but suggests that much clinical activity of that era continued to centre on the identification of particular diseases and morbid pathology.\textsuperscript{284} Routine autopsies in hospitals contributed to the localization of disease, an increasing definition of aetiologies and a move away from symptom-led descriptions. These developments were part of a process whereby vague and

nonspecific causes of death (such as “exhaustion”, “senility” and “old age”) were replaced with terms at least more precisely descriptive, if not always accurate. Mooney and colleagues comment that death certification was not a high priority amongst doctors of the period, as it was believed to be of little clinical value. He speculates that all post-mortems contributed to was “diagnostic reductionism” and therefore only the reporting of fewer multiple causes on the certificate.\textsuperscript{285} However, Reid and colleagues show that post-mortems aided more specific causes of death particularly in old age but conclude that we should remain wary of overenthusiastic interpretation of theories based on causes of death in this period.\textsuperscript{286}

Both Edinburgh and Glasgow, where many of the alienists featured in this study trained, were leading exemplars of the burgeoning of pathological science and teaching in this era. Indeed, there is evidence that the quality of post-mortem reports studied here is likely to have been bolstered by the ward and clinic-based nurturing of patho-anatomical benchwork at these top medical centres. A good exemplar is offered by the career of John Bennett (1812-1875), who Jacyna highlights as a leading proponent of pathological science. In 1848, he was appointed Professor of Medicine in Edinburgh. Although Edinburgh University did not appoint their first full-time Professor of Pathology until 1912, Bennett and his successors, notably William Smith Greenfield — an accomplished pathological anatomist who was part-time Professor of Pathology from 1881 to 1912 — did much to establish Edinburgh as a centre of excellence for pathological research and practice. Students were taught via observation of patients on the wards of the teaching hospitals and on the post-mortems conducted on those same patients after they died, thereby learning the habit of seeing diagnosis in terms of pathological lesions and processes. Such methods dominated clinical teaching in late nineteenth-century Edinburgh (and other European medical schools of that time). Greenfield’s course on clinical diagnoses and related autopsies was a major feature of the Edinburgh clinical course. Lorrain Smith, Greenfield’s successor, distinguished the main uniqueness in Greenfield’s teaching as “the mental attitude he makes one assume of scepticism.”\textsuperscript{287}

Universities were not the only place where students and doctors and indeed the public were exposed to pathological specimens. Alberti traces how many institutions over the course of the


\textsuperscript{286} Reid, Garrett, Dibben and Williamson, “‘A Confession of Ignorance’,” 320-44.

\textsuperscript{287} Sturdy, “Scientific Method for Medical Practitioners,” 787.
nineteenth century brought objects and bodies—dead and alive—into increasingly public spaces for display.\textsuperscript{288} He documents over a hundred pathology museums for medical teaching purposes across Britain in the nineteenth century, originally in hospitals but more latterly in Royal Colleges. Dingwall traces the history of the Pathological Museum of the Royal College of Surgeons of Edinburgh, which languished during the mid-nineteenth century but was revived as a site of teaching and research from the 1880s.\textsuperscript{289} At the same time, not to be outdone, developments in pathology were under way in Glasgow. Jacyna concludes that “pathology probably occupied a more prominent position in Glasgow than any other British city.”\textsuperscript{290} The Western Infirmary in Glasgow was unique in Britain in hosting a Pathological Institute where academic and clinical pathology teaching were combined and which, from 1893, was the base of the Chair of Pathology. However, Jacyna also documents resistance from several surgeons to these developments despite the scientific training that many of them had received from Lister.

Andrews outlines several key purposes behind the practice of post-mortems in asylums. Firstly, the examination was to appease public and political anxiety and the demands of overarching official oversight; secondly, to protect the asylum against accusations of malpractice; and finally, to clarify “doubts over causes of death (and) the wider pursuit of psychiatric knowledge.”\textsuperscript{291} The latter purpose included the LC’s ambitions that the performance of post mortems would provide data for the dissemination of knowledge and incentivise and contribute to training junior staff in pathological bench science thereby aiding their career progression. The success or otherwise of this strategy in attaining these ideals is discussed throughout this thesis. After the 1836 Births and Deaths Registration Act, it became a legal requirement to report all deaths. Subsequently, the 1862 Lunatics Amendment Act required all asylum deaths to be reported to both the local Coroner and the Commissioners.\textsuperscript{292} In response to this pressure, it became common that post-mortems were carried out on the majority of patients dying in asylums from the 1860s onwards. In 1870, the Commissioners urged that “all patients dying in asylums should have post–mortem examinations,” not only to make the asylum more like “a hospital” and to definitively establish

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\item \textsuperscript{288} Alberti, Samuel, \textit{Morbid Curiosities: Medical Museums in Nineteenth-century Britain} (Oxford; Oxford University Press, 2011).
\item \textsuperscript{290} Stephen Jacyna, “The Laboratory and the Clinic: The Impact of Pathology on Surgical Diagnosis in the Glasgow Western Infirmary, 1875-1910,” \textit{Bulletin of the History of Medicine} 62 (1988): 404.
\item \textsuperscript{291} Andrews, “Death and the Dead-house,” 9.
\item \textsuperscript{292} Jennifer Wallis, “The Bones of the Insane,” \textit{History of Psychiatry} 24, no. 2 (2013): 196-211.
\end{itemize}
causes of death, but also to advance knowledge of the pathology and treatment of various forms of insanity. Hurren shows how this strong recommendation also led to increased numbers of the insane cadavers being dissected in medical schools, where, particularly in centres like Oxford and Cambridge, in addition to them being used for anatomy teaching, there was a considerable research effort to show that insanity was not solely a disease of the mind but also one associated with bodily lesions, possibly evolutionary in origin. Professional campaigning was mounting for post-mortems to become a universal automatic process in medical institutions and a number of prominent alienists and the Journal of Mental Science supported this, but this plea was rejected in 1877. The numbers of deaths and post-mortems was an item of statutory inquiry which the Commissioners in Lunacy published in their annual report. Anything less than an 80% autopsy rate was subject to critical comment. As Wickham, Physician Superintendent of the Newcastle Borough Asylum, rather more polemically alleged, failure to meet this officially mandated post-mortem target placed asylum practitioners in “constant hot water,” or on what he termed the “Black list,” amounting to them being publicly “gibbeted.” Adam (an alienist from Malling, Kent) gave seven good reasons for carrying out a post-mortem in asylums but many alienists were sceptical about their value. Bucknill, for example, was clear that the carrying out of a post–mortem did not improve accuracy on cause of death. Wickham considered their scientific value was “not worth the candle” and that the reason superintendents made themselves “liable to worry, odium, and censure, by making post mortem examinations” was because the LC commended the practice “so strongly as virtually to amount to an order.” Despite these protestations, the Commission persisted further proclaiming that post-mortems should be performed routinely unless specifically objected to by the relatives. Notwithstanding dissenting viewpoints, by the late nineteenth century most alienists at least tacitly supported post-mortems and rates rose reaching about 50-60% between 1870 and 1886 to between 70 and 80% between 1886 and 1896. Rates however varied. They were much lower in parochial asylums and those with a high number of pauper wards. The Commissioners in Lunacy praised “a satisfactory proportion” of 84% in 1906. The high rate of post-mortems continued until the outbreak of World War I “when standards were

293 Hunter and McAlpine, Psychiatry for the Poor, 243-6.
298 Wickham, “Post Mortem Examinations,” 381.
lowered all round.” In 1915, the Commissioners of the then Board of Control rescinded rules concerning entries in case notes and performance of autopsies “in the circumstances of the national crisis” but continued to “rely upon such being made, when... deemed essential.”

Asylum post-mortems were not infrequently the basis of a struggle between coroners, wishing to determine the cause of death, families wanting appropriate after-care and those medical superintendents who wished to advance the science of the pathology of insanity. The 1832 Anatomy Act laid down that those dying in institutions, including asylums, could be sold to anatomy schools for dissection by those authorized to do so. Hurren summarises the evidence that much of this “trade” was unrecorded and that as many as 30% of pauper lunatic corpses were sold on for dissection. There were occasional battles between anatomists and pathologists competing to gain ownership of the asylum dead, although Hurren also documents examples of collaboration between these agencies. Strange shows that many relatives claimed bodies from institutions so as to avoid dissection, carry out mourning rituals and arrange decent burials. There is no mention in the records I have examined of bodies being sent from the Berkshire or Montrose Asylums to nearby medical schools. Hurren notes that the closest medical school to the Berkshire Asylum, Oxford, had a deal for the cadavers of the insane from a Leicester Infirmary. Andrews stresses that the growth of pathological research on the dead insane turned patients into “objectified institutional specimens” to some extent and explains how this was compounded by an absence of clarity (and consequent local and national variations) over consent. While a post-mortem to elucidate the cause of death may have had the relatives’, sometimes grudging, consent, there was unlikely to be consent, or even passive assent, for research and the removal of tissue for specimens but this is what happened not infrequently. Hurren highlights how the insane body was seen as a source of fear and dread but also fascination and how findings from thousands of insane cadavers were documented in an “Abnormalities and Deformities” register.

Scholars, including Andrews, but also Scull and Philo, have documented the development of asylum mortuaries across the land, as prompted by the LC and the provision of better facilities for

300 Hunter and McAlpine, Psychiatry for the Poor, 243-6.
301 Hurren, “Abnormalities and Deformities,” 65-77.
staff and bereaved relatives. Many asylums followed the blueprint for ideal mortuary facilities published by Burdett in 1891. Mortuary laboratories and the appointment of pathologists became more common. There was, in spatial, resource and personnel terms, in Andrews’s words, an “increasing primacy of anatomico-pathological research.” Wallis comments that as asylums filled with large number of patients, often for extended periods of time, many seriously physically as well as mentally ill and frequently incurable, many alienists were “compelled to look beyond the psychological in their day-to-day work” and towards a search for a somatic seat of mental disease. Scull, however, takes a decidedly more cynical view, speculating that performing post-mortems was a way “to minimize the amount of time” medical men “were forced to spend in the unpleasant and disturbing company of patients.” However, Scull’s comments underplay the sincerity of the scientific ambitions behind such endeavours. Research-minded clinicians, recognising certain limits to moral management and psychologically-based mental medicine, clearly sought to bolster the knowledge base and credibility of mental science by a concerted focus on insanity’s underlying organic pathologies. Typifying this perspective, Crichton-Browne, from the West Riding, took the view, which was echoed by many scientifically orientated colleagues, that an increasing focus on moral management had “hustled science into a subordinate place.” He stressed the benefit of combining scientific and the psychological approaches observing that “It is when [the two] converge and rush together that a spark of genuine illumination is certain.” Wallis describes how Crichton-Browne was instrumental in developing research at the West Riding Asylum which had one of the first asylum laboratories and was the first to appoint a pathologist onto the staff, as early as 1872. Notably, the West Riding was the base of Sir David Ferrier who did seminal work on cerebral localization. Charles Darwin visited

305 Henry Burdett, Hospitals and Asylums of the World, 4 vols (London: Churchill, 1891). Burdett’s recommendation was that “the mortuary should consist of two rooms — one a general dead-house where bodies are received, washed and prepared for burial, the other a smaller room in which one body may be placed for the friends of the deceased to see. This latter room may be made to take the form of a mortuary chapel, and should at any rate be finished inside so as to suggest a decent and reverent care for the dead.” Burdett, Hospitals and Asylums of the World, vol 4, 81. In both asylums studied here, these recommendations were followed closely.
308 Wallis, Investigating the Body, 225-6.
309 Andrew Scull, The Most Solitary of Afflictions, 263.
310 Wallis, Investigating the Body, 10.
the laboratory during some of his research. Despite its exemplary reputation, the West Riding set-up was something of an anomaly within British asylums, at least until the turn of the century.\textsuperscript{311}

The idea that mental disease could be explained by structural anomalies within the body gained ground in the latter part of the nineteenth century, evidenced in the increasing interest of asylum doctors in pathological and physiological research. An underlying reason for this, as Shortt comments, was that asylum superintendents were engaged in a desperate search for scientific legitimacy.\textsuperscript{312} To those who believed that “insanity had no pathology”, the counter-argument was that this was merely a consequence of “imperfect means of investigation.”\textsuperscript{313} Prospects seemed good in the latter decades of the nineteenth century for understanding the dramatic behaviour and language of insanity. This optimism was buoyed by discoveries, principally in Germany, by Greisenger and Alzheimer in particular, of specific pathological changes in some psychiatric disorders and by the elucidation of the pathological basis of general paralysis of the insane (GPI).\textsuperscript{314} The findings in GPI may have acted as a stimulant for the acceptance of the German neuropsychiatric tradition in Britain. However, by the late Victorian era it was apparent that, despite eager searching to support biomedical theories of mental disease, there was no obvious focal lesion in the brain in insanity. The successes in the understanding of disease in general medicine and in surgery which were dependent on good clinical observation combined with scrutiny in the autopsy room, were not replicated in the field of mental illness where insanity came to be explicated primarily through functional physiological (rather than structural anatomical) language.\textsuperscript{315} Shorter and Jacyna trace alienists taking a path during the course of the nineteenth century, moving from the prospect of determining biological causes of mental illness to the pessimism of the theory of degeneracy. Shortt describes how even “innovative neuropsychiatric theorists” of the day such as Hughlings Jackson and Adolf Meyer, incorporated elements of degeneration theory into their biological postulates. Thus, post-mortem investigation of the brain came also be to be a way of searching for “morphological deviation” to verify the

\textsuperscript{311} Ibid.
\textsuperscript{312} Shortt, \textit{Victorian Lunacy}, 140.
\textsuperscript{313} Duncan Greenlees, “Observations with the Sphygmograph on Asylum Patients,” \textit{Journal of Mental Science} 32 (1887): 472.
\textsuperscript{315} Wallis, \textit{Investigating the Body}, 206.
stigmata of degeneracy.\textsuperscript{316} While the medical emphasis on the central somatic origins of insanity was increasingly accepted or at least became the norm, limited and or vague, non-specific signs of disease in the brain at post-mortem made it difficult to explain causes of death. This posed problems for an asylum movement which was partly predicated on the basis that insanity was a clearly diagnosable and treatable disease.

While the stated aims and objectives of asylum post-mortems are generally understood and understandable, the outcomes of all this endeavour are less clear-cut. Andrews calculates that, in England and Wales alone, over 6500 asylum patients had post-mortems in 1901 and observes the lack of consistent findings on insanity for all this effort (with the exception of certain specific conditions, most notably GPI).\textsuperscript{317} Hurren similarly remarks that post-mortem pathology was rudimentary and “seldom established new medical breakthroughs,” while Smith stresses that these “inconclusive” findings of post-mortems in locating disease meant that “metaphysical speculations” were not dismissed.\textsuperscript{318} Cullen comments that post-mortem reports were often sparsely filled out with minimal detail about each organ. However, this was not the case in the asylums studied here – the reports are detailed with apparently correct use of anatomical and pathological terms. Post-mortem reports at Littlemore Asylum used informal, nonmedical language such as body parts being described as the “size of an orange” or nodules the “size of a millet seed” or a liver having a “nutmeg appearance.” Cullen concludes that the use of such subjective “emotive” language shows the post-mortem was for the medical officer’s own interest rather than for “the benefit of a medical audience.” In my view, this is an erroneous conclusion; the use of commonplace objects as descriptors in post-mortem reports is widespread in pathology practice to this day.\textsuperscript{319} However, Cullen makes the important point that there was no dissemination or collation of findings or any contribution to wider debates and agrees with Mackinnon’s conclusion that “the valuable material of the post-mortems and entries in the case books continues to be wasted, and no one except the medical officer of the asylum profits by the

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\textsuperscript{317} Andrews, “Death and the Dead-house,” 18.
\textsuperscript{318} Ibid, 9; Elizabeth Hurren, “Abnormalities and Deformities,” 65-77; MacKinnon, \textit{Bodies of Evidence}, 99; Smith “‘Visitation by God’,” 113.
\textsuperscript{319} Personal communication from Professor A K Foulis, Professor of Pathology, University of Glasgow.
\end{flushleft}
large and varied experiences,” observations which, in the main, chime in with the findings of this study.  

The supposition that the results of a post-mortem have inherent validity has been questioned by historians. Burney is sceptical about the validity of Victorian asylum post-mortems and makes the point that the doctor who treated the patient at the end of their life would approach the post-mortem with a cause of death already in mind. It was not until later that a few asylums employed pathologists who, Burney argues, would examine the body objectively.  

MacKinnon describes a proposal in Victoria, Australia whereby post-mortems in asylums would be done by an external doctor but this was not followed through and post-mortems continued to be performed by resident medical officers. In her study of Littlemore Asylum in Oxford, Cullen shows that there is no evidence that the performance of a post-mortem led to an amended register or notice at death. Thomas Clouston reproached asylums and alienists for statistics based on “ante-mortem supposition” but there is no real evidence that the harder scientific nature of post-mortem observations materially changed statistics or the subsequent individual or group of patients’ outcome and none was forthcoming in the records I have perused in these two asylums. Andrews shows that post-mortem records acted as a teaching resource at Gartnavel Royal Asylum in Glasgow but I could find no evidence of the pathology records being used for this purpose in Oxfordshire or Angus.  

The widely held contemporary view that better regulation and monitoring were the ways to counteract high asylum mortality is described by several commentators. So, notwithstanding the LC’s rather lofty reasons for insisting on the practice of post-mortems in asylums outlined above, there were underlying, largely unstated, aims of using post-mortem to identify and prevent asylum malpractice, appease public and political anxiety and instil overarching official oversight. Another objective was to protect the asylum against accusations of malpractice. The Lunacy Acts Amendment Act of 1885 sought in part to protect the asylum from charges of negligence by

320 MacKinnon, Bodies of Evidence, 85.
321 Burney, Bodies of Evidence.
322 MacKinnon, Bodies of Evidence, 85.
324 Clouston, “Tuberculosis and Insanity,” 38.
encouraging the practice of keeping detailed pathological records in asylums. The monitoring of attendants' behaviour also became an increasing focus during the period. Some were dismissed for neglect and mistreatment, and this led to the public's brutish portrait of the asylum attendant. However despite these stratagems, the high death numbers, often of young people, was unremitting. As outlined at the start of this chapter, the practice of performing post-mortem in British asylum settings has been discussed extensively in recent historiography but empirical examination of post-mortem findings and their impacts has been limited and attention will now turn to that analysis. In what follows, I employ detailed pathological, clinical, and administrative records from two asylums to examine to what extent the often-conflicting aims of post-mortems in asylums were fulfilled.

### 3.1.1 Deaths and post-mortems in Montrose Royal and Berkshire Asylums

As was shown in Chapter 2, in the late Victorian era about 10% of asylum patients died each year. The rate was slightly higher in the Berkshire Asylum on occasions, in part due to surges in deaths from gastrointestinal infections like typhoid (enteric fever) and dysentery. By this time, it was understood that such infections were due to problems with water and sanitation. The Physician Superintendent’s and Visitors’ reports have frequent entries going back decades describing the multiple problems with water and sanitation and the increasing frustration that attempts to solve the issues were unsuccessful. The proximity of the adjacent farmland was seen as part of the problem and Wheeler, the asylum’s historian, comments that bans on the use of sewage as fertiliser were frequently adopted and that, less commonly, the growing of vegetables was banned. The toilets were inadequately drained leading to sewer gas ingress. Mains sewerage did not reach the asylum until the 1950s, at which point the entire system was deemed to be prehistoric.

Despite about one in ten of the inmates dying each year, there is very little in the extensive records of both asylums about the processes and procedures relating to death and its aftermath. For example, it is not clear where bodies were taken after death. This was likely to be the

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327 Cullen, “Post-mortem in the Victorian Asylum,” 280-96.
329 A similar point is made in Cullen, “Post-mortem in the Victorian Asylum,” 287.
330 Ian Wheeler, Fair Mile Hospital, 39-102.
mortuary for those having a post-mortem but where the remainder lay after death is not clear. The dead-house in Berkshire Asylum from at least 1870 can be seen on the 1870 asylum plan (Fig 3.1). In 1904, a new purpose-built dead-house was opened on the same site beside the boiler house and, according to Wheeler, there was no “interruption in service.” This mortuary building is shown in Figure 3.2.

331 Ian Wheeler, personal communication.
Figure 3-1 Above: The 1870 plan of Berkshire Asylum (Berkshire Records Office, C/Cl/G1/25/2). The dead-house is the very top right building. Below: Enlarged version of top right corner of plan showing the detail of the dead-house. (Photograph courtesy of Ian Wheeler).
Figure 3-2 The renovated mortuary at Berkshire Asylum (now a private dwelling) photographed by author in 2017.

There were two parts to the post-1904 dead-house. The body lay before and probably after the post-mortem under the right-hand set of eaves. Post-mortems were conducted in the left-hand part of the building. The shelves used to store bodies and the block on which the corpse’s neck was placed are shown in Fig 3.3. These pictures were taken at the time the asylum closed in 2003 but local historians say these are the ones used at the time of the post-mortems studied here.

Figure 3-3 Shelves and neck block at the Berkshire Asylum mortuary photographed in 2003 by Bill Nicholls (under licence, Spackman collection).

The mortuary at the Montrose Royal Asylum was at the back of the main hospital near the wing that was used to house those with physical frailty. As with the dead-house at Fair Mile and many asylum mortuaries across the land, the facility was at the edge of the hospital perimeter to
provide privacy and protection of patients’ sensibilities while not attracting attention.\textsuperscript{332} Burdett’s blueprint for asylum architecture emphasized this necessity: “For obvious reasons the mortuary and the post-mortem room should be quite outside the ordinary paths of intercourse and near the outer wall, so that corpses may be removed through a back door by hearses,... whose work should be completed without alarming the inmates.”\textsuperscript{333}

The Montrose building was in use until the 1980s. The building, photographed in 2017 (Fig. 3.4), is very similar to the building as it was in the late Victorian period. At the back, there were ramps leading up to the first floor where staff would bring bodies to be dissected. The body was subsequently transferred to the ground floor, which had a public facing entrance, and it is here where the body lay, potentially to be viewed by the family before leaving for burial. Andrews describes how, in the late Victorian period, anterooms of mortuaries in asylums were made much more friendly places for families to view bodies with the provision of chairs and furnishings.\textsuperscript{334} Whether the mortuaries in the two studied asylums had such facilities is not known, but Berkshire Asylum had a tiny chapel of rest, probably contained inside the mortuary in the right-hand part of the building, rather than a separate, standalone facility, as at some more generously resourced institutions.

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\textsuperscript{333} Burdett, Hospitals and Asylums of the World, vol 2, 148. Burdett also recommended this separation as a way of keeping the wards “safe”. Ibid, vol 4, 65.
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The resident staff doctors performed the post-mortems. Mortuary attendants probably assisted them but no detail of them has been uncovered. The Montrose annual reports record that on the staff there was an “orraman” or odd job man and perhaps mortuary work fell into this category. The performing doctor signed the post-mortem reports in both institutions. Cullen asserts that the fact that the examinations were performed by resident medical men raises issues of definition and reliability of the post-mortem.\textsuperscript{335} However, as Sturdy argues, pathology was a large and fundamental part of doctors’ training in this era. This became even more the case as physiology in Britain had become a separate scientific discipline, taught by full-time scientists rather than clinicians and characterized by its own culture and goals. The skills of pathology and pathological techniques became an increasing part of what doctors did; some argue that pathology expertise

\textsuperscript{335} Cullen, “Post-mortem in the Victorian Asylum,” 280-96.
was central and was what made doctors special.³³⁶ Medical students of the day would have seen a very large number of post-mortems performed and discussed and would often take part and handle specimens.³³⁷

At Montrose Royal Asylum, a manual had been prepared in the 1880s by Dr Howden, the Physician Superintendent, to aid post-mortem performance although unfortunately no detail of what it consisted of survives. We know the names of the medical officers who performed the post-mortems at Montrose but little of their background or training. More is known about those who carried them out in Oxfordshire. In many asylums it was the most recently appointed medical officer who performed the post-mortems but, at Berkshire, a large number of the post-mortems were performed by the First Assistant Medical Officer, Dr Edwin L. Dunn, who attended from 1895 to 1905, returning as Physician Superintendent on the death of Murdoch in 1918.³³⁸ According to Wheeler, he was highly thought of and was “highly intelligent, well informed and keen on research.”³³⁹ Drs Thomas Johnstone and A. S. McSorley (2nd Assistant Medical Officers) carried out a few post-mortems but were replaced during 1901-04 by Dr A. P. L. Brown (or Dr Lennon-Brown, as he styled himself on his arrival) who carried out many more.³⁴⁰

Limited detail of what happened after the post-mortems is known. At Berkshire, the body probably lay in the chapel of rest within the mortuary. The body could then either be removed by the family for external burial or buried in a pauper grave in nearby Cholsey Parish Church. While it has been impossible to ascertain what proportion of patients’ bodies were reclaimed by the family, it is evident that many hundreds of bodies were buried at Cholsey having been taken there by cart. Funerals at Cholsey were carried out by the asylum chaplain, (during 1896-1910), the Reverend F. T. S. Dyer, most weekdays at 3.30 pm (a practice that had also occurred with his predecessor, Rev. A. E. Farrar). According to the Chaplain’s reports, if Rev. Dyer was unavailable then the funeral was taken by a Rev. H. Field (not Maynard the local vicar). This arrangement was

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³³⁸ Buklijas, “The Laboratory and the Asylum,” 311-25.
³³⁹ Cullen, “Post-mortem in the Victorian Asylum,” 281.
³⁴⁰ Wheeler, Fair Mile Hospital, 21.
a consequence of a heated dispute over land and money between the church and the asylum, which was eventually resolved in 1912.

Anonymous interment for the poor was commonplace in this era and this was the practice for the Fair Mile deceased. At Cholsey Parish church, the pauper lunatics were buried in plots, which were reused “after a decent interval.” No headstones graced the graves but simple iron crosses and a number. Over time, the markers were removed (no doubt, as Wheeler remarks, because they were an “impediment to mowing.”) Hurren has stressed the multiple burial of paupers after post-mortem, sometimes with missing parts, but asylum-based archives reveal no details of such practice. In 1917 Murdoch, the Physician Superintendent from 1892 died (of acute appendicitis) leaving explicit instructions to be buried near the pauper graves, in a sense still watching over his charges. The area of the unmarked graves and Murdoch’s adjacent elaborate tombstone can be seen in Fig.3.5.

Figure 3-5 The grave of William Murdoch, amidst unmarked pauper graves, photographed by author in 2017.

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341 Sylvia Barnard, To Prove I’m not Forgot: Living and Dying in a Victorian City (Manchester; Manchester University Press, 1990).
342 Wheeler, Fair Mile Hospital, 159.
343 Hurren, “‘Abnormalities and Deformities’,” 65-77.
When Berkshire Asylum closed in 2003, it was decided to commemorate the unmarked pauper graves. A plaque was erected on the wall visible in Fig. 3.5 and is shown in Fig. 3.6.

![Plaque on wall](image)

Figure 3-6 Plaque on wall bordering pauper graves at Cholsey Parish Church, photographed by author in 2017.

At Montrose, some deceased patients who had family were taken by them for burial. From 1895, those whose family did not take an interest, or could not afford to do so, were buried in unmarked graves in Sleepyhilllock Cemetery (situated on the north side of Montrose Basin) in a designated area for these patients. Where pauper patients were buried prior to 1895 is not known but may have been Rosehill Cemetery in Montrose.  

There was a hospital chaplain at Sunnyside but he was paid £70 per annum and was therefore likely to be part-time. The yearly Chaplain’s report documented the regular Sunday services (and their large attendance and the positive feedback), the bible classes on Wednesdays and the special services for feast days, but without any mention of involvement with the dying or dead, so the assumption is that the asylum paupers were buried by local ministers.

3.1.1.1 Methodology for data gathering

Two periods in the history of the Montrose Royal Asylum and the Berkshire Asylum (henceforth respectively referred to as Sunnyside and Fair Mile, as that is how they were known locally) were

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344 Personal communication from Linda Fraser, former custodian of Montrose Museum.
345 One minister, Rev. Thomas, left the post because of the “pressure on his Sundays” adding strength to the notion that the job was part-time and combined with Parish duties elsewhere.
selected based on the availability of comprehensive post-mortem records and death registers. Post-mortem records were scrutinised and cases meeting the following criteria selected: 1. Mania or melancholia (or one of their variants as described in Chapter 2. in the “Mental state on admission” entry) and 2. Death at or under the age of 55. This cut-off was selected on the basis that this was the average age of death of the whole group of deaths with these labels (data in Chapter 2) so the younger half (approximately) of this cohort would be studied. Other historians have also used this age cut-off to delineate the elderly. At Sunnyside, the ten-year period from 1892-1901 captured sufficient data (n=72) with only a short three-month gap in the records. In Fair Mile’s case, data collection was extended for two months further than ten years as that allowed the collection of 100 of the target cases. The period selected was from 8 November 1895 (the start of a new and slightly enhanced pathological record book) to 8 January 1906 (122 months).

Despite the two asylums being in different countries with differing regulations and traditions, the proformas for post-mortem reports were all but identical. An example of the Fair Mile proforma is to be found in Appendix A. The proforma was one A4 sheet, in a book of consecutive post-mortems. The information contained in the Fair Mile post-mortem reports is laid out schematically in Figure 3.7.

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346 Reid et al, “‘A Confession of Ignorance’,” 320-44.
The vast majority of the post-mortem proforma entries were complete except for the “Microscopic appearances, and any Special Notes” section which was very rarely filled in. At Fair Mile there appears to have been no suitable instrument for microscopy. An entry in the General Statement book in 1903 bemoans the fact: “The want of a good microscope has been felt much lately.” A suitable one was available for £22 but it is unclear if it was purchased, perhaps revealing the relatively poor resourcing of this particular rural asylum, certainly so when contrasted with some asylums like those at Claybury, the West Riding and Edinburgh. In any event, in Berkshire, a microscope would have been more employed for the growing amount of bacteriological work undertaken there. The mental state at death section was recorded. In most cases it was exactly the same as that of admission, in other cases, phrases such as “chronic mania” or “secondary
dementia” were seen. No analysis of this latter information has been performed. Organ weights were also taken but due to the need for a control group, this information has not been analysed further.

The reports were very complete for the most part. In just 6 (3.5%) of the 172 cases, there was evidence of rush or external pressure. The head seemed to be most often the region to be left out under these circumstances. The report on Case 29 from Sunnyside, a 51-year-old, under the “Head section”, stated “Minimal examination – body needed for burial” and, similarly, in a 39-year-old man (Case 66, from Sunnyside) “Friends waiting to take him.” In case No 62 from Sunnyside the brain was not examined as the post-mortem “was conducted in haste.” Case 38 from the same source did not examine the thorax but no explanation was given. Cases of less than complete procedures were even less frequent at Fair Mile but again it was the “Head” examination which was omitted; in one case (No 20.) the report simply stated, “Not examined” but in another of a 44-year-old domestic servant with melancholia (No 93.) the report stated: “The head was not examined at the request of friends.” The omission of the head (and therefore the brain) examination has been mentioned in other accounts which concur that this was the most often omitted part of a post-mortem. Cullen highlights that “the laying out and funeral of the body in its entirety (my italics) was considered necessary in order for the deceased to secure a place in the afterlife.” Andrews indicates that this concern might be particularly evident in families where religious beliefs emphasised resurrection. Wallis observes that across many cultures the feeling that the brain is intimately linked with the self is commonplace.

These observations bring the issue of consent into focus. There was no mention or evidence of consent forms in the records of either asylum. In the Sunnyside Physician Superintendent’s report of 1901, it was stated that “a post mortem was carried out in all cases where the consent of the friends could be obtained” and similar ideas were expressed in 1890s reports. It appears, however, that in practice the consent of relatives for a post-mortem to be carried out was assumed and tacit. Andrews illuminates the wide variation in and heated debates about the practice of obtaining consent for post-mortems across Victorian asylums. Cullen observes that the procedure in several asylums was that demurring relatives had to formally opt out in advance: “the remains

349 Wallis, Investigating the Body, 64.
of a patient are examined, unless previous written notice to the contrary shall have been sent to the Superintendent” and that, at Littlemore Asylum, the LC backed this practice in a contested case.350 There were several examples of tacit consent documented in the Fair Mile Minute Books. For example, in 1900 it was stated that “forty-five post-mortems were performed and in forty of these, consent of the relatives was obtained.” Andrews describes how in some asylums, relatives, particularly those of paupers, were denigrated as being likely to refuse consent based on irrational prejudices and their views therefore dismissed.351 Fennell and Wallis describe the difficulties in obtaining information on how frequently relatives or friends objected and the even greater lack of certainty about how often these objections were adhered to, observations echoed in the current research.352

From the 1860s, there were instructions to asylums to notify coroners in the event of the death of an inmate. Scholars like Cullen, Hurren and Fisher have pointed out that knowledge of the relationship between the coroner and institutions, particularly asylums, and the operation of the coroner’s office is lacking.353 Zuck argues that because many coroners had held their office for long periods, local accords and “gentlemanly agreements” whereby resident doctors tended to be left to examine the dead body without the involvement of the coroner were commonplace.354 There were several cases, including some in this cohort discussed in more detail below, who were referred to the coroner and then an inquest carried out. Unfortunately, inquest reports could not be examined adequately.355 Referred cases were usually those of sudden death. For example, the coroner decided to hold an inquest on a patient who died four weeks after a fracture of the femur but who had no history of falls, concluding that the death was accidental with no blame to staff. Another case who had “banged” his head and later died was reported to the coroner. Others also reported this case, but the coroner decided there was no need for inquest. It is not clear from the

352 Phillip Fennell, Treatment without Consent: Law, Psychiatry and the Treatment of Mentally Disordered People since 1845 (Abingdon: Routledge, 2002); Wallis, Investigating the Body, 81.
354 Zuck, “Mr Troutbeck as the Surgeon’s Friend,” 260.
355 Coroner’s reports from Fair Mile were in a volume in the Berkshire Records Office but this was embargoed as more recent cases were also included in this book. Attempts to have those from the above period copied separately were technically unsuccessful.
records what the coroner’s examination consisted of, but it does not seem that a separate post-mortem was carried out or that the coroner attended the post-mortem. It may be that some of the cases where a post-mortem was carried out without consent were due to the coroner mandating a post-mortem as part of an inquest, but this is conjectural.

The information from the post-mortem reports was collected onto a spreadsheet using the method outlined in Chapter 1. Many of the entries on the post-mortem report were copied in verbatim including, importantly, the cause of death and the general appearance. Details of the main pathological findings from head, thorax and abdomen were transposed to the results file and where there were specific findings the information was copied unchanged. However, a couple of issues should be highlighted. Firstly, where no abnormality was reported the information was summarised briefly (usually using the acronym NAD (no abnormality detected). Secondly, details of findings of the meninges (coverings of the brain) and of the basal ganglia were not progressed as the findings were many, myriad and complex. Examples of a post-mortem report from this cohort can found in Appendix A and the derived summaries for each asylum cohort in Appendices D and E. A number of case-notes of patients in this post-mortem cohort were examined. These included all cases of exhaustion and findings and implications from these post-mortems and clinical notes are discussed in Chapter 4. Similarly, clinical, and pathological information from cases with a vascular cause of death is collated and examined in Chapter 5. In this chapter, the focus of the clinical enquiry is on the six cases where there was a sudden death or there was a suggestion of violence whether self-induced or external.

3.2 Findings and Commentary

3.2.1 Post-mortem delay

Details of the delay between the date and hour of death and date and hour of the post-mortem were recorded. The vast majority of post-mortems were within 36 hours of death and most within 24 hours. A few were performed two days later and for a very small number, there was a post-mortem delay of 60-72 hours. An overview of the data showed no impact from the more prolonged delays. However, it is important to remember that there was no refrigeration in the mortuary and so it is plausible that some of the reports of, for example, cerebral softening were consequences of the long time the cadaver lay at room temperature and were actually
liquefaction. Occasionally, a note was added to the effect that “the weather was very warm” but whether this was an observation based on what must have been an unpleasant task in these circumstances or a more scientific point is unclear.

### 3.2.2 Rates of post-mortems

In Fair Mile, 92% of deaths had a post-mortem over the ten-year period of this study (1895-1905). The LC reports on Fair Mile during this period were uniformly positive, the efficient running of the establishment and the high post-mortem rate coming in for specific praise. The near universality of post-mortems may have been driven by the Physician Superintendent Murdoch, who Wheeler describes as a “determined and industrious Scot”, perhaps linked to his training at Glasgow University Medical School which was a centre of pathological training and expertise. The post-mortem rate in Sunnyside was very substantially lower at 57%. This may have partly reflected the fact that at Sunnyside there were two fewer medical officers than at Fair Mile and this may also be the explanation of gaps in the Sunnyside post-mortem records (but not in deaths) of a few months here and there. Medical Officers performed the post-mortems in asylums, but this was something for which their medical training had prepared them. Nystrom argues that for the nineteenth century medical student, anatomy and pathology represented the most significant components of their education and that this knowledge was considered the basis of medical professionalism and a benchmark of their social identity. Andrews comments that Commissioners’ reports showed that post-mortem rates were lower in mixed Royal Asylums like Sunnyside, possibly related to the extra difficulty of getting consent from fee-paying relatives of private patients. The lower rate was not a reflection of any ambivalence on the part of the Physician Superintendent, Dr James Howden, a graduate of Edinburgh University (as stressed above, another centre of pathological excellence). Howden was an enthusiastic proponent of pathological research in asylums as evidenced by his writings, his encouragement of associates and the very high post-mortem rate at Montrose during the 1880s and early 1890s. Dr Howden published a paper on his findings from 235 post-mortem brains where he found 189 presented abnormalities. An associate, John Batty Tuke, of Fife and Kinross Asylum, also began publishing tables of observed brain lesions as an

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appendix to his asylum’s annual reports. Then in 1873, Howden’s former assistant William G. Balfour, who became Superintendent of Hampstead Asylum, collated all these observations with even more collected at the Colney Hatch Asylum, to present the records of 700 insane brains. Howden published his “Pathological Index” in 1891. In his annual report to the LC of June 1893 he stated that he trusted that it will “be found a valuable aid in other Asylums and general hospitals in the pursuance of pathological research.” The Index was warmly supported by the MPA the same year who hoped its use would become widespread although there is no evidence that transpired. However, several detailed manuals for post-mortems were available in the UK at this period. In particular, two alienists, Newth from Sussex and Lewis from the West Riding, authored texts specifically for use in asylums which focussed on techniques for handling and assessing brain. Dr John Havelock who replaced Howden in 1897 was another graduate of Edinburgh but his commitment to pathological enquiry in asylums is less clear and the rate of post-mortem began to fall sharply under his tenure (from about 65% to 30%). In 1901 Dr Fraser, a Commissioner in Lunacy for Scotland, exasperated about this decline, recommended the appointment of a third medical officer, some of whose time could be devoted to scientific research in connection with the Scottish Asylums Laboratory scheme which had been set up at the REA by Thomas Clouston and the resident pathologist, William Robertson, in 1896. The Scottish scheme mirrored the English set up at Claybury under William Mott, a full-time pathologist (the first laboratory for research into mental pathology funded by a public health body). In 1897, Sunnyside duly (or perhaps dutifully) joined the scheme, whose main purpose was research on the pathology of the brain in insanity. What impact this affiliation had, however, is difficult to discern.

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362 James Howden, *Scheme for Pathological Index* (Glasgow: MacDougall: 1891).
Over the 120-month period in Sunnyside and the 122-month period in Fair Mile there were respectively 72 and 100 deaths of inmates with an admission label of mania or melancholia who were aged 55 or younger at death who had a post-mortem. Over the same periods, the number of cases of mania or melancholia in the same age range who died in these asylums but did not have a post-mortem were 45 and 23 respectively. This means that there was a post-mortem rate of 62% in Sunnyside and 81% in Fair Mile in this population. These figures are comparable to an overall post-mortem rate of 57% in Sunnyside and that of 92% in Fair Mile over the same period. The lower post-mortem rate of 81% in the younger mania and melancholia group at Fair Mile and the more or less equal rate at Sunnyside shows that these deaths were not prioritised for post-mortem examination. A few deaths in the register but without post-mortem had a note to the effect that consent had not been forthcoming but the reasons why other patients had post-mortems and others not is not clear in either institution. The demography of the cases with and without a post-mortem were almost identical as were their causes of death (data below). Thus, at least to that extent, the studied post-mortem samples are representative. Of the 172 selected cases, there are 116 cases with an admission label of mania and 56 cases with one of melancholia, with a 2:1 female/male ratio. The mean age at death of the mania cases was 42 years and that of the melancholia group 43. The average age of death of mania cases was broadly similar in both establishments, but the deaths of melancholics at Sunnyside were younger than those at Fair Mile, with a mean age of death of 40 years, generated by the many young deaths from tuberculosis in Montrose.

3.2.3 Length of admission

As discussed in Chapter 2, one of the main interpretations of the high death rate in asylums is that the frequency of deaths soon after admission is a direct consequence of the weak and parlous physical state of those admitted. It is therefore of interest to test whether this observation is also found in this younger population of deaths where physical conditions, at least those of a degenerative nature, might be less common. The length of stay, prior to death was divided into five arbitrary categories for this cohort and is shown in Table 3.1.
<table>
<thead>
<tr>
<th>Asylum &amp; number of cases (n)</th>
<th>Length of admission before death</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Less than 1 week</td>
</tr>
<tr>
<td>Sunnyside n=72</td>
<td>n</td>
</tr>
<tr>
<td></td>
<td>%</td>
</tr>
<tr>
<td>Fair Mile n=100</td>
<td>n</td>
</tr>
<tr>
<td></td>
<td>%</td>
</tr>
<tr>
<td>Combined n=172</td>
<td>n</td>
</tr>
<tr>
<td></td>
<td>%</td>
</tr>
</tbody>
</table>

Table 3-1 Length of stay in the Asylum prior to death in the post-mortem cohorts.

It can be seen from Table 3.1 that the phenomenon of death early in admission was seen in both asylums. 18% of cases died within a month of admission and half of those within a week. 40% died in the first year in the asylum while only about 25% of those who died had been resident for more than five years at the time of their demise. These figures are very comparable to those reported elsewhere, confirming that factors associated with the admission and the disorder itself, rather than the incarceration, were critical in the demise of many patients.

### 3.2.4 External appearance

As shown in Fig. 3.7, the first component of the post-mortem proforma was to describe the external appearance ("External Appearances, and whether or not Bedsores present"). Four patients were noted to have bruises: three of them had multiple bruises. One of the patients with many bruises (GH) did experience violence and her case is described in detail below. In the other two multiple bruises cases, it was not clear whether they were the result of their severe manic insanity or being restrained during it. Five patients had bedsores: all were "much emaciated", and most had very marked tuberculosis. One patient who died of pneumonia and was said to have had
meningitis had ulcers on the skin. One man had had his penis amputated for reasons that are obscure. Part of the rationale for post-mortems being prescribed by the LC was to act as a check on staff and detect and prevent potential ill treatment. These findings were reassuring to visiting Commissioners and this satisfaction was documented in various Annual Reports.

3.2.5 Ribs

Of particular concern during this period was the number of fractures particularly of ribs found in asylum post-mortems. It was mandated in the proforma in both Fair Mile and Sunnyside, and in many other Victorian asylums, to “Describe condition of Ribs.” Asylums were accused of wilful mistreatment on this basis, but further study summarised by Wallis showed that the insane - particularly, for various reasons, those with GPI - were peculiarly prone to bone disease. The General Statement Book of Fair Mile Asylum documents a case of GPI who died in 1903 with fractured ribs. The Commissioners were involved, and a special meeting held into the circumstances, but no action was deemed necessary. In the 172 post-mortems of this younger cohort examined here, no cases with fractured ribs were seen. Nonetheless, there was obvious concern in both asylums about being accused of maltreatment based on fractured ribs: both asylums emphasised in their reports that a patient found to have fractured ribs on the wards had been admitted with the ribs already broken.

3.2.6 Causes of death

The “certified cause of death” was taken from the pathological records. In the rare instances where two causes were given, only the first of those was analysed. Using the method described in Chapter 2, the causes of death were allocated to one of five sub-categories: Exhaustion, Vascular, Cerebral, Infective and Other. The 18 “Other” cases included 2 cases of probable suicide, 2 cases of “choking” and 7 deaths from cancer. The remainder comprised deaths from intestinal obstruction and/or perforation of the bowel. These latter deaths may reflect the rudimentary nature of medicine and surgery in the rural asylum setting. Prompt surgery in a general hospital setting might have been lifesaving for these cases. Care was taken to distinguish between evidence of past, but non active, tuberculosis (e.g., scars) and active tuberculosis (e.g., caseation, nodules).

First, the cause of death in the post-mortem records was compared with that in the death register. In cases where there had been a post-mortem, the cause of death in the register is exactly the same as that found in the pathological records. Therefore, it seems safe to assume that at least the cause of death section in the register was completed after the post-mortem was performed. The nomenclature used in the death register to describe the cause of death in those without a post-mortem was almost identical to those cases who did have one. No unexpected causes of death were evident in either group. Overall, the cause of death attributions gives the strong impression of homogeneity rather than heterogeneity, supporting Sturdy’s contention that the principal concern of late nineteenth-century pathology was to “effectively... write out any idiosyncrasy from the clinical narrative” with the identification of “typical rather than singular cases.”

The proportion of female to male deaths and the mania to melancholia ratio were very similar in the groups who did and did not have a post-mortem and the average age of the two groups was also nearly identical. Deaths in the various sub-categories, taking mania and melancholia together, were compared between asylums and between those with and without a post-mortem and the results of this enquiry are shown below in Table 3.2.

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<table>
<thead>
<tr>
<th>PM or no PM</th>
<th>Asylum and total number of cases (n)</th>
<th>CAUSES OF DEATH</th>
<th>Exhaustion</th>
<th>Cerebral: Senile decay/Cerebral softening/Cerebral atrophy</th>
<th>Vascular disease: Morbis cordis/Heart disease / Heart failure / apoplexy</th>
<th>Infection</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>With PM</td>
<td>Sunnyside n=72</td>
<td>n</td>
<td>5</td>
<td>2</td>
<td>10</td>
<td>46</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>%</td>
<td>7</td>
<td>3</td>
<td>14</td>
<td>64</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>Fair Mile n=100</td>
<td>n</td>
<td>13</td>
<td>7</td>
<td>17</td>
<td>55</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>%</td>
<td>13</td>
<td>7</td>
<td>16</td>
<td>55</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Combined n=172</td>
<td>n</td>
<td>18</td>
<td>9</td>
<td>27</td>
<td>101</td>
<td>17</td>
</tr>
<tr>
<td></td>
<td></td>
<td>%</td>
<td>10</td>
<td>5</td>
<td>16</td>
<td>59</td>
<td>10</td>
</tr>
<tr>
<td>No PM</td>
<td>Sunnyside n=45</td>
<td>n</td>
<td>7</td>
<td>6</td>
<td>5</td>
<td>24</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>%</td>
<td>16</td>
<td>13</td>
<td>11</td>
<td>53</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fair Mile n=23</td>
<td>n</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>16</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>%</td>
<td>9</td>
<td>13</td>
<td>9</td>
<td>69</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Combined n=68</td>
<td>n</td>
<td>9</td>
<td>9</td>
<td>7</td>
<td>40</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>%</td>
<td>13</td>
<td>13</td>
<td>10</td>
<td>59</td>
<td>4</td>
</tr>
</tbody>
</table>

Table 3-2 Recorded causes of death (placed into 5 sub-categories) in mania and melancholia cases dying ≤ 55 years of age in those with and without a post-mortem (PM) in the two Asylums.

As can be seen in Table 3.2, the frequencies of causes of death were broadly similar between the two asylums and between those who had and did not have a post-mortem. The only exception to
this, and this difference is of degree rather than kind, is the higher rate of infective deaths in Sunnyside (64%) compared with Fair Mile (55%). This difference was largely driven by the somewhat higher death rate from tuberculosis in Sunnyside (44% of deaths) as compared to Fair Mile (32% of deaths). 40% of the cases with mania and melancholia who did not have a post-mortem died of tuberculosis although this cause of death ascription was of course made without the benefit of post-mortem pathological examination. Cerebral causes of death were 5% in those who had a post-mortem compared with 13% who did not. This finding supports MacKinnon’s assertion that the terms used to denote a cerebral cause of death were, at least sometimes, “generic labels for mental illness.” Three cases from Sunnyside are ascribed as “organic brain disease” which might be an acknowledgement of the difficulties of ascribing a cause of death in insanity given the very abnormal mental state of sufferers and the lack of an obvious cause of death. However, 3 cases were given the label of “cerebral atrophy” and 3 “cerebral softening” without the benefit of a post-mortem. The issues thus raised are discussed in Section 3.2.8.1 (below). The percentage of patients ascribed a cardiac death was higher in the group with a post-mortem (15%) compared to those who did not have a post-mortem (10%). The differences in vascular causes of death between the groups with and without a post-mortem might be a selection bias revealing more interest in a post-mortem in those with a likely vascular cause rather than those with features suggestive of one of cerebral causes, but it is more likely this finding reflects the nature of vascular disease in this era with it not being frequently considered, unless obvious, and often only found at post-mortem. The similarities between these disparate asylums’ datasets are more compelling than the relatively small differences and provide some reassurance that the results reported here are a good approximation of the pattern of asylum deaths in the late Victorian era.

The cause of death data was then examined to compare deaths in mania with deaths in melancholia. The results of this comparison are seen in Table 3.3 below. This table also contains data on the mean age of death in the various subgroups and in deaths with tuberculosis (TB) in particular.

369 MacKinnon, Bodies of Evidence, 80.
<table>
<thead>
<tr>
<th>Asylum Group</th>
<th>CAUSE OF DEATH AFTER POST-MORTEM PERFORMED</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Exhaustion</td>
</tr>
<tr>
<td>Sunnyside Mania n= 45 Age= 43</td>
<td>n(%)</td>
</tr>
<tr>
<td></td>
<td>Age</td>
</tr>
<tr>
<td>Sunnyside ME n= 27 Age= 40</td>
<td>n(%)</td>
</tr>
<tr>
<td></td>
<td>Age</td>
</tr>
<tr>
<td>Fair Mile Mania n= 71 Age= 42</td>
<td>n(%)</td>
</tr>
<tr>
<td></td>
<td>Age</td>
</tr>
<tr>
<td>Fair Mile ME N= 29 Age= 45</td>
<td>n(%)</td>
</tr>
<tr>
<td></td>
<td>Age</td>
</tr>
<tr>
<td>TOTAL Mania n= 116 Age= 42</td>
<td>n(%)</td>
</tr>
<tr>
<td></td>
<td>Age</td>
</tr>
<tr>
<td>TOTAL ME n= 56 Age= 43</td>
<td>n(%)</td>
</tr>
<tr>
<td></td>
<td>Age</td>
</tr>
</tbody>
</table>

Table 3-3 Recorded causes of death (placed into 5 sub-categories) in mania and melancholia (ME) cases dying ≤ 55 years of age in the two Asylums and age at death (years).

As can be seen from Table 3.3, deaths from tuberculosis occurred on average at an earlier age than other causes of death, particularly in Sunnyside where the average age of deaths from...
tuberculosis was 37 years. The overall death rates from tuberculosis in this sample was 37%, much higher than the 15% of all asylum deaths reported in Chapter 2. The reason behind this difference is the fact that this cohort have been selected based on relative youth and tuberculosis was a major cause of premature mortality in the Victorian era. However, it is noteworthy that despite the young average age of death with TB in the current sample (at just under 40), this is considerably older than the age of death found in the community at that time which was centred between the ages of 15 and 35. Drapes showed that the prevalence of tubercular death of younger inmates of the asylums was identical to that of young adults in the community. The massively increased death rate from tuberculosis in asylums occurred in those over 40.

In 1863, Thomas Clouston used post-mortem records to establish phthisis as a large component of asylum mortality.370 Contemporary authorities, such as Drapes and Crookshank, agreed and also came to the view that this excess of tubercular deaths was a consequence of inmates having acquired tuberculosis during their stay in the asylum. Mott, a pathologist for the LCA commented that the “large class of the chronic insane who become demented after the acute attack has subsided are especially liable to tubercular infections; body functions and movement are slow, and the lungs not properly expanded.” 371 The views of these authorities are supported by subsequent commentators.372 Harris and colleagues show that tuberculosis was a particular hazard for younger patients admitted to the asylum and that, as here, the main spike in deaths from this cause was two to five years after admission.373 However, several patients (n=11) in this cohort died of tuberculosis within a year of being admitted to hospital and therefore it is plausible that those patients were suffering from severe and active tuberculosis on admission. Indeed, it is possible that the infection, coloured their mental state on admission. Even more strikingly, several young men with melancholia died of tuberculosis within a month of their admission. One can speculate that in these cases the melancholia was linked to or secondary to an active and debilitating tubercular condition and they were thus cases of “phthisical insanity”, as discussed in Chapter 2, Section 1.2.2.

Table 3.3 also shows that, while causes of death are broadly similar between the two conditions, deaths from vascular disease were more common in mania (20%) compared to melancholia (7%). Vascular pathology is discussed in more detail below. The opposite pattern was seen with infective diseases of which 66% of melancholics died compared to 55% of mania cases. This latter difference was largely driven by tubercular deaths: 34% of mania cases died of tuberculosis but 45% of melancholia cases succumbed to this disease. The high death rate from tuberculosis in melancholia had been noted in contemporary enquiries into asylum deaths. Esquirol reported on his new disease “lypemania” (as he dubbed what Berrios shows is essentially melancholia) in 1820 and found that about a third died, “often of tuberculosis.” Historians have uncovered further evidence to support these conclusions. Harris and colleagues in their history of deaths in melancholia from a North Wales asylum show that tuberculosis accounted for 30% of the mortality. This figure is compatible with that found here in a younger cohort (40%). This high death rate in melancholia continued well past the time the disease was getting under control in the general population. For example, similar findings to those of Harris were found by Malzberg in 1937 and as late as 1959, Norris observed that tuberculosis contributed substantially to the total number of deaths in her sample of mentally ill from London.

The association of melancholia, tuberculosis and death was well recognised in the eighteenth and nineteenth centuries and was often romanticized in literature and music. Susan Sontag shows how, paradoxically, tuberculosis served as a metaphor for positive attributes, such as “heightened beauty, refined sensibility, and artistic creativity.” She describes how this “melancholy character – or the tubercular – was a superior one: sensitive, creative, a being apart.” Dumas is quoted as declaiming that “… it was the fashion to suffer from the lungs; everybody was consumptive, poets especially; it was good form to spit blood after each emotion that was at all sensational, and to die before reaching the age of thirty.” The romantic allure of tuberculosis has been much examined

374 Berrios, “Melancholia and Depression,” 300.
by medical and literary historians. For example, Clark Lawlor and Akihito Suzuki demonstrate how tuberculosis’s “localization in the “upper” part of the body, the slow and gentle emaciation, pale skin-color, flushed cheeks...were vital components of the metaphor.” This narrative was particularly powerfully attributed to women. Lawlor and Suzuki describe many examples in literature of women dying of a “romantic love-melancholy that begets a consumption.” The disease in popular imagination became enmeshed with the notion of sensibility in which “the slender body became a sign of personal sensitivity, creativity, and taste.” Traditions of “the good death” combined with this sensibility to create “a culture of an often pleasurable and aestheticized consumption, and so powerful was this “fantasy” that people would attempt to live (and die) according to its image.”

The metaphor was so pervasive at the time that it led, in part, to conjecture that tuberculosis-prone individuals had particular predispositions. Gotthard Booth, a commentator on the history of psychoanalytical theory, baldly states, “It has been known since ancient times that they are romantic characters.” He describes how a view developed amongst some practitioners that patients with tuberculosis showed that “satisfaction of their affectionate needs is a vital necessity which they often pursue in blatant disregard of physical health and socioeconomic considerations.”

Sontag observes that the TB “myth” survived despite human experience and growing medical knowledge for nearly two hundred years. However, these cultural discourses were much less extended to the pauper classes and not at all to the pauper insane. The late nineteenth century was also the period in which the negative image of the disease and its position as a major threat to public health began to burgeon and a large-scale war against tuberculosis took root.

Bryder argues this onslaught was fuelled by concerns over national efficiency and the view that the poor health of the working class was undermining Britain’s ability to remain competitive as a world power. In 1899, in a study of the decline in tuberculosis, Sir Hugh Beevor, Assistant Physician to the City of London Hospital for Diseases of the Chest, attributed the decline in phthisis to the effect of improved nutrition and in particular to the use of cod-liver oil for the treatment of the disease. According to the historian Leonard

Wilson, Beevor identified a coincidence between the fall in phthisis mortality and a decline in the price of wheat, a doubling in the real incomes of wage earners and a drop in the number of paupers. In contrast to England, tuberculosis mortality had not declined in Paris, a difference that Beevor attributed to the vegetable diet and lower wages in France. A major proponent of this theory was Thomas McKeown who published his famous graph of the decline of pulmonary tuberculosis mortality in England and Wales and expressed the view that the management strategies in use in the nineteenth or early twentieth century had had no significant influence on the disease or its course. However, Wilson shows that, after the discovery of the tubercle bacillus in the 1870s, the question of effectiveness of treatment was not the pivotal issue and that prevention of tuberculosis became the focus of public health policy - a campaign that was eventually effective. Wilson and Worboys insist that McKeown’s “frequently repeated” claim that the public health movement played no significant part in the decline of tuberculosis is invalid. Interestingly, medical authorities of the early twentieth century, like Arthur Newsholme, Medical Officer of Health, Brighton, considered that institutions such as workhouses, infirmaries and asylums played a role in bringing about the decline of tuberculosis in Britain by segregation of those afflicted thus reducing spread to the young. Newsholme estimated that 20% of the total number of consumptives were so segregated and that, if their average period of stay was one-third of a year, representing one ninth of their period of infectivity, then their segregation would prevent the spread of about 2% of the total tubercular infection per year which was, in fact, the annual decline in tubercular mortality from 1871 to 1905. Thus, Newsholme argued that the influence of segregation was fully adequate to produce the recorded historical decline of tuberculosis. In this conclusion, he is broadly supported by Wilson’s subsequent scholarship. Thus, the asylum (via partial quarantine of tubercular inmates) played its (largely unwitting) part in reducing public tuberculosis while, at the same time, unfortunately increasing the risks for its inmates, especially those with melancholia.

Pneumonia was also a particularly common cause of death in those with melancholia and, to a lesser extent, mania accounting for 16% of the deaths. It is noteworthy that the numbers to whom pneumonia was ascribed as the cause of death likely represents the true figure as it was based on the presence of the correct pathology for pneumonia (with consolidation in one or other lung and usually a finding or either red or grey hepatinisation, the hallmark terms for the pathological changes of pneumonia). It seems probable that crowded asylums encouraged the spread of respiratory infections, but the predilection of the insane to such conditions may also relate to the debilitated state of many patients linked with the then widely held notion that lung expansion was less adequate than normal or showed, in Crookshanks’s words, “peculiarities” in those with prominent mental symptoms. Cullen argues, using contemporary sources, that there was a class effect in the reporting of lung pathology: she collates evidence that patients who were reported to have suffered “congestion” may have been considered the more upstanding members of society, while those who died of “phthisis” or “inflammation” were seen as members of the lower classes. If such an effect exists, it is not discernible in the current findings.

Deaths from gastrointestinal infections such as dysentery and enteric fever (typhoid) accounted for 8% of the mortality in Fair Mile but were not seen at all in Sunnyside. At Fair Mile, there was no evidence of the epidemics that had ravaged asylums in the earlier part of the century, but little “waves” of gastrointestinal infections were evident. As described above, these deaths were of great concern to the staff, Visitors and visiting Commissioners and were probably linked to water and sanitation problems on that site. In January 1904 there was an epidemic of dysentery. In his 1904 report, Murdoch bemoaned “This disease is common to all asylums and in the larger ones becoming a regular scourge... the insane appear to be particularly susceptible.” The source, he wrote, is “a harassing mystery” as all surfaces were said to have been disinfected. Perhaps the better water in Angus (and the colder temperatures) protected the Sunnyside inmates? These enteric infections, however, are more likely to occur in those already run down, emaciated or poorly nourished for other reasons. Contaminated water seems only a partial explanation as,

390 Cullen, “Post-mortem in the Victorian Asylum,” 289.
391 The water at Sunnyside was tested and shown to contain “no colonies or poisons” but the water analyst did not consider it first class water (it had high ammonia and organic material content). He noted, however, that it was not dangerous.
despite asylum staff drinking the same water, I found only one report of a death from enteric fever amongst them.

In the younger cohort of patients with mania and melancholia, infections were the main killers - about 3 out of 5 patients succumbed to an infectious disease. Commentators share the view that a factor underlying this high frequency is the susceptibility of sufferers of insanity to severe infections which, as Ernst adduces, were conditions that particularly affect those already suffering from a fragile state of health, particularly those with chronic debilitating disorders, including insanity.392 This evidence supports my overall viewpoint that, if infections were the seed, the soil was already prepared with the ravages of insanity and the deprivation, both social and economic, that insanity brought in its wake. All of this was amplified by the pitfalls of incarceration, most notably overcrowding and the stress of continual uncertainty and loss of agency.

### 3.2.7 Relationship of bodily state at death to cause of death

Malnutrition was commonplace in the late Victorian era, albeit to a lesser degree than that of the earlier parts of the nineteenth century. Recurrent ravages from infection and economic hardship took their toll on the population’s nutritional status and these effects were even more marked in the insane.393 Therefore, an assessment of the impact of nutritional status on the causes of death in this cohort was carried out. In the more detailed analysis of sub-groups carried out in succeeding chapters, nutritional status on admission is examined but here the entries under “General appearance” at post-mortem were examined. All cases dying of tuberculosis were emaciated at death and have been omitted from this analysis. The habitus (body build and constitution) of the remainder essentially fell into two groups, either emaciated (sometimes qualified with words like “very,” “much,” “a little,” or “rather”) or well nourished. The findings are shown in Table 3.4.

---

<table>
<thead>
<tr>
<th>Asylum Number (n)</th>
<th>Group</th>
<th>Emaciated (or similar)</th>
<th>Well nourished (or similar)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fair Mile n=68</td>
<td>Total</td>
<td>39 (57%)</td>
<td>29 (43%)</td>
</tr>
<tr>
<td></td>
<td>Exhaustion</td>
<td>11</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Cerebral</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>Infective (non-TB)</td>
<td>12</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Sunnyside n=40</td>
<td>Total</td>
<td>20 (50%)</td>
<td>20 (50%)</td>
</tr>
<tr>
<td></td>
<td>Exhaustion</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Cerebral</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>Infective (non-TB)</td>
<td>8</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Combined n=109</td>
<td>Total</td>
<td>59 (55%)</td>
<td>49 (45%)</td>
</tr>
<tr>
<td></td>
<td>Exhaustion</td>
<td>14 (78%)</td>
<td>4 (22%)</td>
</tr>
<tr>
<td></td>
<td>Cerebral</td>
<td>7 (78%)</td>
<td>2 (22%)</td>
</tr>
<tr>
<td></td>
<td>Vascular</td>
<td>10 (37%)</td>
<td>17 (63%)</td>
</tr>
<tr>
<td></td>
<td>Infective (non-TB)</td>
<td>20 (54%)</td>
<td>17 (46%)</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>8 (47%)</td>
<td>9 (53%)</td>
</tr>
</tbody>
</table>

Table 3-4 Body habitus at death and cause of death in both Asylums, n=109 (TB deaths excluded).

A degree of emaciation at death was somewhat more common (55%) than not (45%). Therefore, an emaciated state at death was very far from universal. There was no major difference between the two asylums and the pattern of distribution of the different categories of death was nearly identical from both settings, which may add some credence to the reliability and representativeness of the findings. It can be seen that a very high proportion of those dying with exhaustion were emaciated. The issue of whether this is cause or effect will be taken up in the
following chapter. Conversely, only approximately a third of those dying with a vascular disorder were emaciated at death, lending weight to the standpoint that the mechanics and modes of such deaths showed different characteristics from the remainder of the deaths, as will be argued in Chapter 5.

About half of those dying of a non-tubercular infection were well nourished; a finding that belies the widely held notion that it was the combination of infection and malnutrition that led to the high frequency of asylum deaths. It is clearly more complicated than that. To back this up, it has been shown that the reduction in mortality in the late Victorian period in England was evident across all social groups and is argued that a nutrition-based effect on mortality would have shown a class effect. In a significant number of cases, emaciation was an expected association to find at death. Examples of this include deaths from exhaustion, cancer and prolonged gastro-intestinal infection. If these cases are removed for the purpose of a sub-analysis, then about three out of five of the remainder were well nourished at death. These observations, taken together, suggest that malnutrition is neither necessary nor sufficient to explain death in asylums. A degree of emaciation at or during admission may well have tipped the scales and hastened death in some, but a simple linking of poor nourishment to the excess deaths of the asylum insane does not hold water.

### 3.2.8 Specific features of the post-mortem findings

Considerable time spent scrutinizing and categorising the post-mortem reports led to the identification of a pattern of common findings, on which this section provides a commentary. Although there was some variation in the language used between the differing staff doctors who performed these procedures in the two asylums, similarities in the accounts were striking. Considerable uniformity and coherence of praxis was exhibited in the asylum necroscopy, reflecting that pathology was extensively and relatively consistently taught in late Victorian medical schools and had become an established bench science. The observation that the post-mortem proforma and the descriptions therein were almost identical in two asylums 500 miles apart also supports this conclusion.

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Key words or phrases from the reports were recorded and quantified. There were long and detailed accounts of active tubercular lesions when present. Cases with active tuberculosis also had many changes in the brain and heart. These were documented but are not particularly relevant or important since they conform to well-known physical changes associated with active tuberculosis and are likely to obscure changes in or associations with any pathology related to the underlying insanity. The findings from cases of death with active tuberculosis have therefore been excluded from the assessment of changes in brain and heart, found below, but all other subjects have been included.

3.2.8.1 Changes in the brain

Post-mortem reports were replete with references to the brain, grey matter or cortex being soft or a seemingly equivalent word such as friable. Similarly, there were multiple mentions that the grey matter was shrunken, often associated with the phrase “sulci gaping.” In Fair Mile’s post-mortem reports, there were numerous accounts that the white matter was oedematous and, more rarely, swollen. The Sunnyside reports contained frequent mentions of generalised cerebral oedema or swelling. Both asylums reported frequently that the whole brain or a part of it was “congested” and/or “hyperaemic.” The state of the basal arteries of the brain (principally the basilar and middle cerebral arteries) - the account of them in the post-mortem reports was restricted to either “atheromatous” or normal - was also documented. It was not clear whether this was assessed by the arteries being cut or simply observed from the outside – the latter is apparently easy to do. Another finding quite commonly reported in both asylums’ records was “punctate” lesions or “punctate vasculosa” in the white matter. The use and meaning of such terminology are outlined below. Other pathological changes in these brains were reported infrequently. Some of these will be discussed below in relation to individual cases but only the above common observations are analysed. Mania and melancholia were examined separately but no clear difference in the frequency of these brain findings was seen and, likewise, no difference in how often these pathological features were mentioned in the reports from the two asylums was

395 A sulcus in neuroanatomy is a depression or groove in the cerebral cortex.
396 An excess of small active blood vessels throughout the tissue.
397 Atheroma is a deposit of fatty material on the inside surface of an artery.
398 Personal communication from Professor A K Foulis, Professor of Pathology, University of Glasgow.
399 Punctate white matter is where lacunae (gaps or holes) are found. “Punctate vasculosa” is a term used to describe the situation where the lacunae are associated with an excess of small vessels.
observed. The information of the most commonly reported brain changes for both mania and melancholia and for both asylums was therefore combined and is laid out in Table 3.5 below.

<table>
<thead>
<tr>
<th>Pathological Finding</th>
<th>Number</th>
<th>Cause of death sub-category</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>Exhaustion n=18</td>
</tr>
<tr>
<td>Brain ‘softening’</td>
<td>38 (37%)</td>
<td>5/18 (28%)</td>
</tr>
<tr>
<td>‘Shrunken’ grey matter or cortex</td>
<td>32 (31%)</td>
<td>6/18 (33%)</td>
</tr>
<tr>
<td>‘Oedema’ of white matter</td>
<td>32 (31%)</td>
<td>5/18 (28%)</td>
</tr>
<tr>
<td>‘Congestion/Hyperaemia’</td>
<td>39 (38%)</td>
<td>14/18 (78%)</td>
</tr>
<tr>
<td>‘Atheroma’ of basal vessels</td>
<td>28 (27%)</td>
<td>7/18 (39%)</td>
</tr>
</tbody>
</table>

Table 3.5 Pathological findings (using closely equivalent terms) in the cause of death sub-categories in post-mortem brains of deaths in mania and melancholia (n=103, excluding deaths from TB, n= 64, and the 5 cases where the head was not examined).

It can be seen in Table 3.5 that each of these abnormalities was found in about a third of cases. Several cases had more than one finding and, occasionally, several findings were reported together. The detail can be seen in Appendices D and E. The most common numerically was “Congestion/Hyperaemia” with 38% of cases having this term applied. “Congestion” was the most common term, followed closely by “Congestion and hyperaemia”, with occasional mentions of just “hyperaemia.” Congestion is a highly subjective parameter and one that may be linked to the agonal status of the person who died. It has long been thought of as a non-specific finding, particularly in the context of asylum deaths where commentators have thought its use represented the (valiant or vain) search for brain change in insanity. The vague non-specific changes of cerebral congestion, was frequently described in studies of the period but
commentators have noted these studies lack both consistency and control.\textsuperscript{400} However, congestion was not reported in two thirds of cases, which may be evidence for some degree of specificity. It is noteworthy that congestion and hyperaemia were more common in cases dying of or with exhaustion. The implications of this finding are discussed in Chapter 4. Conversely, and somewhat surprisingly, congestion was not seen in what I have dubbed cerebral causes of death (that is cerebral softening or atrophy) and rare (11\%) in patients dying of other causes. These observations also suggest some specificity but the low frequency of congestion in these groups, who comprised inmates who had a label of insanity may also point to congestion being related to the mode of death, rather than a feature of insanity itself, echoing Pritchard’s contention, expressed in 1844, that the congestion frequently found in lunatics’ skulls was “an effect rather than a cause.”\textsuperscript{401}

Many of these issues apply to the findings of softened or shrunken brain or cortex and to white matter oedema. These are subjective observations likely to have relatively poor intra- and inter-rater reliability. However, for many patients, it is likely these pathological changes were present as the degree of them appeared marked. Examples include comments like brain “extremely soft” and white matter “sodden and oedematous.” In many cases, the shrinking of the cortex led to the folds of the brain (the sulci) being described as “gaping.” Atheroma in the brain vessels was seen in 27\% of cases and was more common in those groups with more frequent brain changes (the cerebral and exhaustion groups) and uncommon in those groups with infrequent brain changes. This association is further examined below.

3.2.8.2 Changes in the heart and vessels

There were many reports that the heart muscle (myocardium) was soft and this was often accompanied by a qualifier such as “and friable” or “and fatty.” All these terms were placed together for the purpose of this analysis as they are thought by commentators to be synonymous.\textsuperscript{402} Similarly, there were multiple references to the heart being hypertrophied (larger in size). Sometimes only the left ventricle was described as hypertrophied and rarely only the right


ventricle, but these variants have been placed together. Finally, the arteries within the heart were frequently described as “atheromatous” and sometimes as calcareous (calcified). Atheroma in the aorta (sometimes the reference was to “commencing atheroma of the aorta”) was also not an infrequent finding. It was not clear whether these findings were made by the arteries being cut or simply observed from the exterior. Mania and melancholia had different rates of vascular death but there was no clear difference in the frequency of these pathological findings between these conditions and, likewise, no difference in the frequency of these post-mortem findings between the asylums was observed. The information for both clinical labels and both asylums was therefore combined and is laid out in Table 3.6 below.

<table>
<thead>
<tr>
<th>Pathological Finding</th>
<th>Cause of death sub-category</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total group (n=103)</td>
</tr>
<tr>
<td></td>
<td>n (% of group)</td>
</tr>
<tr>
<td>Hypertrophy n (%)</td>
<td>30 (28%)</td>
</tr>
<tr>
<td>Myocardium soft/friable/fatty n (%)</td>
<td>46 (43%)</td>
</tr>
<tr>
<td>Atheroma of cardiac vessels n (%)</td>
<td>41 (38%)</td>
</tr>
</tbody>
</table>

Table 3-6 Pathological findings in the cause of death sub-categories in post-mortem hearts of deaths in mania and melancholia (n=103, excluding deaths from TB (n= 64) and the 5 cases where the head was not examined).

The most commonly report finding was that the myocardium was “soft” or “soft and friable” or “soft with fatty change.” The phenomenon was intriguingly common (56%) in cases who died by exhaustion, and this association is examined in Chapter 4 where an extended cohort of such cases is examined. The softening of the myocardium has often been dismissed by commentators as vague and therefore insubstantial, but, in this context, it is noteworthy that the finding was rarely found in the cases who died of “Other” causes. The history of the development of concepts of heart disease and atheroma and where the label “fatty heart” fits into these narratives will be
comprehensively addressed in Chapter 5. Hypertrophy of the ventricles, most commonly the left ventricle, was seen in a significant number of cases particularly, and not unexpectedly, among those cases with a vascular cause of death. Myocardial hypertrophy is a relatively “hard” sign and a finding that generally indicates cardiovascular pathophysiology as is discussed further below and in Chapter 5. A category where atheroma was reported either in the vessels of the brain or the heart or both was created to investigate whether vessel pathology was associated with changes in the brain at post-mortem. The details are shown in Table 3.7.

<table>
<thead>
<tr>
<th>Pathological Finding</th>
<th>Number (% of total)</th>
<th>With atheroma of vessels (% of group)</th>
<th>Without atheroma of vessels (% of group)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brain “softening”</td>
<td>37 (35%)</td>
<td>22/37 (60%)</td>
<td>15/37 (40%)</td>
</tr>
<tr>
<td>“Shrunken” grey matter or cortex</td>
<td>31 (30%)</td>
<td>18/31 (58%)</td>
<td>13/31 (42%)</td>
</tr>
<tr>
<td>“Congestion/Hyperaemia”</td>
<td>41 (40%)</td>
<td>20/41 (49%)</td>
<td>21/41 (51%)</td>
</tr>
<tr>
<td>“Oedema” of white matter (Fair Mile only, n=65)</td>
<td>29 (44%)</td>
<td>17/29 (59%)</td>
<td>12/29 (41%)</td>
</tr>
<tr>
<td>Total group</td>
<td>103</td>
<td>51 (50%)</td>
<td>52 (50%)</td>
</tr>
</tbody>
</table>

Table 3-7 Pathological findings (using closely equivalent terms) in the brains of patients with and without vessel atheroma (in heart and/or brain) (n=103, excluding deaths from TB (n=64) and the 5 cases where the head was not examined).

It can be seen in Table 3.7 that almost exactly half of these patients had atheromatous change in either the heart or brain vessels or both. Brain softening and brain shrinkage was seen in about 60% of the patients with a report of an atheromatous artery but only in 40% of those without such a report. This suggests that the brain process may have been caused or worsened by the presence of atheroma. Atheroma of the vessels is likely to be associated with disruption of the blood supply and with either ischaemia (shortage of blood) or haemorrhage (bleeding). Karl Rokitansky, (1804–1878) an eminent Viennese pathologist, described various types or stages of cerebral softening in 1855. He and a number of eminent pathologists wrote about the association between brain softening (and, to a lesser degree, brain shrinkage) and changes in vessels and speculated about
probable ischaemic or haemorrhagic origin of cerebral softening. The relationship evident here between the changes found in the vessels and those found in the brain supports those contentions. This association is also an argument against the notion that both sets of findings were random and adds credence to the idea that other processes, not just susceptibility to infections, were operating in these young cases of insanity. It is conceivable that disrupted blood supply to the brain may have been causing both the pathological changes found in this study and some or all the symptoms experienced by some of these insane patients prior to death. However, it is clear that atheroma is neither necessary nor sufficient to cause such changes in the brain, as some cases with atheroma did not show brain softening or shrinkage, while some cases without any atheroma manifested such changes.

White matter oedema was frequently reported in the post-mortems completed in Fair Mile but not in Sunnyside, where reports of oedema, while quite common, had no location stated, often being described as generalised. Cases with white matter oedema were also more likely to have atheroma in a vessel. The nature of the white matter oedema seen in these reports is elusive and therefore it is inappropriate to speculate on the mechanism of this association, particularly as, unlike the other post-mortem findings, there was less convergence between findings from the two asylums. The relative specificity of atheroma to these changes in grey and white matter contrasts with its lack of relationship with congestion which was reported in about 40% of these deaths: congestion was just as likely in cases with and without atheroma. This fits in with the widely held view that congestion is a vague, potentially unreliable finding. In this post-mortem series there were several cases (n=15) without any brain changes reported but there were a few more (n=17) with reports of just congestion. I speculate whether this latter finding was a consequence of doctors performing these examinations being under pressure or, more likely, feeling under pressure, to find and report something in cases incarcerated with insanity.

By the later part of the nineteenth century, most alienists took the view that insanity could not “exist...without a morbid change in...the brain” and most concurred with Gray’s view that post-mortems had the power to “answer all the speculations of materialistic philosophers.”

However, this view was not universally held. For example, Barfoot and Andrews both stress how David Skae, Physician Superintendent at the REA (1846-73), while being a strong advocate of post-mortem research on insanity and the first superintendent to institute separate post-mortem records, thought that the search for brain lesions was likely to be fruitless.\textsuperscript{405} Skae made much of the fact that many inmates who had exhibited marked “raving” insanity in life had no morbid changes in the brain after death. This view is partly echoed here as about one in six of the insane inmates were judged to have structurally normal brains at post-mortem.

As one part of their survey of the history of Colney Hatch Asylum in North London from 1850 to the twentieth century, Hunter and McAlpine analyse the changes reported in post-mortems performed in that institution. They comment that, overall, there was, as found here, apparently good use of anatomical and pathological terms. However, they also point out that the examination of the brain in the late Victorian era was often unrevealing and, in their opinion, unreliable: “Standards of abnormality in judging diseases of the brain were less precise. There was no refrigeration, no fixation, no staining methods and they had no microscope.” Despite these drawbacks, Hunter and McAlpine were also struck by the finding of “cerebral atheroma,” although the frequency of this finding is not reported from the Colney Hatch setting.\textsuperscript{406}

In this study, a palpable (if rather weak) association between vessel disease — or at least a marker of it (atheroma) — and cerebral softening was found. Therefore, the development of theories about cerebral softening and the possible role of disrupted blood supply in its genesis are explored. The term softening originated with Louis Rostan, a physician at the Salpêtrière, Paris, who, in 1820, claimed in his book \textit{Recherches sur le Ramollissement du Cerveau} that softening (“ramollissement”) in the brain was “the most frequent cerebral lesion.”\textsuperscript{407} In 1836, John Abercrombie (1780–1844), an Edinburgh physician with an interest in pathology, posited that cerebral softening was analogous to gangrene, that is that it followed blockage or “ossification” of an artery.\textsuperscript{408} In 1843, Maxine Durand-Fardel, a Parisian neurologist and pathologist, published his \textit{Treatise on Cerebral Softening}. This volume largely focused on the lacunae (small cavitary lesions)

\textsuperscript{405} Barfoot, “David Skae: Resident Asylum Physician,” 469-88.
\textsuperscript{406} Hunter and McAlpine, \textit{Psychiatry for the Poor}, 196.
\textsuperscript{408} John Abercrombie, \textit{Pathological and Practical Researches on Diseases of the Brain and the Spinal Cord} (Edinburgh: Waugh and Innes, 1828), 204.
seen in the softened brain that he speculated may be due to abnormalities in vessels.\textsuperscript{409} Rokitansky described cerebral softening in detail in his 1855 book \textit{A Manual of Pathological Anatomy}. Later, Rokitansky was a strong proponent of gross anatomical pathology; it is said that he performed over 30,000 autopsies during his 45-year career. Rokitansky divided cerebral softening (which he termed encephalomalacia) into three varieties: red softening (which he considered inflammatory in nature), white softening caused by congestion and oedema and, the most common variety, yellow softening, of which the origins were unknown\textsuperscript{410}. In 1856, Virchow opined that “yellow softening” of the brain was secondary to arterial obliteration and that inflammation was secondary.\textsuperscript{411} Gunning describes how others, most notably the Austrian pathologist Hans Chiari, drew attention to the frequency of atherosclerosis in the region of the great arteries and the suggestion that embolization of atheromatous material might be a cause of cerebral softening.\textsuperscript{412} In the current research there was a numerical link between atheroma in arteries and cerebral softening and thus this chimes in with the contemporary views of Abercrombie, Virchow and Durand-Fardel.

Although various types or stages of cerebral softening were described in the nineteenth and early twentieth centuries, there appears to have been no later systematic study of the phenomenon and the topic does not appear to have been the subject of much scrutiny by historians. In 1968, Sir Bernard Tomlinson, a neuropathologist from Newcastle, reported softening in a number of normal elderly brains and demonstrated that it can be a co-incidental finding at autopsy, though then usually confined to a small area.\textsuperscript{413} The cases identified here, however, were neither normal nor elderly and the finding, when present, seemed to be widespread suggesting the softening was of pathological import. However, it is salutary to note that the term softening of the brain was quite widespread in clinical parlance in the late nineteenth century, even to the point of its use as a clinical descriptor of mental symptoms. Indeed, three cases of insanity in Fair Mile who did not even have a verificatory post-mortem were ascribed cerebral softening as a cause of death. The eminent neurologist John Hughlings Jackson railed against this practice in a \textit{Lancet} article in 1875.

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\begin{itemize}
\item \textsuperscript{409} Maxine Durand-Fardel \textit{Traité du Ramollissement du Cerveau} (Paris: JB. Baillière, 1843).
\item \textsuperscript{410} Rokitansky, \textit{A Manual of Pathological Anatomy}.
\item \textsuperscript{411} Mayerl et al, “Atherosclerosis Research,” 101.
\item \textsuperscript{413} Bernard Tomlinson, Garry Blessed and Martin Roth, "Observations on the Brains of Non-demented Old People," \textit{Journal of the Neurological Sciences} 7 (1968): 331-56.
\end{itemize}
“the pathological term ‘softening’ has come... to be used, even by some medical men, as a name for a certain rude clinical grouping of symptoms in cases in which there really is no softening. This use of the term is to be deprecated.” By contrast, he considered that localised cerebral softening was associated with mental symptoms such as excitement or delirium including the transitory delusions after apoplexy by virtue of a loss of inhibition. He was adamant that the seat of the abnormalities that lead to softening was not the brain itself, but degenerated, usually atheromatous, vessels of all sizes in the brain or the heart.\footnote{Jackson, “A Lecture on Softening of the Brain,” 335, 336-39.} We can thus hypothesise that at the time alienists carried out the post-mortems studied here, the notion that mental symptoms might be associated with softening of the brain was so commonly held that a confounder of this study is that they may have been predisposed to search for and identify this ultimately subjective finding.

Turning now to critique the finding of cerebral congestion in these brains, it is important to do so in the light of the clinical lore prevalent at the time. Román traces the history of the term, although in a rather flat, uncritical account.\footnote{Gustavo Román, “Cerebral Congestion: A Vanished Disease,” \textit{Archives of Neurology} 44 (1987): 444.} Cerebral congestion was a commonly reported phenomenon in the seventeenth to nineteenth centuries. William Gowers (1845-1915), the pre-eminent British neurologist of the time, devoted ten pages of his influential, highly cited, 1887 textbook, \textit{A Manual of Diseases of the Nervous System}, to the topic of cerebral congestion and hyperaemia.\footnote{William Gowers, \textit{A Manual of Diseases of the Nervous System} (London: Churchill, 1886).} Román describes how Gowers and others made a causal link between congestion and apoplexy (an association which had been first proposed by Morgagni in 1761). Román additionally points out that influential physicians of the time, notably William Hammond, were of the view that cerebral congestion was "more common... than any other affection of the nervous system" and how it was responsible not just for apoplexy but for “depression, maniac outbursts, headaches, coma, and seizures.” Hammond was an army Surgeon- general and one of the founding fathers of the American Neurological Association in 1875, although one with an interesting back story.\footnote{Frank Freemon, “Lincoln finds a Surgeon General: William A. Hammond and the Transformation of the Union Army Medical Bureau,” \textit{Civil War History} 33 (1987): 5-21. Hammond was Surgeon General in the Army but was court-martialled in 1863 for purchasing irregularities. He was exonerated many years later. He had initially been admired and supported by Abraham Lincoln, but not during or after his court martial.} He published what some have considered to be the first modern textbook of neurology (\textit{Treatise on Disorders of the Nervous System}) in 1876. The first chapter of his treatise was devoted to cerebral congestion which he classified into two types, an active form
consequent upon an increase in cerebral arterial blood flow and a passive form secondary to venous congestion. According to Hammond, the clinical manifestations of the active cerebral congestion included “difficulty sleeping, restlessness, intellectual confusion, memory problems, emotional upset and occasional illusions, hallucinations and delusions.”

In his practice, Hammond had apparently seen 307 cases of active cerebral congestion but how many of them had the above complaints is not clear. He described four cases of paroxysmal psychomotor agitation and he remarked that “what is called temporary insanity, mania ephemera or impulsive insanity, depends on cerebral congestion.” Hammond reported that cerebral congestion was associated with several brain changes though he did concede that “occasionally some or all of them were absent.” Hammond was clearly convinced by his own case because in 1878 he published another book, Cerebral Hyperæmia which promulgated these ideas and indeed went further claiming that cerebral congestion was the result of “mental strain/ excessive brainwork,” or “emotional disturbance/ the passional (sic) excitement so often produced in men and women.”

Román stresses, in a rare critical moment, that all sorts of conditions, most often chronic depression or situational stress, were concertinaed into those Hammond’s theories were based on. Román additionally comments that cerebral congestion “vanished” when an accurate method for bedside determination of blood pressure, the sphygmomanometer, became available from 1905. This is not accurate, however, since cerebral congestion was never a disease, and its “disappearance” was a consequence of its highly subjective nature. The historian Blustein goes significantly further in pouring cold water on the claims of Gowers, Hammond and others in a critical article on cerebral hyperaemia and its “brief career,” denigrating Hammond’s book on cerebral hyperaemia as “self-puffery.” She pointedly comments that Hammond saw hundreds of patients who had a myriad of minor complaints in his “well-appointed offices,” and readily diagnosed cerebral congestion and/or hyperaemia in one after another. She describes how Edward Seguin, an equally distinguished neurologist and an (alleged) friend of Hammond, said, in 1889, that cerebral hyperaemia was a condition “known through a feat of the imagination” and that the “creation of the “disease” was mere theorizing of an ambitious specialist.”


\[419\] Hammond, Cerebral Hyperæmia, 6.

How these debates about cerebral hyperaemia/congestion, and their alleged clinical effects, impinged on the medical officers performing post-mortems in semi-rural asylums in Britain a decade later is uncertain. As Worboys observes, there were significant shifts in medical ideas and practices over the decades but “the balance of continuities and changes” was uneven. These alienists are likely to have been taught about Gowers’s work and to be influenced by his well-read textbook. The condition did not “vanish” (to use Román’s inappropriate term) in Britain or elsewhere until the later 1910s and 1920s (after the period of this study). Congestion as a sole pathology was reported in this series in 17 patients but was more usually accompanied by other grey and white matter changes. It is probable, therefore, that most of the documented congestion was a non-specific finding, or one associated with an otherwise damaged brain. The highly subjective nature of congestion and its lack of clear-cut boundaries does raise the possibility that these alienists were looking for something, perhaps anything, in the brains of what had been highly disturbed inmates. However, there is also the possibility that undetected elevated blood pressure may have accounted for the observed congestive changes in at least some of the patients in this study. Changes in the white matter of the brain were frequently seen in in these asylum post-mortems. In their account of post-mortems performed at Colney Hatch Asylum in a similar period, Hunter and McAlpine report an unspecified number of reports of “état criblé,” characterised by “opacity of membranes with serous effusions, the white substance extensively cribiform, as if moth eaten ... vessels changed.” Etat criblé was a term first coined by Durand-Fardel in 1842 to describe the “cribiform state” (often described as “moth-eaten”) of brain white matter. According to early neuropathologists, it was associated with small cerebral softenings in the cortex and atrophy of the white matter because of chronic cerebral congestion and disrupted blood flow. Hunter and McAlpine highlight that such brain changes were often associated with hypertrophy of the left ventricle of the heart. Etat criblé was not fully described in the cases studied here but several patients were noted to have punctate lesions or “punctate vasculosa” and these were probably the terms the medical officers in these asylums used to describe what pathologists called état criblé. In this series, there were several cases, in both the mania and melancholia cohorts, with hypertrophy of the left ventricle, softening and shrinkage of grey matter and swelling, with punctate lesions, of white matter, cerebral congestion and atheroma in vessels.
both of brain and/or heart. With the advent of blood pressure measurements, état criblé became known to be associated with hypertension. Thus, it can be speculated that a component of the changes observed in the brain in this cohort may have been a result of elevated blood pressure that, of course in the period under study here, would have gone completely undetected. However, high blood pressure is unlikely to account for all the changes seen in these brains and other mechanisms are both plausible and likely. Virchow, and subsequently other nineteenth-century pathologists, conjectured that these brain changes may have been part of an inflammatory process, perhaps a consequence of frequent and unrestrained infections. This is another plausible mechanism for the patients studied here as recurrent infections were common in these sufferers. These topics will be further explored in Chapter 5 which examines vascular pathology and vascular causes of death, and their underlying narratives, in greater detail.

3.2.9 Post-mortem findings in individual cases

Several cases from this sample were examined in more detail. These include deaths from exhaustion, which are described in Chapter 4, and from vascular causes, which are discussed in Chapter 5. Six sudden deaths are discussed further here – two cases where ante-mortem violence may have played a part in the death and two cases in whom “choking” was stated as the cause of death. These cases are included because they throw light on the question of whether post-mortems in asylums had utility and contributed to patient care. Two cases who died by their own hand are described in Appendix F.

GH, who was admitted in December 1904, was a 27-year-old single schoolteacher in her first attack of mania of two weeks duration. Her father, also a schoolmaster, said that she was normally in good health and was intelligent and temperate but that she had changed completely since a trivial boating accident. She had become over-anxious in preparing for an examination. She became deluded that the doctor who was called to see her was someone else. On admission, she was restless. She said she had only one foot and that she had come to the asylum by motor from India. That night, she became very restless and excited and was talking as if she was in a school

exam. She became resistive and a struggle with nurse H ensued. The patient snatched the nurse’s cap off and pulled her hair. GH was taken to the lavatory, but fifteen minutes later, apparently had a “fit.” A doctor was summoned who also called Dr Murdoch, the Physician Superintendent. Respiration had ceased. Artificial respiration was attempted, and strychnine given, without success.

However, subsequent entries in the General Statement Book, a monthly account of matters in the asylum, tell a different story. GH had apparently previously attacked Nurse H and when GH became resistant again, Nurse H lost her temper, shook the patient by the hair and knocked her down onto the tiled floor of the bathroom. Almost immediately, the patient died. There were two other nurses present who initially denied that there had been any violence but later confessed the details. The post-mortem revealed there was extensive haemorrhage in the scalp and “on both sides” of the brain but no fractured skull. The post-mortem report and the detail of the brain findings are reproduced in Appendix A (Case 3). The Verdict of Coroners Jury was that the cause of death was “Cerebral haemorrhage” and that the patient died from effects of excitement causing a stroke and not the violence. As the full story emerged, unease increased. The General Statement Book recorded that “it will be the duty of the Visitors to consider prosecuting the nurse” and the details of the case were forwarded to Chairman of the LC. An entry a few months later states that several of the nurses have been unnerved by recent “painful events” which may well be a reference to this incident, though this is unclear. Tellingly, however, these events were followed (so far as I can find) by a complete silence and no reference is made to the subject in subsequent reports. It seems incredible that this young previously healthy patient had a cerebral haemorrhage just at this juncture (particularly as haemorrhage was on both sides of the brain and the vessels were apparently healthy). Bleeding into the brain following trauma seems an almost certain cause of death. No decisive action appears to have been taken, by the coroner, the asylum authorities or the LC. The post-mortem did its job in detecting the pattern of bleeding in the brain: the problem was in the interpretation of these findings which has more than a flavour of a cover up.

EP was a 47-year-old single charwoman who was admitted in 1904 and died 10 days later. She was in her first attack of melancholia which had started suddenly a fortnight previously. She was described as intelligent and temperate but had lived in poor circumstances for some time with insufficient diet. She had been in the Union workhouse for one week prior to admission where she had been spiteful and had refused food. On admission, she had delusions that her food was poisoned and that there were people walking on ceiling. She was poorly nourished and had weak
musculature. She had a melancholic expression and kept repeating over and over, “Oh God, I never did.” She was restless with poor sleep but was given sulphononal which helped. On the morning of her death, she suddenly became excited and talked rapidly, and was then given breakfast and put on commode. After a few minutes, EP appeared to faint. A doctor was called but the patient expired prior to his arrival. The post-mortem revealed heart disease and a large subdural haemorrhage. The latter is only found after trauma to the head that must have occurred at the start of her illness, in the workhouse, in the asylum or (least likely) after falling off the commode. There was no mention of anything untoward in her notes and no injuries reported in any of her documentation. The coroner was not informed, and the cause of death was ascribed as “heart disease.” Once more, the post-mortem did its business in detecting a violent cause of death that, at the very least, flagged up the need for closer scrutiny but again, seemingly, the fault lay in the interpretation of the findings and in subsequent inaction.

FD was a 33-year-old single lady’s maid. She was admitted in November 1902 having been transferred from Wandsworth Asylum. She had been admitted there seven years previously following her first attack of mania. Her family came from the vicinity of Fair Mile asylum and had lobbied for her transfer. When she had been admitted to Wandsworth, she had exhibited clear-cut persecutory delusions and felt that everyone was working against her and that “they” were breathing in her face to stop her breathing and sleeping. On transfer, she exhibited disordered thinking and thought that the admitting doctor had met her in Uxbridge seven years ago and had “ill-used her.” During the next few weeks, she was described as voluble and incoherent, and she continued to express persecutory delusions. Suddenly, one morning, she choked on a piece of bread. The doctor was called who found her moribund. He tried to manually remove bread that was “crammed in her mouth” and he also passed a tube and tried a tracheostomy. She was pronounced dead. The post-mortem revealed bread the size of a “cob-nut” in her trachea above the vocal cords, but no other abnormality was seen. The post-mortem cause of death was given as “Suffocation from impacted bread in larynx.” An inquest was held later that day and “no blame attached” according to the General Statement book. This case had raised alarms of abuse or neglect by the attendants, but the history and the post-mortem convinced the coroner that self-choking was the mechanism of death. In the end, FD’s greatest fear of stopping breathing came true but not, it seems, because of her feared other people but as a result of her abnormal mental state leading to abnormal food intake, allied with a probable disordered pattern of respiration.
AY was a married housewife and mother of four admitted in 1901 with her first attack of mania with evidence of both persecutory delusions and auditory hallucinations. Initially she thought it was her neighbour who was watching her and stealing her food but during her nearly 2 year stay in the asylum came to believe that everybody was trying to poison her food in order to kill her. She was known to have the signs and symptoms of heart disease (probably of her valves). One morning while having breakfast she had a syncope (faint) and died despite attempts at artificial respiration. She was found to have a “lot of food in her mouth” and initially choking was given as the cause of death. The post-mortem revealed food stuck in the trachea but also extensive heart disease and softening and shrinking of the brain. The verdict of coroner was of “Syncope from heart disease.” Here it was not the food she so feared that killed her but more likely than not her severe physical condition. The post-mortem, as in the other case of choking, was a helpful redress to an incident that would otherwise have been labelled as non-accidental, with the likely implication of staff mismanagement.

3.3 Conclusions

This chapter has traced the growth of the performance of post-mortems in British asylums and the factors which underpinned that development. There are a number of narratives which bear on the question of why post-mortems were performed in asylums at the turn of the nineteenth century on such an industrial scale. The first is the notion that the supervising authorities saw post-mortems as a method, justifiable in medical terms, to identify and, more crucially, prevent violence and non-accidental injury in asylums. We have to recall that at the time post-mortems were effectively mandated, there was still a lot of abuse going on in institutions, the majority of which were publicly funded and that many issues (including the genesis of fractured ribs in the insane) were unresolved. Reports of abuse and scandal lessened across the nineteenth century but whether post-mortem practice had any significant part to play in this reduction seems speculative. Burney and Cullen have drawn attention to asylum medical officers’ performance of post-mortems mitigating against them being impartial assessments.426 “Gentlemanly agreements” between asylum staff and coroners means that, the impact of this layer of public protection may

426 Burney, Bodies of Evidence; Cullen, “Post-mortem in the Victorian Asylum,” 284.
have been minimised although the extent of this is difficult to assess. There may well have been similar unspoken accords between medical officers and asylum attendants so that those performing post-mortems reported here were disinclined to find changes that may have stemmed from abuse or neglect by their staff. However, the fact that a post-mortem was effectively routine may have acted as a preventative measure, additional to other processes (for example the monitoring of restraint) which were carried out to reduce violence and encourage moral management stratagems. Post-mortems had an aura about them that they could reveal the truth and this idea was likely to be held by the attendants and may have given them pause for thought. This pretty standard notion was aired by the alienist Adam, in 1884, as one of the main reasons to do post-mortems in asylums: “The practice of making post-mortem examinations in asylums deters attendants and others from ill-using patients, as injuries inflicted by them are certain to be detected, and it thus proves a safeguard and protection to patients.”

The post-mortems appeared to have been carried out with care and with attention to detail but what stands out is that even though signs of violence were identified in the post-mortems of two cases, no formal outside action ultimately resulted from those findings. The coroner at Fair Mile seems to have been particularly quick to “wave” cases through and to rubber-stamp ascribed causes of death. So, while the post-mortem itself may have been conducted with care and diligence and an open enquiring mind, the processes thereafter for independent scrutiny seem to have been deficient. Thus, there is debatable evidence that post-mortem played a part in reducing asylum abuse and violence but much points to any effect being minimal. The improved situation in the later part of the century more probably followed from generalised improved standards of care along with other methods of scrutiny. Another objective of the asylum post-mortem was that they acted as, in Andrews’s words, “insurance against lawsuits” combatting allegations of staff brutality.

Against this yardstick, the routine post-mortem seems at least to have achieved its aim and the performance of the post-mortem usefully reduced potential opprobrium for the staff in the two cases of choking.

An additional principle underpinning the pressure to do post-mortems was to uncover the cause of death and identify physical conditions that were linked to insanity. In this endeavour the post-mortem reports provide a detailed account of pathology, and the findings seem, for the most part,

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to be consonant with the ascribed cause of death. However, there are a number of caveats to this positive statement. There continued to be ample use of vague terms, particularly when describing brain changes. Attention was drawn above to the finding of congestion in the brain which in several cases was the sole finding. This term was in popular use and was promoted by some influential practitioners and it is hard to avoid the suspicion that congestion, a term with no clear limits, was found because of a preconditioned search for something in the brain of a highly disturbed individual. However even congestion was not ubiquitous, and two out of three cases did not exhibit it. The causes of death following the post-mortems seemed accurate but, as other commentators point out, were very homogenous and no unusual findings were made which is out of step with most post-mortem practice.429 It seems that the medical officers were happy to pigeonhole cases into accepted causes of death. Another finding is that there was very little difference between cases who had and did not have a post-mortem. On the one hand, this finding could be an indicator that the regular practice of post-mortems was useful in the sense that it improved cause of death reporting in similar cases who did not have post-mortems but, on the other hand, this finding could point to the relative futility of the complex and laborious procedure itself.

Finally, another driver, arguably the most prominent, for carrying out post-mortems was to provide and more widely disseminate material and data for research into insanity and for advances in its treatment. Here it is clear that these complex, labour intensive and potentially distressing procedures in these non-academic settings contributed little to the understanding of processes underlying insanity and how to treat them more effectively. There is no evidence that the findings of the 172 post-mortems studied here were ever part of a publication or similar public output other than a footnote in the LC’s Annual Report. Valuable findings from post-mortems in asylums in this late Victorian period were made but they emanated from study of a small number of cases in specialist centres, usually involving a pathological expert. Of course, this negative outcome must be seen in the context that even pathological experts struggled with finding anything consistent in insanity or later in, as it was dubbed, psychoses. This situation essentially pertained for the next seven decades leading to psychiatry being dubbed “the graveyard of Pathology” by Plum in 1972.430 More recently using complex refined techniques and a great

attention to detail and controls, consistent findings in the brain in psychoses have been found, even though modern neuro-imaging is seen by many as a more fruitful pursuit than neuropathology. Thus, the massive amount of work in the asylum post-mortem, for example in weighing the organs or carefully dissecting and examining the meninges, led to very little. A saving grace is that some of the medical officers and some of the relatives may well have come away from the process with the hopeful idea that something was being done. As Wallis observes, “though unsuccessful, the process of investigation could nevertheless lead to practical and theoretical developments that were important to contemporary observers.”

It is also true to say that if no-one looked, no-one would have found anything.

So, it can be concluded overall from the findings of this study into asylum post-mortems that they were carried out because they were mandated but served limited purposes and that they illuminated little. There is no evidence the LC relented in its demands for post-mortems until effectively forced to by the advent of the First World War. There may have been an adequate rationale for post-mortems earlier in the nineteenth century, but it is questionable whether their routine performance continued to serve much purpose in the late Victorian or early Edwardian periods. Once initiated, one surmises that the Commissioners found it hard to reflect and put limits on the process or perhaps they were afraid to even contemplate that? Andrews and Hervey emphasize that the Scottish and English Commissioners respectively were imbued with an essential conservatism, an attachment to red tape and legalism and, perhaps even more importantly, tended to take views that were self-justifying – all factors that mitigated against them opting out of a practice once established.

This leaves the question of whether the material from a post-mortem series like this provides some insights for a medical historian. I argue that it does. Here it useful to recall the findings of Chapter 2 which showed that people with mania and melancholia in this study were dying, on average, 15 years younger than the general population, an observation which requires explication. The information on the frequency of deaths from infections is likely to be reasonably accurate and informative and provides important information towards understanding this mortality gap as does the lack of evidence for the role of malnutrition in causing these deaths shown here. However, the novel findings of vessel changes in a young insane population and the linking of those changes to

431 Wallis, Investigating the Body, 182.
pathology in the brain provides information which may help to place some of the premature mortality of these forms of insanity into a different context. However, there are historians who are critical of those interpreting pathological findings too directly. For example, Alan Blum stresses that making inferences about causality from the presence of noted irregularities is a speculation after the fact about dysfunctions. He argues that there can be no demonstration that an irregularity causes the bizarre behaviour since there can be no controlled observation of onset. Notwithstanding this critique, it can reasonably be suggested that the current findings show that while early death was linked to a liability to severe, often fatal, infections, additional pathology in the heart and vessels was often present and were associated with brain pathology. These findings implicate a combination of infections, stresses and deprivation as risk factors for morbidity and mortality and raise the suggestion of mediation by an inflammatory process. These ideas will be developed in succeeding chapters starting with the specifics of deaths linked to exhaustion and vascular disease in Chapters 4 and 5.

Chapter 4. Death by Exhaustion in Asylums

4.1 Introduction

Exhaustion was a common symptom and/or a diagnostic condition ascribed to asylum patients by Victorian alienists and was often linked with issues such as poverty, destitution, malnourishment, and stress. The condition was generally thought to be preceded by marked excitement, often associated with delirium and confusion. Contemporary clinical discourse on exhaustion was underpinned by the widely held concepts of degeneration and toxaemia. Exhaustion was usually associated with a poor outcome and high mortality. The data analysed here is contextualised against the leading contemporary psychiatric literature on the topic and investigates the frequency, coherence, and validity of the label, more especially as a cause of death. A key aim of this chapter is to assess the key factors associated with deaths by, with or from exhaustion in late Victorian and early Edwardian asylums, and to explore whether the environmental conditions of asylums and their ethos and treatment (including chemical or non-chemical restraint) contributed to these deaths.

Nineteenth-century alienists consistently reported high death rates amongst the acutely insane, particularly amongst those whose condition had features of confusion, delirium, and exhaustion. Some commentators propose Louis-Florentin Calmeil (1798 – 1895), the French alienist and medical historian, as the first, in 1832, to describe the association of insanity and death from exhaustion, but Andrews points out that alienists had frequently identified exhaustion from maniacal excitement as a leading cause of asylum mortality from 1800 onwards. John Haslam gave examples of death from exhaustion following the excited behaviour of mania and the self-starvation of melancholia in 1809. George Man Burrows wrote about how in melancholia there could be “a sudden” and rapidly mortal “prostration of strength” concluding, after post-mortems failed to reveal “any assignable cause”, that death was due to the “simple effect of exhaustion”

conjoined with “raving and excitement.” Alienists continued to highlight exhaustion as a major source of asylum mortality in the decades that followed. In his influential treatise of 1835, James Pritchard drew particular attention to the mortal dangers of mania and excitement, warning that exhaustion and “a fatal termination” could follow. In one of the earliest and largest published studies associating mania with sudden death, Luther Bell, an American alienist from the McLean Asylum, Boston, reported on 40 cases of mania with exhaustion admitted during 1836-49. A patient suffering from (what was later dubbed by Kraines) “Bell’s mania” was noted as getting “so little food, so little sleep” and “be exercised with such constant restlessness and anxiety, that he will fall off from day to day... At the expiration of two or three weeks, your patient will sink into death.” Although several of the cases in Bell’s report recovered completely, the majority had a fatal outcome. Bell attributed this illness to inflammation of the brain and meninges and to “passive congestion” of the cerebral circulation yet found no evidence of any of these conditions on autopsy. He concluded that those experiencing similar symptoms in mania would be at great risk of sudden death and that, perhaps, those suffering from this condition had an illness distinct from any previously defined. While asylums achieved success in large part in the prevention of suicide by all those except the most determined, asylum staff felt substantially powerless and therefore often nihilistic in the face of florid maniacal excitement, and similarly in profound melancholia, as they were unable to prevent their relatively frequent fatal outcome. Cases of death by exhaustion in acute insanity continued to be regularly reported by asylum medical officers through the nineteenth into the early twentieth centuries. For example, Cecil Beadles, Assistant Medical Officer at Colney Hatch, stated that of 1000 deaths in that asylum from 1880-96: “By far the greater proportion... were due to the exhaustion of one or other of the various forms of insanity.”

Despite the extensive contemporary clinical attention to this topic, the epidemiology of exhaustion as a cause of death in asylums has been relatively little discussed by historians. The main explanation for this appears to be that the study of psychiatric mortality patterns remains underdeveloped as a field in general, by comparison with the history of institutional care,

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436 Burrows, Commentaries, 554.
437 Pritchard, A Treatise on Insanity, 114.
treatment, architecture and personnel. Historians have also grappled with the fact that the issue is complex and subject to many vagaries, including shifting nosology, changing patterns of death certification and shifts in the asylum’s place within society. A minority of scholars, while acknowledging these difficulties, have analysed death by exhaustion as a percentage of total deaths in various asylum settings, but their estimates of the frequency of such deaths vary, as do their views on the temporal trends of this phenomenon. Leonard Smith found many such deaths in the early decades of the century in the records of Stafford Asylum, including three cases in one week in 1827. Cathy Smith examined records from Northampton General Asylum over a 30-year period from 1846 and concluded that only general paralysis and general debility killed more than exhaustion. The balance of evidence from both primary sources and secondary studies suggests that exhaustion was regarded as a commonplace cause of asylum deaths in the earlier part of the century. For example, Andrews concludes that exhaustion comprised a “significant share” of mortality statistics from both British and European asylums during the period 1800 to 1860. In some localities and periods, the death rate from exhaustion was even higher, including the 1850s peak analysed by Catherine Cox and colleagues in Liverpool. In this case, there were unusual circumstances: mortality patterns were evidently impacted by the post-famine influx of Irish immigrants and their subsequent precarious position in England. Later studies of the Victorian era show smaller rates of these deaths. David Wright’s analysis of deaths in four asylums in Ontario across the period 1841-75 found the average death rate from exhaustion was 7%. Wright conceded several caveats to this dataset which need to be borne in mind generally for the majority of asylum mortality data, including the significant proportion of cases (about 20%) with no recorded cause of death and the imprecise and subjective nature of cause of death attribution. Edward Renvoize and Allan Beveridge carried out an analysis of causes of death at three Yorkshire asylums in the period 1880-84, which attempts to make retrospective diagnoses (at least into broad groupings). In the case of exhaustion as a cause of death, these scholars sought to separate cases where exhaustion was the sole cause of death from those where it was given as the outcome of some physical disorder, such as epilepsy or apoplexy. The rates of death from exhaustion from mania and melancholia were significantly (and somewhat surprisingly) higher at

441 Smith, “Visitation by God,” 104-16.
the Retreat (14%) than at York and North Riding asylums (9%) but the authors were unable to provide a satisfactory theory to explain the overall and differential rates. Healy’s research team find that about 15% of the deaths in melancholia and mania in a late Victorian asylum in Wales could be attributed to exhaustion. It appears therefore that death by exhaustion became a less non-specific and perhaps less common, though not unusual, descriptor of deaths in the later nineteenth and early twentieth century period. This contention is tested here by examining the use of this and related terms across a 35-year late Victorian and early Edwardian period.

In their consideration of exhaustion as a cause of asylum deaths, many British and international scholars interpret its usage in the earlier parts of the nineteenth century as vague and imprecise. Wright argues that exhaustion was a “general category” akin to “debility” and “senility,” designating exhaustion as “that ubiquitous [my italics] Victorian cause of death.” Similarly, Steven Cherry observes that “deaths were often casually [my italics] attributed to old age, general decay, general paralysis or “exhaustion after mania.” In his Ontario Asylum study, Samuel Shortt concludes that such causes of death in asylums were “vague and imprecise” even by nineteenth-century standards and suggests that this vagueness may have been deliberate to aid doctors escaping censure for culpability in these deaths. These commentators echo the views of influential alienists of the period. For example, John Bucknill derided his colleagues in 1862 for their frequent use of exhaustion as a cause of death along with the equally imprecise terms prostration and decay. Bucknill appealed for a separation between the cause of death (which he saw as the mental illness itself) and the mode of death, emphatically advocating the cessation of the term exhaustion as a cause of death. A year later, Thomas Clouston similarly reproached asylums for using imprecise terminology such as exhaustion and decay. The interpretations regarding the ill-defined nature of deaths by exhaustion offered by the historians quoted above and the primary reports on which they are based are centred on the mid-nineteenth century. No such comments are evident for the later period around the turn of the century. The key issues are whether this change reflects a real reduction in the proportion and incidence of such cases for

445 Renvoize and Beveridge, “Mental Illness and the Late Victorians,” 19-28.
448 Cherry, Mental Health Care in Modern England, 72.
449 Shortt, Victorian Lunacy, 61, 104.
whatever reason, a decreasing interest in the topic, or a more precise, less heterogeneous, use of this term as a consequence of the strictures of these influential alienists. This thesis strives to arrive at a set of evidentially robust conclusions about these competing interpretations.

Anna Schaffner recently took a broad historical and cultural approach to the topic of exhaustion, arguing that exhaustion can be “understood not only as an individual physical, mental or spiritual state but also as a broader cultural phenomenon.”452 She stresses how theories concerning exhaustion have moved back and forth between physical and mental models, which in turn resulted in many different suggestions for its cure “from bloodletting and emetics, through tonics, stimulants, rest cures and exercise therapy, to electrotherapy, psychotherapy and drugs.”453 Germán Berrios also usefully charts the historical ebbs and flows of the labels fatigue and exhaustion.454 Berrios comments that both terms have been used, often interchangeably, to denote disparate aspects of the mental state and different underlying concepts. He quotes Angelo Mosso, the nineteenth-century Italian physiologist and neurologist, on the problems of definition: “Fatigue is too featureless an internal sensation to distinguish any varieties.” Mosso talked of the “vagueness and inaccessibility of fatigue” and observed that a distinction needed to be drawn between objective and subjective exhaustion and between fatigue and weariness.455 Some Victorian alienists defined fatigue as a process and others as the effect itself: Shorter describes these discourses in detail in his survey of the topic.456 A debate ensued in the nineteenth century as to whether fatigue was a primary experience or secondary to perception of peripheral somatic changes. In overviewing this topic, the French physician Pierre Bugard considered these ideas irreconcilable and advocated a middle way in which fatigue was the magnification of peripheral sensations. He concluded that there was general agreement only with the notion that fatigue was a state of “nervous suffering which, together with insomnia, constitute the prodromal phase of many mental diseases.”457

Degeneracy theory was a prominent notion amongst alienists of the late Victorian period, and its relationship with the concept of exhaustion needs elucidating. Degeneration was commonly

453 Ibid, 7, 12.
455 Angelo Mosso, La Fatica (Treves: Fratelli, 1891).
posited to underpin many conditions including neurasthenia, fatigue and insanity as well as exhaustion. Exhaustion was also sometimes used as an equivalent of neurasthenia in writings of the time, but the exhaustion reported in asylum practice was a qualitatively different condition from the neurasthenia observed in the clinics, although some common features were considered to underlie both neurasthenia and exhaustion in mental illness. George Beard, the American neurologist who developed, defined and popularised the concept of neurasthenia, believed that inheritance of a “nervous diathesis” underpinned neurasthenia and that the theory of degeneration “provided a context in which neurasthenia found a natural and immediate place.”

Charles Féré (1852-1907), an illustrious French physician, also grounded proneness to fatigue within the theory of degeneration and described how fatigue and exhaustion after prolonged exercise could in themselves lead to psychosis. These ideas are reflected in Schaffer’s description of exhaustion as a “vampiric depletion or harmful consumption of a limited (and usually non-renewable) resource.” Many contemporary alienists and neurologists were of the view that there were strong links between neurasthenia, insanity and exhaustion, although these views appear to be based on opinion rather than evidence. Foster (a physician in the US Government Hospital for the Insane) in an 1899 address asserted categorically that “Insanity is but an advanced and grave form of neurasthenia in which the cerebral involvement predominates.” More pertinently, his contention was that neurasthenia, with its severe insomnia, was a forerunner of insanity and exhaustion in the predisposed. Clifford Albutt, Regius Professor of Physic at Cambridge, and a strong proponent of the neurasthenia concept, similarly stressed that a neurasthenic was one “who never had much reserve... who needs inordinate time for repair... and may be exhausted beyond the possibility of repair.” Albutt claimed that there was an association between neurasthenia and insanity as individuals of “nervous stock” were “apt to warp in more than one plane.”

Historians observe that toxaemia was another explanatory narrative for the mechanisms of exhaustion deaths frequent in Victorian asylums. There was a widely held view in clinical scientific

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460 Schaffner, Exhaustion, 7.
circles in the late nineteenth century that toxins of various kinds were responsible for mental symptoms such as acute excitement and delirious mania. For example, Clouston postulated in 1883 that “imperfectly purified blood” could cause both acute and permanent damage to the brain, although later, in 1904, he suggested caution in adopting toxic explanations for insanity too readily, indicating that its wholesale adoption was a fashion.463 Before the turn of the century, Emil Kraepelin, the famous German alienist best known for his contributions to the nosology of insanity, was a powerful advocate of toxic and metabolic theories of insanity but later became what Noll calls “agnostic” as to a toxic causation. Noll traces the evolution of such theories and the changes in blood which were purported to account for severe mental illness in the late nineteenth century and how theories of “corpuscular richness” and then metabolic theories (and their derivative the auto-intoxication theory) consecutively held sway.464 There has been little attention to the issue of whether the concepts of neurasthenia, degeneracy and toxaemia impacted on the labelling of exhaustion and associated mortality in Victorian asylums. It seems probable that these ideas percolated into the asylums studied here as terms like wasted and degenerated abounded in the descriptions of post-mortem tissue.

Two main scholarly narratives have influenced our understanding of the label of exhaustion in Victorian asylums: i) that exhaustion led to and was a cause of insanity, and ii) that conversely insanity, or at least its behavioural components (the over-activity of mania or the agitation or stupor of melancholia), led to exhaustion. Smith shows how deaths from exhaustion were of two broad types which corresponded to these narratives: those chronic cases of insanity admitted in putatively exhausted states after being unwell for a significant period of time, typically accompanied by marked weight loss, whose mental condition was further worsened by incarceration; and those whose insanity was acute and rapidly advancing, so that death from exhaustion appeared to be the direct consequence of their mental disorder. The former group was numerically greater, but Smith charts the increasing emphasis by alienists on the importance of

463 Clouston, Clinical Lectures on Mental Diseases, 1883, 596 and 1904, 111.
the latter group. Chapter 2 of this thesis, a comprehensive study of such deaths in a reasonably representative asylum, showed that these narratives have basic statistical validity.

Laurence Ray argues that the “stress model” of insanity, which postulated that events and circumstances, often called “moral” causes, were the forerunners of both insanity and exhaustion, held sway in the nineteenth century. He highlights the work of John Charles Bucknill and Daniel Hack Tuke in leading the way in promulgating these views: they indicated that “exciting causes” in the lead up to insanity were commonplace and included overwork, over-study and marked muscular exertion, as well as other emotional stressors in this category.

These precipitants, when marked, were seen as important forerunners of exhaustion, particularly in those with a hereditary predisposition. Changed circumstances, particularly those leading to poverty or family breakup (such as separation or death) were routinely looked for on admission and were commonly found. Modern scholarship on asylums also highlights the prominence of stress factors in the moral causes ascribed to admissions. Franklin Klaf and John Hamilton’s study of mid-1850s admissions to the Bethlem Royal Hospital shows that, in about a quarter of male admissions, overwork or religious excitements were attributed as “exciting causes.” Renvoize and Beveridge provide evidence to show that, in rural Yorkshire asylums, business anxieties, pecuniary difficulties, overwork and domestic trouble (including bereavement, disappointment in love) and fright accounted for about a fifth of the alleged moral causes of insanity, with even higher figures at the Retreat in York with its different clientele and ethos. They point out that from at least the mid-nineteenth century, the importance of early admission to an asylum and thereby seclusion from outside stressors and responsibilities was viewed positively by many alienists. However, asylum care for patients with so-called “moral” causes failed to reliably protect these sufferers from exhaustion associated mortality post-admission. Far from being transitory and amenable to early recovery, cases admitted with the “moral” causes label were just as likely to die, and to die from exhaustion, as groups without such precipitants.

Furthermore, Melling’s study of Exminster Asylum in the 1880s shows that only 2% of male admissions were ascribed to “overwork/over

study” and comparable figures can be obtained from different locations and times. So, while Ray’s stress model is widely supported and has explanatory credence in understanding the origins of insanity, these underpinnings were not pervasive and therefore stress and/or physical or mental over-exertion by themselves seem unlikely to account directly for deaths from exhaustion.469

A related perspective, that death from exhaustion commonly followed a severe stressor/trauma or “fright”, appears, however, to have more traction. Death related to fright has a long history in culture and medicine which has been comprehensively surveyed by George Engel, the American pathologist and psychiatrist, who sees such deaths as a challenge to the biomedical model of insanity.470 Atchison Robertson, a Scottish public health physician and Fellow of the Royal Society of Edinburgh, put together reports from Victorian alienists and summarised the view that this type of death occurred because of “inhibition” induced by intense emotional disturbances, or “emotional and psychic shock.”471 He collated cases of sudden death which seemed to have been induced by emotions, particularly fear and anger but also joy. Post-mortem examination of these cases provided no meaningful counter-narrative. Several commentators observe that the insane suffering from the more acute and severe illnesses had often experienced sudden severe emotional shocks at the onset of their conditions and that such cases often died with symptoms of exhaustion.472 Donald Gregg who practised as a psychiatrist and neurologist from 1907 to 1939 and was, latterly, President of the Massachusetts Society for Mental Hygiene, postulated that “the greater the emotional element involved... the greater the relative incidence of death.”473 There is however a dearth of research supporting this theory as the primary underpinning of death by exhaustion in asylums other than some anecdotal evidence. These concepts were examined in the current sample, but antecedent acute shocks were found to be rare and lacking specificity.

I now turn to the second key narrative about exhaustion in insanity, namely that the latter caused the former and to the clinical issues and associations addressed in this research. Nineteenth-century asylum case reports record frequent and startling accounts of mania with furious over-

activity, marked restlessness and violent behaviour. Mania was regarded as dangerous and difficult to manage carrying with it the risk of exhaustion. Melancholia too was frequently dreaded for its slow and often undetected onset, so that by the time it was spotted it had commonly become entrenched and difficult to cure, its sufferers worn down. In their study of reports from Yorkshire asylums, Renvoize and Beveridge show that the exhausted patients had previously exhibited very marked motor behaviour. They suggest, in line with Klaf and Hamilton’s work, that severe agitation and restlessness were pronounced components of insanity in the nineteenth century, with exhaustion as a consequence in a significant number of patients. Historians have highlighted that exhaustion was a common feature in those whose insanity was coupled with physical weakness, either as a result of pre-existing hardship or inter-current disease, or as part of a predisposition. In their account of admissions to Lancashire asylums, Catherine Cox and Hilary Marland report that in 1856, five years after Rainhill Asylum opened, its Superintendent, Dr John Cleaton, observed that 50% of all admissions were in an “exhausted state of health.” Cleaton commented that recovery rates were lower and death rates much higher amongst patients “shattered in bodily health and condition.” At that time, a high preponderance of such cases were Irish migrants. Cox and colleagues emphasise the large numbers of Irish migrants admitted to Lancashire asylums in the late nineteenth century, many arriving in poor physical condition because of hardship, unemployment, poverty and factors associated with poor diet and malnutrition. Over half of all patients were described as “reduced and exhausted” with their exhaustion attributed to socio-economic hardship issues exacerbated by factors such as intemperance and the consequences of unchecked mania. The death rate of this group was very high (over 50%). Yet, despite these very clear risk factors for both insanity and for exhaustion, Cox and colleagues show how such factors were exaggerated as a result of negative ideological perceptions abounding amongst the public and asylum staff alike, including notions of degeneracy and racial stereotyping (for example, seeing the Irish as more prone to mental and constitutional weakness).

Cathy Smith outlines the special demographic character of the group with insanity and exhaustion in the Victorian asylum. They were more likely to be single and have fewer family members and

other resources to draw on. This was especially striking for men but also evident amongst female admissions, where widowhood was common. Smith additionally underlines the social factors associated with exhaustion in asylum inmates – age, gender, adverse circumstances and, most notably, the late and moribund stage when admission took place.478 Similar patterns were traced by Melling and Forsythe in admissions in the early 1880s to Exminster Asylum, where many of those who succumbed to exhaustion, especially the majority of males, were in feeble health when admitted.479 This thesis examines the prevalence of these factors in a cohort of deaths with exhaustion at the turn of the nineteenth century.

Another contention of some scholars is that the use of exhaustion as a cause of death, and more particularly its imprecise usage, arose because it medicalised deaths where no cause was found and served as a mitigation to make asylum mortality more acceptable to outside observers. David Ranson, for example, remarks that alleging exhaustion deaths were directly due to mental illness where the death had occurred in circumstances where individuals had been restrained could be seen as a way of deflecting investigation away from the contributory actions of medical or nursing staff.480 However, Kodikara points out that because clear-cut signs of pathology at post-mortem were commonly absent amongst those dying from exhaustion in mania, ascribing death to an antecedent like restraint or malpractice was conceptually and legally challenging.481 In this thesis, case notes were perused to see if the violent nature of the patient’s symptoms was linked to similarly violent staff conduct and whether there was evidence of non-accidental injury, such as a suggestive history of force or violence or post-mortem indications of such conduct. Very few such cases were identified, though evidence of these issues being hidden or overlooked was also found, while the involvement and impact of the coroner or procurator fiscal in such cases appeared to be limited.

This chapter seeks to enhance our understanding of what led to death by exhaustion and what the key underlying clinical associations of that label were. The theories of contemporary observers and modern historians about the modes, mechanism and impacts of treatments on death by exhaustion will first be discussed. As described above, a prominent narrative about the underlying

478 Smith, ‘‘Visitation by God,’’ 110.
mode of death, which first developed in the late Victorian period, was that emotional events coupled with the excesses of extreme exhaustive behaviour, including malnutrition, placed undue strain upon the heart which led to demise of the sufferer. This became the accepted explanation by the 1930s when several commentators argued that death by exhaustion was cardiac in origin in sufferers with underlying vulnerabilities. A second, but related, explanatory theme was that exhaustion had affected vital brain centres controlling the heart and circulation. In 1860, Zachariah Laurence, a London surgeon who worked at Queen’s Square and the South London Ophthalmic Hospital, proposed that cessation of the heartbeat in a case of insanity was caused by irritation of the vagus nerve. Later, Kraepelin reported changes in heart rate and temperature regulation in mania which he ascribed to derangement in the central control of the heart; increased central sympathetic output with decreased parasympathetic modulation both of which would speed the heart up, potentially erratically. In 1935, the German alienist Stefan agreed with the suggestion that the nerves controlling the heart were implicated in exhaustive deaths: “we suspect a nervous influence upon the respiration and circulation by means of the channels of the parasympatheticus (sic) and vagus nerves causing atony of the circulatory system and all smooth muscular organs.” Bamford and Bean suggested that exhaustion and emaciation led to degeneration of the large nerve cells of the cerebrum, which in turn eventually produced death by circulatory collapse, though the evidence on which this assertion is based is less than convincing. By contrast, Larson, a pathologist from Washington State, was of the opinion that exhaustion was a direct result of dehydration and fall in total blood volume. He posited that reduced fluid intake of these inmates was pivotal rather than the diminished food intake which had so preoccupied Victorian alienists (see below). However, the limitations of all the above hypotheses must be emphasised. The original studies on which these theories were based were all anecdotal and the patients examined heterogeneous. There have been very few studies where post-mortem reports have been examined and those that have done so have only reported a few cases from small highly selected

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populations. In contrast, this study examines post-mortem reports from all those dying with this label in two institutions.

Leonard Smith discusses the treatment methods employed for severe mania and excitement in the early nineteenth century such as restraint, purgatives and emetics and contends that they may have exacerbated the risk of death rather than reducing it. Smith finds a series of cases firmly yoked to mechanical restraint, but points out that the relatively limited (and potentially biased) records of the time make the contention that it hastened the demise of many sufferers somewhat speculative. Furthermore, perhaps because of the risks but, more likely, the dissemination of the ethos of moral treatment, the use of physical restraint fell across the nineteenth century in British asylums. There was also a closer monitoring of restraint when employed and a rising use of seclusion consequent upon lunacy reform and legislation (from 1828) and of central LC surveillance of restraint registers (post-1845). Reduction in the use of restraint, however, was not universal nor without critique. In the asylums studied here, the use of purgatives remained commonplace but so universal as to make their role in patients’ demise impossible to assess. Chemical medication (from hypnotics and sedatives to anti-convulsives) started to be used more widely. Klaf and Hamilton outline the wide range of medications given at the Bethlem in the 1850s and point out that views on the benefits of such approaches was equally wide. This ambivalence is well marked in the primary accounts at the time on this topic. Another factor in enhancing ambivalence to drug treatments was cost. Shepherd finds that because of “financial constraints... medication was discontinued if an improvement was not apparent.” Primary accounts highlight significant attitudinal ambivalence to medication use amongst influential alienists. For example, John Havelock (Medical Superintendent at Montrose Royal Asylum in the late Victorian era), thought positively about use of some sedatives and hypnotics such as paraldehyde, chloral hydrate and, for urgent cases, hyoscine, which had come to prominence in the previous few decades, but was sceptical of a number of the “new drugs,” including sulphonial. He was one of the first to warn of the dangers of cumulative poisoning with these types of drugs which were becoming much more frequently used around this period, perhaps following on from the advocacy

490 Shepherd, Institutionalizing the Insane, 117.
of eminent Scottish colleagues such as Clouston.\textsuperscript{491} [Sir] Robert [Armstrong-]Jones (1857-1943), the distinguished alienist and onetime MPA President, first Medical Superintendent of the first LCC asylum at Claybury, condemned the excessive use of calmatives and hypnotics in the general treatment of insanity but supported the use of sedatives for the excitement of severe insanity and as a way of mitigating fatigue and exhaustion.\textsuperscript{492} Jones discounted the widespread use of alcohol in insanity, but said that he did not “hesitate to use it in small doses every few hours in the exhaustion of acute mania.” He felt alcohol was useful as “it increases the force and frequency of the heart’s action and stimulates vascular tone,” effects generally highly prized by Victorian alienists because, as outlined above, a number thought circulatory collapse was the mechanism or mode of death in exhaustion. Jones felt that strychnine was a cardiac stimulant especially good in countering the “cerebral anaemia”, strongly believed to underlie the mortality from exhaustion in acute insanity.\textsuperscript{493} The English alienist, John Ferguson claimed good results for the same reason from a mixture of strong, all potentially toxic, drugs including digitalis, ergot, belladonna and strychnine.\textsuperscript{494} More critical consideration of the role that the treatment of exhaustion may have had in the demise of sufferers is required as there has been little research looking at the potential association between the increasing use of drug treatment and medicinal cocktails to manage these conditions and death.

Jones also provided a useful summary of the management of exhaustion in the asylum and Victorian alienists’ widely held view that food intake was key to the prevention of death by exhaustion. Jones was a prominent campaigner for the early treatment of incipient cases of insanity, which significantly dovetailed with his perspectives on exhaustion. He stressed that maintaining the patient’s strength was particularly necessary in insanity because the “abnormal rapidity of disintegration which occurs in acute insanity impairs nutrition so quickly that death from exhaustion often takes place before the delirium subsides.” Jones advised “un-irritating food, easy of digestion and nourishing” (such as peptonized beef tea) to be given “little and often.”

\begin{footnotes}
\footnotetext{493}{Ibid, 878.}
\footnotetext{494}{John Ferguson, “The Insanity Following Exhaustion, Acute Diseases, Injuries etc.,” \textit{Alienist and Neurologist} 13 (1892): 407-38.}
\end{footnotes}
Heightened concerns about the dangerous symptoms and deleterious consequences of exhaustion clearly contributed to the great stress in Victorian asylums on artificial and force-feeding and on developing its technologies. Jones was far from alone in emphasising how often it was deemed necessary in cases of acute insanity to “feed (even forcibly) with tube and funnel, by the mouth or nose, two or three times daily... to counteract the dangerous profound exhaustion,” for without “sustaining treatment... the exhaustion may prove fatal in a few hours.”

495 Syndromes such as, neurasthenia and symptoms like weakness and malnourishment (with or without food revulsion/refusal) were commonly noted in the primary literature as common precipitants for exhaustion related admissions and have been seen as crucial by historians, but evaluation of this cohort throws doubt on whether death from exhaustion was significantly related to underlying malnutrition.496 The use of tube or artificial feeding (sometimes referred to as force-feeding with a rather different, more censorious, connotation by later commentators) and restraint (both physical and chemical) in the management of both highly overactive and markedly underactive inmates will be considered to see whether their use or abuse was as harmful as has been often conjectured, but less often evidentially established, by historians.

To examine these theories, an investigation of the case notes and pathology reports of a cohort who were given exhaustion from insanity as a cause of death was carried out, partly adopting the methodology of Renvoize and Beveridge, in that exhaustion cases where cause of death was clearly linked to a physical disorder were excluded.497 This dissertation is distinctive, however, in utilising the ascribed cause of death from post-mortem records which offers a more reliable basis for excluding obvious physical pathology, thus providing a more precise estimate of the prevalence of this cause of death and the strength of prevalent explicatory models for exhaustion. Critical attention is given, however, to whether this post-mortem endeavour in asylums represented a significant advance in understanding of the pathology and mortality, as proponents of a more scientific patho-anatomical approach to insanity claimed. The approach taken here offers the advantage of examining in tandem the clinical features of a more distilled and carefully selected cohort of cases, without any obvious physical cause of death, to elucidate with greater precision than in previous studies the antecedents (physical, psychological, and social) of the insanities that led to death by exhaustion. The widely held contention of historians that stress, social pressures

497 Renvoize and Beveridge, “Mental Illness and the Late Victorians,” 20.
(economical and/or inter-personal) and loss were the driving forces for this critical outcome in asylums could thus be tested.

The alternate less commonly held view that exhaustion associated mortality occurred in relatively specific clinical circumstances (such as severe agitation, religious delusions and food refusal) will also be assessed, as will whether deaths from exhaustion related to the presence of specific clinical states like agitated depression, catatonia or delirium. Death by exhaustion amongst the insane was examined in the 1950s by Jarvie and Hood, and although they write from a medical standpoint, they take a broad historical sweep and a critical look at the key issues. They agreed with Stefan’s view that exhaustive death was cardiac in origin and was precipitated by central dysfunction in sufferers with underlying vulnerabilities. Their review of case reports also leads them to conclude that an agitated depression, triggered by adversity, with its concomitant aberrant behaviours such as restlessness and stupor rather than psychosis or exaltation, was the forerunner of this syndrome. This contention is partly supported by Margaret Harris’s subsequent work on an asylum in North Wales which showed exhaustive deaths to be linked to severe melancholia rather than mania. This thesis exploits the opportunity to further test these theories. There are several other conditions which have been associated with death by exhaustion in Victorian asylums, including acute delirious mania and puerperal insanity. The study assesses how commonplace these conditions were in a cohort of all deaths from exhaustion in two asylums examined over three decades from 1872. Two other conditions, delirium and catatonia, have also been associated with exhaustion and with death and provide plausible explanations for the observed linkage between death of the insane and exhaustion. The presence or absence in this cohort of catatonic symptoms and dysregulation of vital signs in life seen by several authorities to be hallmarks of catatonia will be surveyed together with a search for evidence that delirium may have been a more appropriate clinical label for some of the cases.

\[\text{498} \quad \text{Hugh Jarvie and Martin Hood, “Acute Delirious Mania,” } \text{American Journal of Psychiatry} \ 108 \ (1952): \ 758-63.\]
\[\text{499} \quad \text{Harris et al., “The Morbidity and Mortality linked to Melancholia,” } 3-14.\]
\[\text{500} \quad \text{David Healy, The Creation of Psychopharmacology (Boston: Harvard University Press, 2009).}\]
Case finding was performed by looking through the Register of Deaths from each asylum and extracting case details where exhaustion from or with mania or melancholia or one of their variants was mentioned as a cause of death. At the same time the total number of deaths per annum in each asylum was recorded so as deaths by or with exhaustion could be presented as a proportion of total deaths. Since analysis of the post-mortem record was pivotal to this research, the years chosen for enquiry reflected the availability of post-mortem records. For the Berkshire Asylum, the years from 1872 (when post-mortems first became frequently performed) to 1906 (see Chapter 2 for the rationale for this date) were chosen and for Sunnyside from 1872 (for the same reason that this date was chosen for Berkshire) to 1902 (when post-mortems ceased to be performed so regularly).

In the asylum death register, exhaustion appeared both as a primary cause of death and as a secondary one. Doctors were told to list diseases as a cause of death in the sequence they had appeared and not the order of their importance. Mooney suggests that whether this stipulation was maintained, particularly during epidemics, is difficult to tell. In a study of more than 13,000 death certificates in 1866 in London, he shows that death certification in institutions was less thorough and diligent than that in the community speculating that death certification was seen as a procedural legal chore in these establishments rather than as a task of clinical worth. It seems likely that this lack of precision operated in asylums where the term exhaustion was liberally used. There are frequent accounts of exhaustion being a cause of death in asylums linked to disparate conditions such as dementia, cancer and epilepsy, and exhaustion was a common second cause of death in infections such as cholera and tuberculosis. Epilepsy could lead to death at any age. Many sufferers did not die of the disorder directly, but as a result of physical injuries or strokes occurring during several years of ongoing seizures. As there were few successful anti-convulsive treatments for epilepsy during this period, epileptic patients not infrequently died from the physical exertion

501 Data from the two asylums were combined. Analyses from earlier chapter showed that the similarities between the two asylums were much greater than their differences.
503 Mooney, “Diagnostic Spaces,” 357-90.
experienced during the often-prolonged attacks and in these circumstances, exhaustion was often recorded as a cause of death.\textsuperscript{504}

Cases where exhaustion was given as the cause of death of a neurological or somatic disorder were excluded from further enquiry. This strategy has the consequence of decontextualising “pure” exhaustion to some extent and not fully clarifying the place of exhaustion in Victorian mental science, nor examining all the diagnostic and conceptual ambiguities with which the concept of exhaustion was being used over time. However, time constraints did not permit this more wide-ranging analysis. Such cases were numerous, amounting to about one in ten of the deaths in the asylums, the most frequent examples being exhaustion from epilepsy or apoplexy. The only exception to this rule was the handful of cases when pneumonia was listed as a second cause of death to exhaustion. This was usually when pneumonia was a terminal event in an already moribund patient. There were a small number of cases of death from exhaustion with senile decay (never just “decay”) recorded who were also not taken further. Practice varied across the country, and, in some establishments, the senile decay label appears to have been used as a synonym for exhaustion. For example, Parker and colleagues in their study of Rainhill Asylum in Lancashire show a high death rate from senile decay (not all of whom were old) with very few cases of death by exhaustion.\textsuperscript{505} However all the cases here with senile decay were old (greater than 70) and there was sufficient uncertainty to exclude such cases and those other occasional causes of death which may have had similarities or associations with exhaustion, such as marasmus, syncope and congestion. It would be of interest to explore these diagnostic terms in a future project as the consistency of the contemporary distinction between them and exhaustion remains an open question.

4.2.1 Numbers and frequency of cases

The above strategy identified 155 cases where exhaustion from mania or melancholia or one of their variants was the label given as the cause of death across the period 1872-1906. All these cases were paupers, except for one lady from Montrose who was initially admitted to Sunnyside as a private patient. After four days she became very unwell and was transferred to the hospital


\textsuperscript{505} Parker et al, “County of Lancaster Asylum, Rainhill,” 104.
wing which involved the loss of her private status. To judge whether the use of the label exhaustion was changing in use over the time period of this study, deaths with the label exhaustion from mania or melancholia are shown in five-year blocks across the period in Figure 4.1. The use of these blocks is to minimise the effect of changes in this infrequent outcome from year to year, to smooth the data.

Figure 4.1 shows that towards the latter decades of the century the number of deaths labelled as exhaustion with insanity was falling. However, there was a rise in the number of cases in the latter part of the 1890s and probably in the first half of the 1900s (although figures are only available for one asylum in this latter period). This late rise was due to the presence of cases of senile mania given exhaustion as a cause of death. This is likely to reflect the increase in the elderly population in asylums which was very evident by that time. As far as deaths in the less elderly population is concerned, there was a fall in the use of the label from the periods reported first by Smith, then Renvoize and Beveridge, and subsequently Wright to the era covered by the current data. The lower prevalence in the current study may represent the more limited use of this label in the two asylums selected for study, although none of the other labels related to exhaustion (such as prostration or decay) were used. Alternatively, it may be that such cases were becoming less frequent overall, as the current data are collected from a period later in the nineteenth century.
than either Renvoize or Wright. It is difficult to distinguish between these two possibilities and other factors which may be operating. For example, the presentations of mania and melancholia may have been less severe in this period than in previous ones or, more likely, changes in diagnostic techniques meant that physical diseases underpinning the final illness were identified more readily. This latter effect may be related to the more widespread use of post-mortems. Such conclusions remain tentative, nonetheless.

Of these 155 cases, 90 (58%) had a post-mortem and all of these post-mortem reports were scrutinized. In the vast majority of cases, the cause of death stated in the post-mortem report was the same as that seen in the Death Register. In some instances, however, a different or altered cause of death was found in the post-mortem records. For the purposes of the analyses below, the post-mortem cause of death was used (see Chapter 3 for a discussion of this issue). Post-mortem data and whether an inquest was carried out (and, if so, its outcome) were recorded onto a spreadsheet. In 19 cases (21% of the 90 who had a post-mortem), there was sufficient evidence to indicate that the cause of death was due to a major physical disease. In nine of these cases, a physical disease which had not been mentioned in the Register appeared as a cause of death in the post-mortem report. Outline details of these cases are shown in Appendix G. In the other ten cases, the cause of death remained as seen in the Register (i.e., exhaustion) despite clear evidence of incapacitating physical illness in the post-mortem report. Six had clear cut evidence of active TB at post-mortem including tubercular masses, with caseation, the size of “walnuts” in one case and “pigeon’s eggs” in another. All were therefore very probable deaths from TB. Two cases, one with senile mania and one with senile melancholia, had highly inflamed bowels at post-mortem and a history of marked diarrhoea in their case notes, so were deemed to have died from gastrointestinal infection.

The two remaining cases where somatic disease was deemed to be the cause of death were particularly interesting. The first (FC) was a 43-year-old married wife of a whitesmith with six children. In her admission records the cause of her insanity was stated as rheumatic fever. Although there was a lot of evidence to support rheumatic fever as a cause of her illness and her death, this was not reflected in either the register or in the post-mortem report. Her insanity had all the hallmarks of a delirium with disorientation in place and time and indeed she was described

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506 Caseation is a pathognomic sign of active TB, recognised in Victorian times and to this day.
as “delirious.” She had an intermittent fever during which she had transient delusions that the nurses had murdered her children. She had physical signs of rheumatic heart disease and at post mortem the heart showed “vegetations” on her valves, the pathognomic sign of infective endocarditis (an infection of the heart lining and valves). This condition had been recognised in 1851, had been extensively reviewed by Osler in 1885 in his three Gulstonian Lectures and was known to cause the sufferer to be exhausted. Here, all the signs and symptoms of this condition had been recognised and reported so it is not clear why it was not recorded as a cause of death. The second case also throws up a few issues. The patient (JH) was a 63-year-old pedlar, married with two children. Although he lived in poor circumstances he had never been in the workhouse. This was his fourth admission having been in a number of asylums in the South of England. He developed melancholia; he was actively suicidal, and he expressed the view that he had offended against “moral law.” He heard the voice of the Lord whispering that he “was going to Hell.” From being depressed and miserable, he suddenly became excitable, restless and then violent. He struck his head against a wall and tried “to tear out his testicles” which he later said was “at the command of God.” There was a violent encounter with the staff, reportedly started by the patient but we have no way of knowing whether the staff’s response to his violence was inappropriate, compounded by the rather inexplicable lack of an inquest. He was put in gloves and placed in a padded cell but sank rapidly over the next few days and died. Post-mortem revealed widespread disease of the vessels to the heart and brain and a cerebral haemorrhage “of two to three days duration.” Thus, he died not of exhaustion (though this was a feature of his last days) but of either a stroke or a bleed in the brain, potentially exacerbated by a possible self-induced head injury. It is not clear why the cerebral haemorrhage and head injury are not reflected in his records, nor why there was no inquest. This would have exonerated the attendants if that was the way the evidence pointed. This must count as a suspicious death, with the question unresolved as to whether asylum staff may have been responsible or at least complicit in the patient’s demise. The finding of these 19 cases indicates that that a significant minority (it was roughly one in five in the current data) of deaths ascribed to exhaustion are in fact secondary to physical disease. A very similar proportion of cases were found by Derby in his 1933 study to be either definitely or probably due to a physical condition. These observations should be borne in mind when considering the

frequency and associations of exhaustion reported in primary and secondary studies which have not carried out this procedure.

The age, gender and principal diagnostic label of the total 155 cases, for the 90 that had post-mortems and the 71 in which major physical disease was excluded, are given in Table 4.1. The ages of the groups were comparable to, but somewhat younger, than that found for exhaustion deaths by Healy and colleagues. There is a slight preponderance of females seen in both groups, paralleling contemporary reports and those of secondary analyses. Mania cases outnumber cases of melancholia by two to one in in the large sample but, for reasons that are unclear, proportionally less cases of melancholia had a post-mortem and so in that group with a post-mortem, mania outnumbers melancholia by three to one.

<table>
<thead>
<tr>
<th>Group</th>
<th>A. Death by exhaustion from mania or melancholia (total).</th>
<th>B. Death by exhaustion with post-mortem report.</th>
<th>C. Exhaustion deaths after exclusion of cases with clear-cut physical disease at post-mortem.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>155</td>
<td>90</td>
<td>71</td>
</tr>
<tr>
<td>Mean Age (years)</td>
<td>55</td>
<td>55</td>
<td>57</td>
</tr>
<tr>
<td>Sex M/F</td>
<td>73/82</td>
<td>41/49</td>
<td>29/32</td>
</tr>
<tr>
<td>Mania/Melancholia</td>
<td>102/53</td>
<td>67/23</td>
<td>53/18</td>
</tr>
</tbody>
</table>

Table 4-1 Number, gender, age at death and diagnostic group of all patients dying of exhaustion between 1872 and 1906 in both asylums combined (A), those deaths who had a post-mortem (B) and those in whom no obvious primary physical cause for death was identified at post-mortem (C).

After exclusion of cases where exhaustion was reported in the Register as a cause of death secondary to a physical disorder, 155 cases were identified where the sole association was with mania or melancholia. This collection was over a 35-year period in the Berkshire Asylum and a 30-year period in Sunnyside. Over these periods, there were approximately 3,000 deaths in the two asylums which leads to a crude proportion of deaths from exhaustion in insanity of just over 5%. As demonstrated above, of those 90 that had post-mortems, 21% had clear-cut and major illnesses to account for a death which was ascribed as exhaustion. Table 4.1 demonstrates that the

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510 The 3000 figure is an estimate but one very close to the correct figure. Unavailability of archive material due to the pandemic prohibited final accurate checking.
demography of the cases who had post-mortems is broadly similar to that of the larger group. Therefore, if it is assumed that 21% of the cohort of 155 had major physical disease to which exhaustion was secondary, then there would be about 122 “pure” cases, giving a rate for death for this outcome of just over 4%. This figure shows close resonance with the data on deaths by exhaustion from the Scottish LC’s report for 1893.511 Across all the asylums in Scotland, including Royal, District, parochial and private ones, there were 813 deaths that year, 33 of which were by exhaustion with mania or melancholia, also giving a rate of 4%, though the degree to which cases of death by exhaustion attributable to somatic diseases were categorised elsewhere in this database is unknown.

This estimate, that 4% of deaths in asylums were by exhaustion, is lower than that cited by other historians but there are several explanations for this disparity. In their study of Yorkshire asylums, Renvoize and Beveridge, using broadly similar methods to those here, found a death rate by exhaustion of 14% at the Retreat and 9% in two other asylums but these authors did not perform post-mortem analysis. Wright quotes a death rate by exhaustion of about 7% but did not exclude cases where physical illness predominated and that study was carried out on Ontarian asylums.512 The strictures of Ernst cautioning about transnational comparisons must be borne in mind, even though the Ontario asylum system in this period is thought to have more similarities to the UK system than differences.513 It is important to take into account the other major challenges in interpreting such data, especially when comparisons across time or across cultures are considered or attempted. Risse reflects that “cultural norms, contemporary scientific knowledge, and technological methods” shape diagnoses at any point in time.514 This echoes Anderton and Leonard’s view that causes of death are constrained by the historical context of the era in which they were recorded.515 Death certificates can inform historians about the spread of medical ideas and are indicators of the links between a disease and demography, but Prior and Ernst discuss the many inherent drawbacks and limitations of this approach particularly related to the changing

511 Thirty-fifth Annual Report of Commissioners in Lunacy for Scotland (Edinburgh: HMSO, 1893). Year chosen as it is that of the mid-point of the cases collected here.
512 Renvoize and Beveridge, “Mental Illness and the Late Victorians,” 19-28; Wright, Jacklin and Themeles, “Dying to Get Out of the Asylum,” 600.
514 Risse, “Cause of Death as a Historical Problem,” 175-88.
conceptual categories used and shifting diagnostic practices.\textsuperscript{516} Prior suggests that the arrangements for registering deaths, dominated as they were by the principles of forensic medicine, are best viewed as a system for policing the dead, rather than as a mechanism for establishing clear-cut data about diseases and their social and temporal distribution.

4.2.2 Case note assessments

The available case notes (and, in most cases, the admission register entries) of the large majority of the remaining 71 cases were summarised to provide data on age, sex, whether this was a first or subsequent episode and length of episode. An attempt was made to compile a profile of the patients’ social circumstances (and whether these had changed recently) and the presence or absence of “life events” (e.g. physical ill-health, death or separation from relations or other relationship difficulty, financial hardship, and overwork) in the run up to the changed behaviour and admission. The “cause of insanity” as documented was recorded. The circumstances of admission were noted along with (a summary of) the physical and mental state on admission and the case note diagnosis. The course in the asylum of these cases was traced and the circumstances of death summarised.

Of these 71 cases, 14 had a diagnosis of senile mania. The mean age of the senile mania group was 72 years with the youngest 62 and the oldest 86. This mean age and age range is very different from the group without the label senile, which was 52 years with very few cases over the age of 65. Furthermore, it became apparent that the label of senile was more than just a marker of age: in the majority of cases, the clinical picture and the post-mortem findings in the brain were very different from the group not given such a label. Nearly all these 14 cases had frequent references to cognitive compromise and poor memory in their notes, in distinction to their sparsity in those without such a label. Examples include “incoherent and rambling speech”, “disorientation”, “unable to understand simple questions,” along with statements about deficits in memory for both past and recent events. These cases often exhibited symptoms seen in those with marked memory impairment such as misidentification of staff, relatives and locality, sometimes reaching delusional proportions. Dementia was mentioned in a significant number of these cases as a descriptor rather than a specific label. The post-mortem brain findings of the group with senile

Mania also showed differences from the group without such a label. Atrophy, particularly of the convolutions, and shrinkage of the brain, particularly the sulci, were commonly mentioned. The brain was often described as excessively soft in these cases, though that finding lacks any specificity as it was fairly frequently reported in the non-senile group as well. There were two cases ascribed as senile mania which did not fit the pattern of the other 12 cases of senile mania closely: their details are given in Appendix H.

It is noteworthy that the label of senile mania (and, in one case, senile melancholia) was applied to the cases reported here in the late 1890s and early 1900s. Prior to this time, the term senile mania was used to designate those with mania who were old, or considered old, irrespective of memory problems. Dementia was widely used in the earlier decades of the century usually, but not invariably, to denote an insane person with acquired memory deficits. Berrios charts how the concept of dementia changed significantly over the course of the nineteenth century, the “cognitive paradigm” eventually becoming dominant by the century’s end. In some asylums (like the ones here), the term senile mania rather than dementia was used to designate those with cognitive problems in the later part of the century. However, this use of the term senile mania for dementia was patchy: Yorston and Haw describe how senile mania was commonly used at one Oxford asylum (the Warneford) but rarely at another (the Littlemore). In the latter asylum, medical staff began to use the term dementia for such cases more frequently across the last decade of the nineteenth century. This became universal practice by the early 1900s. It is conceivable that the rise of the term dementia was linked, in part, to reduced and more specific use of the term exhaustion in the later part of the nineteenth century and beyond but this requires further enquiry. Senile insanity of various types continued to be used to denote those with psychosis occurring in later life. The social history of the use of the terms senile and dementia are well traced by Emily Andrews.

These terms were explained and discussed in Chapter 3.

There was only one use of that diagnostic descriptor prior to the 1890s, which was in 1873, in this data set. This case was also given a cause of death of “senile decay” and was found to have had severe diarrhoea and an inflamed bowel at post-mortem. This led to her exclusion from the study.

Berrios, “Memory and the Cognitive Paradigm During the Nineteenth Century,” 194–211.


4.2.3 Circumstances of admission

Of the 71 cases with no clear-cut physical disease at post-mortem, about two thirds had less than 4 weeks between admission and death and in 90% death occurred within 6 months of admission. A very small number had long admissions and death by exhaustion came as a late event. Of those who died within 6 months the average time between admission and death was just over 5 weeks with many succumbing in just a few days. In view of the findings regarding the group with senile mania described above and the historical evidence about the use of that term, it was decided to examine the clinical and pathological underpinnings of death from exhaustion in more detail in the group without that label. This cohort consisted of 57 subjects, the demographics of which are shown in Table 4.3. 38 patients with mania died of exhaustion, as did 19 with melancholia. These findings do not concur with Healey’s group who found a much higher death rate from exhaustion in melancholia and who suggested some specificity of this outcome for melancholia.\textsuperscript{522} The reasons behind these disparities are not clear. Physical co-morbidity may have accounted for at least part of this, but labels varied in their popularity from place to place as well as across time. In the current study, there were interesting differences between deaths from exhaustion in mania compared to those dying of exhaustion in melancholia. The death from exhaustion with mania group was younger (50 years) than the melancholia group (57 years). Death came much more quickly in the mania group with about 75% dying within 4 weeks of admission compared with only about 30% of melancholia sufferers with such rapid deaths. The average length of stay for the melancholia group was more than twice as long at 58 days than the 27 days for the mania group. The finding in mania of a short illness terminated by death from exhaustion is comparable with Derby’s study in 1933, which found that 55% of such cases had an illness of less than three weeks duration.\textsuperscript{523}

Circumstances surrounding the admission of these cases were assessed. Case notes from 34 were perused and data from the admission register has also been examined in many cases.\textsuperscript{524} In the vast majority (27) of these cases, the fatal admission was the first attack. In 6 cases the final admission was for the second attack and in 1 case the third admission. This finding concurs with that

\textsuperscript{522} Harris et al., “Morbidity and Mortality Linked to Melancholia,” 3-14.
\textsuperscript{523} Derby, “Manic-Depressive ‘Exhaustion’ Deaths,” 443.
\textsuperscript{524} Case notes were examined from 34 of the 57 cases with a post-mortem who had had physical disease or senile mania and melancholia excluded. The selection of the 34 was based on the availability of case-notes. Some case notes were missing or unavailable and the arrival of the Covid-19 pandemic hampered further search for these notes.
reported by Smith on a much less filtered population, suggesting that death by exhaustion has a robust association with the first episode of major illness.\textsuperscript{525} The length of the terminal attack before admission was stated in most cases. It is accepted by scholars in this and related fields that determination of onset of episode is a notoriously difficult and subjective measure.\textsuperscript{526} Despite this, the Victorian alienists working in these asylums gave precise figures for the duration of disease for most of this sample. Only in two cases was no figure stated: the duration of illness before admission was said to be “some months” in one and “some days” in another. In a small number of cases, complexity was acknowledged, with the records noting that there was evidence that the patient had been unwell for quite a time, with a recent dramatic worsening. In these cases, it was this worsening that was given as the start of the attack. The giving of precise durations in this cohort may have been justified because many of the insanities were said to be sudden in onset in someone previously well. “Sudden onset” was specifically stated for nearly half the patients in which case notes were examined. The mania cases showed particularly short total illnesses. Of the 25 mania cases looked at in detail, only 3 had a combined length of illness (duration of illness added to length of admission) of more than 2 months and 7 patients with mania had illnesses totalling less than 2 weeks. There were a number of cases where the patient had been completely well and a few weeks later, they were dead. Melancholia demonstrated quite a different pattern with none of the cases having total lengths of illness of less than 2 months, with most being unwell for 4 months and in many cases much longer. It was clear from the case notes, that many of those who died exhausted had been very restless, overactive and in many cases excitable and not open to reason.

As discussed in the Introduction to this chapter, there was a widely held belief by alienists at the time and subsequently by historians of this topic that acutely stressful precipitants were forerunners of death by exhaustion. This contention was examined. Out of the 34 cases which underwent detailed case note analysis, there were 10 cases where such an association was possible. The recorded precipitants of the 8 mania cases with this profile are documented in Table 4.2.

\textsuperscript{525} Smith, ““Visitation by God’,” 110.
<table>
<thead>
<tr>
<th>Name, age (years), gender</th>
<th>Length of attack. Period of illness before plus after Admission. =Total (days)</th>
<th>Type of onset. Number of attack.</th>
<th>Precipitants mentioned in case notes.</th>
</tr>
</thead>
<tbody>
<tr>
<td>RE, 53, M</td>
<td>7 + 100 = 107</td>
<td>Sudden onset. First.</td>
<td>Had a large family. Lost job. Became impoverished with insufficient food and clothing. Quiet, religiously inclined and temperate. “Grief at loss of situation.”</td>
</tr>
<tr>
<td>MN, 33, F</td>
<td>6 + 7 = 13</td>
<td>Sudden onset. First.</td>
<td>Four months pregnant but suckling infant of 16 months. FH +ve.</td>
</tr>
<tr>
<td>RM, 48, F</td>
<td>14 + 8 = 22</td>
<td>Sudden onset. Third.</td>
<td>Single domestic servant, very temperate and hard working. In service to a Baptist minister whose wife died. Because he talked to her and shook her hand, the patient “developed the idea that he was going to propose to her” and she had an unshakeable belief in his love. His engagement was announced to another, and she went “clean off.”</td>
</tr>
<tr>
<td>TB, 48, M</td>
<td>7 + 6 = 13</td>
<td>Sudden onset. First.</td>
<td>Single labourer who has undergone “privation and had business worry.” Attack said to be cause by “disappointment in love” - jilted by his first cousin.</td>
</tr>
<tr>
<td>AW, 66, M</td>
<td>7 + 13 = 20</td>
<td>?</td>
<td>Intelligent, temperate agricultural labourer but no work for 5 weeks. FH +ve. His wife had to go to London for an operation for an internal complaint since when he has not been able to cope.</td>
</tr>
</tbody>
</table>
Onset of illness was sudden in most of these cases and disappointment in love, straightened circumstances and illness in the family were the most common apparent precipitants. Some cases were very dramatic. One single lady (RM, aged 48) had developed what appears to be erotomania. Berrios and Kennedy trace the historical concepts underpinning erotomania, though Ellis and Mellsop throw doubt on the specificity and usage of this term; neither erotomania nor the commonly used “Old Maid’s Insanity” (as defined by Tuke) was applied to this case. Whatever the status of the syndrome of erotomania, this patient’s fatal illness was precipitated by clear evidence that her love was not returned and she was dead within two weeks of this realisation. Another noteworthy case (MMcD, aged 35) had a traumatic encounter and a rapid development of mania. In both cases, it may be relevant that their predisposition was shy and retiring. Several of these cases had a positive family history and the hereditary predisposition may have played a part in their vulnerability to these stressful events. In melancholia, only two cases had any specific life circumstance identified in the prelude to their disorder which, as outlined above, was much more gradual than the mania. One was a wealthy farmer who was preoccupied with his sister’s illness, but this concern was longstanding, and he also had a very strong family history of insanity. Another was a 40-year-old single grocer, the cause of whose severe melancholia was emphatically stated as masturbation. Around this time St John Bullen, assistant medical officer and pathologist from the West Riding Asylum, attributed exhaustion to “marked degenerative tendencies” and outlined “prolonged physical drain, mental exhaustion, or moral depravation of masturbatic (sic) habits” as the immediate causes. Clouston also expressed such ideas forcefully. The reasons

<table>
<thead>
<tr>
<th>Name</th>
<th>Onset</th>
<th>Cause</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>MMcD, 35, F</td>
<td>Sudden</td>
<td>Married cook. Cause “a fright.” Patient had been at Glamis Castle as a volunteer cook at a camp. Returning to the tent was shocked to see a man in there, stealing beef. She got such a fright that she was sleepless resulting in mania. In the asylum she said there was a quantity of meat at the top of her bed.</td>
<td></td>
</tr>
</tbody>
</table>

Table 4.2 Length of illness, number of attack and apparent precipitants in eight cases of mania who died of exhaustion. Presence of family history of insanity is denoted by FH +ve.


behind exhaustion being seen as a concomitant of the insanity of masturbation have been analysed by Hare and Laqueur.\textsuperscript{529} The patient was described as a religious enthusiast who confessed to having practiced the habit for many years. He had marked religious delusions, believing he had “sinned away the day of grace” and that, despite preaching to others, it is he who “falls short.” While masturbation seems an unlikely cause of his death from exhaustion with melancholia, it was deemed to be a marked problem and exhaustion explicator, the case notes indicating that the “seminal emissions occur several times a day.” 16 cases of mania and 8 patients with melancholia had no precipitant identified. It seems likely that, had there been a major life event in the run up to these serious illnesses, it would have been noted, given the well-known interest that Victorian alienists took in such associations.

In summary, it seems that while some cases had their fatal insanity yoked closely to a specific stressful event, the majority did not. Furthermore, in the case notes of young patients who died of causes other than exhaustion, outlined in Chapter 3, such events were equally common, suggesting that acute stress per se is not specifically linked to death by exhaustion. This is not however to say that the stress of being unwell and insane in late Victorian Britain, with its attendant risks, did not have a significant impact on health. In his overview of this topic, Shulack reported 12 cases where the onset of insanity coincided directly with an excitement or fright culminating in death from exhaustion. Post-mortem examination revealed nothing other than minimal congestion.\textsuperscript{530} Such reports strengthen historians’ view of the lethal power of emotions and their inhibitory effect on the cardiac cycle.\textsuperscript{531} However, in this study, only one patient (MMcD) could be said to have died following a fright. Of course, testing this hypothesis retrospectively is fraught with limitations, not least of which is the subjective nature of stress. Therefore, the only feasible conclusion in this regard is the tentative one that acute stresses and frights do not appear to have a specific or strong link to death by exhaustion in this dataset.

\textsuperscript{530} Shulack, “Sudden “Exhaustive” Death,” 3-12.
\textsuperscript{531} Robertson, “Sudden Deaths from Trivial Causes,” 110-24.
4.2.4 Food and nourishment

A number of historians conclude that poor nourishment was strongly associated with the insanities that led to asylum admission, particularly amongst those who subsequently died though, as we have seen in Chapter 3, malnourishment was very far from universal in young deaths. This view reflects the preoccupation that Victorian alienists had with food intake, both in its importance as a factor in illness but also its role in the management of insanity. Historians consider poor nourishment on asylum admission to be a marker of both economic hardship and poor bodily health and often link malnourishment to the predilection for multiple infections. Malnourishment is also a frequent explanatory narrative for those who had exhaustion and, again, those who died from it. The relationship between death by exhaustion and nourishment was explored by looking at body weight on admission (in those whose case notes were examined) and at the external appearance of the body in those who had a post-mortem. The results of this enquiry can be seen in Table 4.3. In several cases there was specific mention of food refusal (usually early in the admission but sometimes only later) and this phenomenon is also documented in Table 4.3 together with a note on whether tube feeding was carried out.

Exhaustion deaths with post-mortem (after exclusion of cases with clear-cut physical disease and senile mania)

<table>
<thead>
<tr>
<th>Number with reported emaciation on admission</th>
<th>14</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number with reported emaciation at death</td>
<td>20</td>
</tr>
<tr>
<td>Number reported as well-nourished on admission</td>
<td>15</td>
</tr>
<tr>
<td>Number reported as well-nourished at death</td>
<td>9</td>
</tr>
<tr>
<td>Number with specific report of “food refusal”</td>
<td>23</td>
</tr>
<tr>
<td>Number tube fed</td>
<td>20</td>
</tr>
</tbody>
</table>

Table 4-3 Bodily state on admission and death in exhaustive deaths. (NB 5 cases unclear/not stated on admission and 10 at death).

As can be seen from Table 4.3, only about half the exhaustive deaths, with a specific record of weight, were described as emaciated on admission, with the other half being described as well nourished. The former state was generally linked to a presentation of acute mania whereas the latter was more commonly seen in melancholia with a longer duration of illness. About two thirds of the cases were emaciated or severely emaciated at death but a third remained well nourished. Food refusal was specifically mentioned in about 70% of the cases during their last illness and was generally associated with tube feeding during life and emaciation at death. This study also confirms Doody’s findings in her survey of Fife asylums that it was males who were more likely to

536 Tube feeding was all but invariably by mouth but in one case, that of a 19-year-old female with a diagnosis of “acute delirious mania,” it was by enema. Further details of this case are outlined in Appendix I.
be tube-fed. It may be, as Doody suggested, that attendants were less inhibited about using force-feeding measures, if necessary, with males.\textsuperscript{537}

As has been highlighted by a number of scholars, preoccupation with religion was commonplace amongst Victorian asylum patients and this was often manifested by delusional thinking, centred on sin.\textsuperscript{538} Smith comments that some Victorian alienists drew particular attention to the risks of food refusal associated with religious delusions.\textsuperscript{539} Doody finds religious delusions in about one in seven inmates but this is a much lower frequency than that found in the current study, where about half the cases who died of exhaustion were preoccupied with religious delusions.\textsuperscript{540} It is noteworthy that all of these cases also exhibited food refusal. One man, a groom, saw visions of God and felt he should have a hole in his hand to let the poison of his sin against God out. He died of severe emaciation as did a female patient from Sunnyside (originally a private patient) who continually called on God, convinced she had been damned.

These findings suggest, as others have concluded before, that death by exhaustion is homogenous in nature and perhaps a final common pathway for a number of syndromes.\textsuperscript{541} Emaciation at admission and at death was common but by no means universal. Some were well nourished and, even when they ceased to eat on account of their moribund state, the final illness was not long enough to lead to much weight loss at death. On the other hand, there were several examples of people whose pre-admission state was parlous and in some of these cases these difficulties were compounded by religious delusions and food refusal. Food refusal manifests several components ranging from profound poor appetite (anorexia) to ideational aversion (including fears that the food is poisoned), but there seemed to be no example where it was primary in the sense that the sole feature of illness was food avoidance, although some cases of melancholia exhibited diminished eating right from the start of their illness. Whatever the reasons behind restricted food intake, the standard asylum response was artificial feeding which must have been a very unpleasant experience for asylum patients, both physically and mentally. Even with the best of intentions (and it was often lifesaving), artificial feeding could easily descend into an abusive, \textsuperscript{537} Doody, Beveridge and Johnstone, “Poor and Mad,” 887-97.
\textsuperscript{538} Renvoize and Beveridge, “Mental Illness and the Late Victorians,” 25; Klaf and Hamilton, “Schizophrenia — a Hundred Years Ago and Today,” 822.
\textsuperscript{539} Smith, “Welcome Release,” 122.
\textsuperscript{540} Doody, Beveridge and Johnstone, “Poor and Mad,” 894.
coercive practice (hence its other designation, force-feeding), serving to emphasise the patient’s lack of agency. Larson postulated that exhaustion was a direct result of dehydration and fall in total blood volume rather than the diminished food intake. Supporting evidence for this proposition is lacking, however. Although there was no mention of fluids or hydration in any of the case notes I examined, fluids were administered frequently both by mouth and by tube to these inmates particularly in the form of beef tea, so Larson’s contention was not specifically supported by this data.

4.2.5 Post-mortem findings

A limited number of modern historical studies have analysed a select sample of post-mortems from patients who died of exhaustion. Many contemporary reports mention congestion of the brain. In his review of them, Locher, however, is adamant that congestion in cases of exhaustion was no more frequent or marked than in cases of insanity who did not die of exhaustion. In the current series however, congestion in the brain was both widespread and common. About 75% of the exhaustion deaths exhibited congestion, more than twice as commonly as the non-exhaustive deaths (33%, Chapter 3). As highlighted in Chapter 3 however, congestion is a non-specific finding, and it may be that the alienists performing the examination were mindful of the exhaustive cause of death and its alleged association with congestion. Examples of post-mortem reports from some of these cases are reproduced in Appendix A. Cerebral softening, shrinkage and oedema were found in about a third of cases, rather less than the non-exhausted group outlined in Chapter 3. Atheroma in basal arteries was however found in about 45% of these cases and focus will now therefore turn to post-mortem findings in the rest of the vascular system.

Some commentators are convinced that exhaustion deaths had the hallmarks of a cardiac death. Derby concludes that “a dilated heart with thinned walls” was a key feature of post-mortem studies and Larson collates evidence of circulatory collapse with right-sided cardiac dilatation and cardiac failure. Building on this evidence, this chapter explores this cardiac hypothesis via

542 Ian Miller, “‘A Prostitution of the Profession?’ Forcible Feeding, Suffrage and Medical Ethics, 1909-14,” Social History of Medicine 26 (2014): 225-45
543 Larson, “Fatal Cases of Acute Manic-Depressive Psychosis,” 971-82.
comprehensive analysis of a specific cohort of exhaustion deaths. The post-mortem findings related to the heart and blood vessels of 57 cases of death by exhaustion were examined. Case notes on the physical examination of heart function of 34 of this cohort were also analysed. Results are shown in Table 4.4.

<table>
<thead>
<tr>
<th>Number of observations</th>
<th>Findings of the pulse\textsuperscript{546}</th>
<th>Heart signs on examination\textsuperscript{547}</th>
<th>Atheroma of basal arteries and/or aorta and/or cardiac vessels at PM</th>
<th>Major Heart abnormalities at PM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of observations</td>
<td>34</td>
<td>34</td>
<td>57</td>
<td>57</td>
</tr>
<tr>
<td>Definitely abnormal</td>
<td>7</td>
<td>12</td>
<td>29</td>
<td>38</td>
</tr>
<tr>
<td>Probably abnormal</td>
<td>15</td>
<td>6</td>
<td></td>
<td>7 with dilatation of ventricle. 23 with fatty heart or equivalent.</td>
</tr>
<tr>
<td>Normal</td>
<td>4</td>
<td>5</td>
<td>28</td>
<td>19</td>
</tr>
<tr>
<td>Uncertain/no mention</td>
<td>8</td>
<td>11</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 4-4 Cardiovascular findings on examination and at post-mortem (PM) in exhaustive deaths.

Victorian alienists were much taken by the prospect of analysing the physiology of the body and keen on developing effective techniques and instruments to aid this endeavour. Stowe reasons that the pulse was seen as a simple means of “seizing upon the essence of the closed, coded body.”\textsuperscript{548} There was a sense that a patient’s emotional state could alter the character of the circulation: the mind was described as a “stimulant” to the pulse.\textsuperscript{549} Wallis describes how the advent of devices to measure pulse (the sphygmograph) and, later, blood pressure and blood

\textsuperscript{546} Pulse: - Definitely abnormal:- 100/min and/or irregular; Probably abnormal:- rate over 90/min and/or weak, poor tone.

\textsuperscript{547} Heart signs: - Definite:- Cardiac hypertrophy and/or murmur; Probable:- heart sounds abnormal and/or weak, muffled.


thickness moved things on from the suggestion that certain forms of mental disease were associated with vessel anomalies on the basis of simple visual or sensory assessments (cold extremities) to reliance on technologized “real” measures. These instruments appeared from the 1860s in pioneering research asylums like the West Riding Asylum but up to 30 years later in most ordinary asylums and were not in evidence in the asylums studied here.\(^{550}\) Only a small number of admissions had normal findings on examination, with a slightly greater number being unrecorded. The majority exhibited an abnormal or probably abnormal pulse (65%) or heart signs (53%) with about 75% having one or both of these. In about half the post-mortems, atheroma was present either in the large arteries such as the aorta or basal vessels of the brain or in the heart itself usually around the coronary arteries.\(^{551}\) This is a striking finding, particularly in a population whose average age was 51 years. Atheroma was seen in several patients in their 40s. Cardiac findings were found in 67% of cases and consisted of major structural changes such as enlarged (hypertrophic) or dilated hearts. Abnormalities of the myocardium were reported frequently with cases being described as having a “fatty heart” or cardiac muscles that were pale, soft, and friable or degenerated. The post-mortem reports regarding the heart were, for the most part, detailed and anatomically thorough. These findings indicate that a substantial proportion of the insane who died of exhaustion in late Victorian asylums had abnormalities of the heart on clinical examination and on pathological examination.

Only 2 of the 34 patients who had their case notes examined had a positive report of a normal heart on examination and no cardiovascular findings at post-mortem. One of them was a 44-year-old woman with a sudden onset of her first attack of severe melancholia. The other was a 44-year-old labourer (RD) who had a rapid onset of mania and who was found to have three fractured ribs at post-mortem. There is a detailed narrative about the issue of fractures of the ribs in asylums which started following what Wallis has dubbed “the broken rib scandal” of 1870.\(^{552}\) More frequent post-mortems were mandated partly as a way of clarifying doubts this scandal had raised about the causes of death in asylums. In RD’s case, there was a lengthy description of the fractures, and the firm conclusion was that the fractures were old and hence pre-dated admission


\(^{551}\) Atheroma is degeneration of the walls of the arteries caused by accumulated fatty deposits and scar tissue, leading to restriction of the circulation and a risk of thrombosis.

\(^{552}\) Wallis, “The Bones of the Insane,” 196-211.
to the asylum. It is impossible to say whether this was true or whether the alienist carrying out the post-mortem was protesting too much on behalf of the attending staff. Given the differences he exhibited from the rest of the group, one could speculate that, after all, his cause of death was no accident. However, while the deaths of RD and JH (described above in 4.2.1) may have been labelled as exhaustion as a way of mitigating or concealing staff abuse, which some scholars suggest was commonplace, we have no way of knowing this for sure. It was clear from the case notes that many of those who died had been very restless, overactive and in many cases excitable and not open to reason. Thus, there is an open question of whether the asylum attendants may have been responsible or at least complicit in patients’ demise. My analysis, however, leads me to be reasonably confident that such cases were uncommon and most certainly not the norm.

Data from the cohort examined in this thesis suggests that the heart is abnormal in a significant number of cases dying of exhaustion, confirming the hypotheses of scholars such as Hood, Jarvie and Adland. However, as Wallis highlights in her book on the body in Victorian asylums, control groups of the general population are needed to help resolve such postulations conclusively. Additionally, we do not know whether these heart problems were longstanding and represented a vulnerability to death when the insanity developed, or the mechanism or mode of death. It seems likely, however, that these heart and vessel changes were linked to the risk of dying when insane. As Adland puts it, exhaustion deaths could be an “expression of emotional problems through a somatic channel.”

4.2.6 Impacts of treatments

As alluded to in my discussion of evacuative and hypnotic medicines in the Introduction, the question is raised as to whether the medication which was widely used in both asylums played a part in the demise of patients with exhaustion. While some authorities had a clear view of what constituted good medicinal practice and some limited evidence to support it, prescribing and dosing were patchy in general settings. Some practitioners used medication a lot and sometimes inappropriately, but others went with the view that there was limited confidence and consensus that newly developed psychiatric drugs provided a real step forward. For instance, Foster argued

554 Wallis, Investigating the Body, 71.
555 Adland, “Review, Case Studies,” 63-64.
that drugs were of secondary importance in the management of insanity and was of the opinion that prolonged use of them caused further toxicity.\textsuperscript{556} In a similar vein, John Ferguson warned against those who might “substitute the insanity of drugs for the insanity of disease.”\textsuperscript{557} Tuke’s view was that “well-ordered arrangements, and the tact of the Superintendent will oftentimes do more to reduce the amount of excitement and noise in an asylum than tons of chloral and bromide.”\textsuperscript{558} More robustly, in 1907, Charles Hill, the President of the American Psychiatric Association, described current drug options as “a pile of rubbish.”\textsuperscript{559} Despite all these strictures, the use of drugs remained widespread but the effects have not been systematically assessed by historians who, as Charles Rosenberg comments, “have always found therapeutics an awkward piece of business. On the whole, they have responded by ignoring it.”\textsuperscript{560} Shepherd remarks that is also difficult to research as there is considerable uncertainty over the concentrations of active drugs administered because of the use of concoctions and tinctures.\textsuperscript{561} The notable exception to this tentativeness is Scull whose overviews of the topic capture the ambivalence of alienists towards medication for the insane.\textsuperscript{562}

The pattern of many cases in this cohort was to be extremely restless and overactive, to be given medication, then to start “sinking” (a commonly used term in these exhaustion case notes) and ultimately to die. So, on one level, there is a \textit{prima facia} case for implicating medications. However, it seems unlikely to have played a major role in the morbid outcome of many cases here. Firstly, there were cases who followed the above pattern and were not given medication at all, and their mode of death has many resonances with accounts of those occurring in the pre-sedative medication era. Secondly, many patients were given medications which were, by the time of the study, well known and whose usage was fully described in the literature of the day. Examples of this include chloral and hyoscine. It is conceivable that these drugs worsened the moribund feeble state which many of these patients exhibited prior to death and this may have been a consequence of large and accumulating doses of these drugs, although actual quantities

\textsuperscript{556} G. W Foster, “Common Features in Neurasthenia and Insanity,” 412.
\textsuperscript{557} John Ferguson, “The Insanity Following Exhaustion,” 438.
\textsuperscript{559} Charles Hill, “Presidential Address: How we can best Advance the Study of Psychiatry,” \textit{American Journal of Insanity} 64 (1907): 6.
\textsuperscript{561} Shepherd, \textit{Institutionalizing the Insane}, 139.
administered are hard to discern in the case notes. It seems unlikely, however, that they were the
direct cause of these exhaustive deaths. Havelock, of Sunnyside Asylum, had a particular concern
about the culminative impact of the newer, putatively more effective, but potentially more toxic,
drugs such as sulphonal and paraldehyde; he advised against these medications.⁵⁶³ These views
were shared by his predecessor, Howden, who Presley notes “felt it unwise in cases of overactivity
to repress or conserve energy whether by mechanical or therapeutical restraint.”⁵⁶⁴ Murdoch of
Fair Mile Asylum was also well known to be conservative in his use of pharmacological agents. So,
in practice, these newer drugs were largely eschewed in both these asylums.⁵⁶⁵ Several patients
were given drugs such as digoxin and strychnine to stimulate the heart’s action and these drugs
have a narrow therapeutic index (meaning that there is only a small gap between the beneficial
and adverse effects of these drugs). However, these drugs were only given when a patient was
already deemed moribund, and their use has the hallmarks of an attempt to overcome the
therapeutic nihilism such cases engendered. Finally, many patients were given alcohol, usually in
the form of beer, which was seen as stimulating, but only in small amounts. Enemas were still
frequently used but their effects are hard to definitively discern. There was no evidence of the use
of emetics in this period of study. The general view of medication held by many alienists at the
time and by subsequent commentators is that they were of little substantial use, but probably not
much harm, and my review of these case notes does not alter that perception.

4.2.7 Clinical states and terminology

The term exhaustion was applied to many inmates on admission and during their stay, particularly
during their last illness. Other terms such as “worn out” or “wasted” are common in the primary
literature and in analyses of case notes. In this study, these descriptors were commonly observed
in the case notes but did not appear as causes of death, whereas terms such as “marasmus,”
“syncope” and “congestion” sometimes did. It is unlikely that these latter terms were describing a
qualitatively different state from exhaustion and scholars agree that all these terms were highly
subjective and impressionistic.⁵⁶⁶ However, these other causes of death were not explored further
and so one caveat to the findings of the current study is that it may not have captured all the

⁵⁶³ Havelock, “Presidential Address,” 104.
⁵⁶⁵ Wheeler, Fair Mile Hospital, 98.
⁵⁶⁶ Samuel Shortt, Victorian Lunacy, 61.
nuances and meanings of the exhaustion narrative. Exhaustion as a cause of death was probably used much more than these other terms because, as Trevor Turner surmises, Commissioners in Lunacy in both England and Scotland accepted it and, significantly conditioned by their wishes, alienists of the period came to rely on “this repetitious terminology.”

A further caveat is that there is a striking lack of patients’ voices in the reports studied here. There is no evidence of asylum inmates themselves reporting the specific complaint of exhaustion. The words they used to describe symptoms are notable by their absence in available records. Roy Porter was one of the most influential amongst Anglophone scholars calling for greater appreciation of the socio-historical contexts which reflect and inform the narratives of those deemed mentally ill. Porter’s perspective was echoed and elaborated on by a range of scholars, including Sally Swartz who, in her examination of South African asylums, emphasises the “ways in which multiple histories are compressed into single psychiatric accounts.”

Bartlett points out the paucity of documents from the inmates themselves and the “vastly divergent explanations” open to official documents. Allan Ingram similarly stresses that medical writings had always been thus and from the eighteenth century rarely reproduced the language of the insane except to “show it for the raving that it was.” As Andrews observes, in his research on the patient’s experience in Glasgow Asylum, “Far from representing patients’ impressions, case notes pre-eminently constitute the impressions of the medical officers who wrote them.” A period in the mid-nineteenth century in the Glasgow Royal Asylum where case notes became more literal and a source of patients’ own words is charted by Andrews, but this was short-lived and was replaced in the later part of the nineteenth century by “evaluative commentary,” whereby the patients’ experience was catalogued into descriptors such as incoherent, confused, violent and indeed exhausted. Andrews reflects on how this later change was associated with an increasing emphasis on a “scientific” approach to mental disorders. In the current work, looking in detail at deaths by exhaustion over a 35-year late Victorian period, these brief clinical descriptors were very evident,

570 Andrews, “Case Notes, Case Histories,” 265.
and the patients’ voice was scarcely heard. So, we must rely on the accounts of their medical attendants which were subjective, impressionistic, and possibly biased.

Questions as to what clinically characterized the patients who died by exhaustion and what their death represents require further scrutiny. The issues surrounding senile mania were outlined above. In the main, cases with this label presented with an illness characterized by cognitive failure, or dementia, as it was increasingly being labelled elsewhere. Such cases were excluded from more detailed case note and post-mortem analysis. Of course, this action is fraught with the dangers of retrospective diagnosis (discussed below and in greater detail in Chapter 2) and the perils of false distinctions but at least this strategy follows a delineation alienists made at the time. As mentioned above, catatonia was increasingly being discussed as a clinical entity. Though arguments raged about its nosology, the possibility that its presence accounted for some of the cases of fatal exhaustion deserves consideration. Fink, Shorter and colleagues have outlined the key points in the historical development of the concept of catatonia. Kahlbaum, coined the term catatonia in 1874 and emphasised the characteristic triad of muteness, rigidity, and stereotyped restricted movements. Despite the familiarity of the symptoms that Kahlbaum grouped together, his disease concept of catatonia elicited a conflict among alienists. Two schools emerged. One view supported Kahlbaum’s proposal of catatonia as a disease in its own right with a generally good prognosis. An opposing view was that catatonia was a complication of different disorders (mood disorders in particular) and not a distinctive disease. The arguments about nosology may have hindered the recognition that catatonia may have been associated with at least some exhaustion deaths although other factors are the syndrome’s complexity and its relative rarity. Fatal variants associated with catatonia were recognised in the late nineteenth century although these were described in greater detail in the twentieth century. For example, in 1934 Stauder described a form of catatonia with an acute onset and quickly fatal outcome. Mann notes that Stauder’s German description was translated as lethal catatonia. Young adults were reported to become suddenly mute, rigid and either stuporous or severely excited. Death occurred with disruption of vital signs (pulse, blood pressure and temperature) and circulatory failure. Shorter

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put forward the idea that lethal catatonia may account for some deaths with exhaustion. His argument has scarcely been cited but Healy’s group agree that a few deaths from exhaustion were, in retrospect, related to lethal catatonia. A summary of the case is given in Appendix J. This case seems to be very similar to those cases described by Stauder. Another variant, Scheid’s cyanotic syndrome, was described by Scheid in 1937 in which acute catatonia was accompanied by cyanosis, fever and tachycardia. This syndrome’s place in psychiatric nosology and its association with death by exhaustion is unclear. This syndrome does not appear to have been present in the current cohort. Post-mortem findings in cases of catatonia in the literature are scanty, as they were in the 19-year-old man from this cohort and cannot adequately explain the death. The one case seen in my cohort of about 50 cases represents a much lower prevalence than that suggested by Shorter and by Healy and colleagues. Debate continues as to the frequency of this disorder but whichever way it is looked at, the syndrome is rare and unlikely to account substantially for the much higher prevalence of death by exhaustion.

Turning now to the issue of whether the cases said to be dying of exhaustion from mania and melancholia had instead a delirium, several complex issues arise, not least of which are the problems, practical and conceptual, of retrospective diagnosis. Most historians agree that the concept of delirium was long mired in complexity, linked as it was to philosophical theories about the body and the psyche. Lipowski comments that delirium was “the Cinderella “of English language psychiatry as it was “taken for granted, ignored and not considered worthy of study.” It was ignored because of its often fluctuating and transitory nature but also because delirium has a predilection for the elderly, often seen at that time to be senile with consequent and inevitable problems. The concepts underpinning the diagnosis of delirium has not been much discussed by historians, with the notable exception of Berrios, who describes how in the nineteenth century the

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574 Mental state was said to be melancholic stupor. He was “oblivious of surroundings, silent and cataleptic and rigid with beads of sweat on his face and voluntary muscular action cataleptic... remaining in whichever posture he is placed in.” See Appendix J for more details.
terms which were applied developed even greater ambiguity.\textsuperscript{578} Delirium is derived from the Latin
\textit{deliro}, “I rave” and came to be used generally and non-specifically for insanity. According to
Berrios, this linguistic issue underpinned delirium’s uncertain place in nosology. The term
“confusion mentale” was introduced in Europe but Berrios persuasively argues that this concept
did not have any discernible impact on the more conservative and pragmatic British psychiatry.
During the second half of the nineteenth century clouding of consciousness became the primary
clinical criterion separating delirium from the rest of the insanities. However, Richard von Krafft-
Ebing (an influential, sometimes controversial, German academic alienist) pointed out that
clouding of consciousness could exist in other mental disorders such as stupor and ecstasy.\textsuperscript{579}
Alienists had been generally aware for centuries that bodily illnesses could bring on mental
symptoms directly (and therefore necessitated different management) but the syndrome’s
importance took a long time to become rooted outside academic circles and delirium remained
mired in a conceptual and semantic muddle in practice. Berrios charts how the separation of acute
organic states from the acute functional states could be made and how scholars of the late
nineteenth century wrote works on this topic but also how these contributions had little impact on
the clinical field. Daniel Hack Tuke provided a definition of delirium in his \textit{Dictionary} in 1892 which
described the symptoms and antecedents of this disorder, including the key observation that it
was reversible, but this work, despite demonstrating Tuke’s usual perspicacity, does not seem to
have impinged on day-to-day practice. Berrios argues that part of the reason for this was that
Tuke’s definition did not summarize 1890s views on delirium well as it did not refer to
disturbances of consciousness which was widely accepted by writers at that time.\textsuperscript{580} So, while
delirium and delirious were terms commonly found in Victorian case notes and papers, it was
almost always used in the “I rave” sense to depict severe mania. It is persuasively argued that it
was not until the work of Lipowski in the 1960s that the diagnosis of delirium gained a firm footing
in psychiatric classifications.\textsuperscript{581}

In the current study, only one case (FC, described in 4.2.1) said to die of exhaustion was positively
identified as a case of delirium. This lady’s underlying illness (probable bacterial endocarditis) was

\textsuperscript{579} Richard Krafft-Ebing, \textit{Lehrbuch der Psychiatrie} (Stuttgart: Enke, 1878).
deemed to exclude her from further case-note and post-mortem study. She had many of the hallmarks of delirium and indeed was frequently referred to as a case of such in the notes. Several other cases had features of delirium (fluctuating levels of consciousness and disorientation and intermittent fever). By and large, these phenomena were understood as late features in someone whose insanity had morphed into a moribund and “reduced” (to use the word frequently applied in the case notes) state. The delirious features may therefore have been secondary to a terminal infection like bronchopneumonia. There were however a few cases in which the mental state on admission was not clear-cut and there were mixed symptoms of both insanity (psychosis) and an organic state (delirium). A few of these cases showed fever. However, in these cases no underlying infection or pathology was determined at post-mortem. This may have been because the underlying physical disorder was too subtle to be recognised at the time, or indeed at all. The ways in which contemporary alienists distinguished between acute mania and delirious mania or mania with delirium needs clarifying as does how the use of these labels impacted on case reports of exhaustion. It is likely there were considerable variations in practice. The jury must therefore remain out as to whether unsuspected and undiagnosed delirium underlaid many or any of the cases described here but the probability is that the contribution of such a process to exhaustion being 4% of asylum deaths (to use this thesis’s conservative assessment) is small.

Jarvie and Hood’s survey of primary reports on exhaustion deaths led them to the opinion that severe melancholia with agitation and stupor were central to the eventual demise of these sufferers. They concluded that an agitated depression often triggered by adversity, rather than mania and insanity, was the forerunner of this syndrome. They claimed that where death by exhaustion in mania had been cited, examination of the cases revealed an underlying syndrome akin to severe melancholia with the restlessness or agitation or stupor. This contention received some support from Harris who considers that the stupor from melancholia was present in many examples of exhaustive deaths.582 The case notes of those labelled as mania who died of exhaustion in the current study’s series were examined to see if this hypothesis had credibility. In my view, it is very difficult to do a “psychological autopsy” on case notes, as Harris and colleagues attempted, given changing language usage and conceptual frameworks. Jarvie and Hood’s proposal was generated from case reports in the literature which are even more removed from

the clinical arena. In any event, in the cases of mania who died with the labelled of exhaustion here, only a tiny handful (four) could be considered as having the features of severe melancholia, although agitation and restlessness were common. Thus, Jarvie and Hood’s hypothesis, built as it is on shaky ground, is not supported and agitated depression does not seem to be a universal syndrome leading to death by exhaustion.

The Victorian diagnosis of acute delirious [sometimes called confusional] mania also needs discussion in this context. In 1890, the Irish alienist Connolly Norman expressed the view that, in what he dubbed “acute confusional insanity,” the admixture of both “maniacal and stuporous” features was associated with a bad prognosis and increased likelihood of death. Norman held the view that there was a continuum of insanity from simple psychosis to delirious mania with organic elements and risk of death increasing across the continuum.⁵⁸³ He quoted Krafft-Ebing who was of the opinion that acute confusional insanity was essentially a condition of brain exhaustion, probably due to anaemia or malnutrition of the cortex.⁵⁸⁴ Norman noted, as others had done before, that this outcome was more likely in those already enfeebled by their insanity, their living conditions or other debilitating illnesses. It is noteworthy that Norman was practising in Ireland where the effects of various famines were particularly marked. Charles Mercier writing in The Lancet was a strong supporter of the reality of acute delirious mania and opined it was one of a very few distinct illnesses: “The clinical picture of acute delirious mania is distinct and prevents it from being confused with any other any other type of insanity... a definite variety of insanity.”⁵⁸⁵ Mercier, however, was an alienist well known for his trenchant views, often out of proportion to the evidence. Subsequent historians have been less convinced by his views of the condition’s position.⁵⁸⁶ There was a prevalent notion that changes in blood were pivotal to this alleged condition. Theodore Deecke, a specialist pathologist attached to the State Asylum at Utica, New York, enunciated the widely held view that the vascular system and/or its contents were central to causing acute insanity by means of toxins of various kinds that were responsible for mental symptoms, such as acute excitement and delirious mania and exhaustion.⁵⁸⁷ In his survey of the field, Shulack summarises the view that a toxaemia induced the inability of blood regeneration and

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⁵⁸⁴ Krafft-Ebing, Lehrbuch der Psychiatrie.
⁵⁸⁶ Turner, “A Diagnostic Analysis of the Casebooks of Ticehurst House Asylum,” 1-70.
capillary toxicity leading to exhaustion and vasomotor collapse. The capillary “toxin” was said to account for the commonly reported congestion and small haemorrhages in this condition. Overall however, the status and cause of this condition and its contribution to exhaustive deaths are uncertain.

The moniker acute delirious mania was applied to the case notes of three patients in the current series. First a man of 52 who died 5 days after admission, but a post-mortem was not performed. The second was a 29-year-old woman who died 6 days after admission. Case notes could not be obtained. Post-mortem revealed congestion in brain, lungs, and liver but nothing else. A summary of the third case, ET, a 19-year-old single female, is given in Appendix I. These three cases all had very short admissions. The clinical features of the third case are typical of a number of reports in the nineteenth century starting with Bell’s seminal description in 1848. Reviewers of this topic describe cases like this throughout the nineteenth and early twentieth centuries but with diminishing frequency. While acute delirious mania was reported as a common concomitant of deaths by exhaustion in the earlier part of the nineteenth century, such reports became rare by the turn of the century and this is mirrored in the current results, where only 2% of the deaths by exhaustion were associated with acute delirious mania. One could speculate that the overall reduction in the frequency of deaths by exhaustion is accounted for by the reduced numbers of these acute delirious mania cases. The demography and clinical course of the second and third cases fit with that of a rare autoimmune encephalitic condition which can rapidly and fatally afflict young women in particular and there are many resonances between the clinical picture manifest by ET and that of sufferers of that condition. However, such speculation would be unwise, given the mysterious nature of this uncommon condition and its uncertain nosological and epidemiological status.

Finally, the link between pregnancy and death by exhaustion requires attention. In the current series, 3 deaths from exhaustion related to pregnancy were found. One case was puerperal in nature, the other two occurred during pregnancy. All were tragic cases as not only the mothers died but, in each case, there was a stillborn child, one near term and the other two at 4 and 7 months of pregnancy. All the women exhibited poor self-care and poor health. The linking of the

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social, bodily and emotional strain of pregnancy and childbirth with exhaustion was a widely held view in Victorian mental science. The deleterious effects of poverty and lack of personal care in cases of puerperal insanity were described by Bucknill and Tuke in 1858. Marland reports that women suffering from puerperal insanity admitted to asylums were badly nourished, even skeletal, exhausted, and weak, their “normally poor physiques further battered by the strains of pregnancy and childbearing.” She shows how other factors were linked to the onset of puerperal mania, “with childbirth sometimes featuring as a mere adjunct to a long line of predisposing factors and circumstances – poverty, financial anxiety, bereavement, and domestic violence.”

The intense emotional state associated with pregnancy and childbirth, the extra burden that poverty and need could place on some women coupled with the demands of motherhood led, in Marland’s words, to the “highly charged set of conditions where body and mind acted upon each other to set a course for disaster and potential death: states of extreme excitability and exhaustion.” This stereotype fits the cases seen here. Like Marland, Susan Hogan similarly highlights how the “insanity of lactation” was thought to be caused by the “debilitating effects of suckling.” In one of the cases reported here, the mother developed a marked mania from which she died while four months pregnant and while still suckling a year-old child.

Irvine Loudon finds that between 1872 and 1876, the number of deaths from diseases or injuries of childbirth in England and Wales totalled 23,051 with 3% of them (573 deaths) attributed to puerperal mania. Marland observes that these rates remained persistently high throughout the nineteenth century. However, in the current study, the number of deaths linked to childbirth is numerically small. The puerperal insanity death died from exhaustion just a few days after admission. Other cases of death with exhaustion endured far longer: Marland describes a case who died 6 months after admission for puerperal insanity from “exhaustive mania,” “being reduced in frame to a mere shadow.” This observation was mirrored in the current data set where two of the pregnancy cases were emaciated at death.

4.3 Conclusions

The notion that the term exhaustion continued to be ubiquitous and uniformly, imprecisely and casually applied in the late Victorian era held by some historians of the topic is challenged by this survey of the literature and these findings. The term exhaustion was utilised a lot when describing death from a number of disparate conditions including epilepsy, tuberculosis and apoplexy and this practice seems to have continued at much the same rate throughout the later decades of the nineteenth century. The use of exhaustion and its synonyms in this scenario represents a descriptor of the mode or style or death. However, when the term was applied to those dying with mania or melancholia, its use appears to have become less common and more precise as the century progressed. Nonetheless, this study found that one in five cases had an underlying physical disorder of a sort that was likely in itself to lead to death. This means that many previous historical analyses, with the exception of Derby’s, have been based on samples that were not of pure cases.\(^{595}\) In the current study, the pathological changes associated with these underlying disorders were identified at post-mortem and sometimes the post-mortem record was amended in line with this, but why changes were not made in the register of deaths (which is more readily accessible to historians) is not clear: in my view, this is more likely to have been a bureaucratic muddle rather than part of any particular intention to mislead.

Examining the cases that were left after the removal of those with underlying physical disease leaves a group that continued to be heterogeneous. In the later part of the century a group of patients whose insanity was labelled senile mania became evident in some asylums and their deaths, often after a period of florid agitation and confusion, accounted for an apparent increase in the frequency of death by exhaustion in mania and melancholia. As argued above, this label was applied, with only a small number of exceptions, to those whose primary problem was related to memory and cognition, and the pathological findings support the contention that these conditions were akin to dementia, this latter label only beginning to be applied to such cases in asylums in this period. More detailed analysis revealed that other causes of heterogeneity were the finding of a few cases with conditions related to mania and melancholia, including one case of catatonia and three cases in the context of pregnancy and childbirth. There were three cases of acute delirious mania, although the nosology of this label was, and is, unclear. However, the contention that

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\(^{595}\) Derby, “Manic-Depressive ‘Exhaustion’ Deaths,” 448.
agitated depression or delirium underpinned a lot of cases was not confirmed. Of course, there is the criticism that to make these distinctions (albeit the main effect of which was to show these alternate diagnoses or syndromes had little impact on the bulk of cases) a retrospective diagnosis is applied. Medically trained scholars writing about mania and melancholia have often done so under the assumption that underlying cultural and temporal differences in language and understanding are not pertinent, and that mental illness is a more or less timeless condition. At the opposite end of the spectrum, historians and literary scholars, critical of presentism, reject any notion of a timeless biological model, arguing that diagnostic concepts are culturally produced and cannot be straightforwardly transposed across time based upon present knowledge. The pitfalls of retrospective diagnosis have been discussed by Bynum and Neve amongst many other historians.596 While decrying the view that medical or psychiatric disorders represent “timeless, ahistorical categories,” they nevertheless argue that it is important to try to understand the clinical nature of the patient population of previous eras. The latter point is the pertinent issue here, not whether it is possible to plausibly diagnose people of the past with today’s diagnostic criteria. It was in this spirit that the cases here were examined with a modern medical eye, strongly conditioned by a contextually sensitive reading.

What led to post-mortems being carried out in asylums in the first place was, in substantial part, concern about whether malpractice accounted for many asylum deaths, but the widening of post-mortem activity also fitted in with the growing belief in the late nineteenth century in insanity’s anatomical and neuro-pathological roots. Here we are concerned with whether exhaustion was employed as a partial mitigation for early and unexplained deaths. The conclusion of this first comprehensive look at this question utilising post-mortem reports is emphatically in the negative, though two rather suspicious cases were found. It must be borne in mind, however, that the information on which this conclusion is based is material written with the intention of satisfying the scrutiny of the LC foremost in the writer’s mind, and with the patients’ voice nowhere to be read.

This study confirmed the acute nature of the insanity in those patients who succumbed to exhaustion. This was the first episode for the majority, and several were apparently well only a few weeks before death. On the other hand, it did not confirm that, for most cases, an acute

stressful event had precipitated these fatal illnesses. Nonetheless, here, echoing Shulack’s conclusions, some cases were found where the stress was acute and apparently yoked to the fatal final illness.597 There seems to be no specificity for the exhaustion outcome in the impacts of these acute stressors, however. Of the two narratives which Smith considered underlay death by exhaustion, the second one of acute severe fatal illnesses seems paramount in the cases described here.598 The premorbid status of such cases has been much discussed by reviewers, but it is noteworthy that not all cases were enfeebled prior to admission. Indeed, as Derby also reported, many were well nourished.599 Perhaps this goes along with general improvements in public health in the study period compared with early- or mid- Victorian times. Some patients were described as having an acute illness with sudden onset but yet were emaciated or poorly nourished on admission, pointing to the likelihood of longstanding difficulties, be they economic, physical or mental. Scholars have argued that many patients were admitted to asylums in a feeble state during the process of dying and died there from inanition, the asylum acting merely as a hospice.600 While this notion may have validity for some patients, it does not seem to apply to many of the cases who died of exhaustion in this study.

During the late Victorian period there was a growing consensus about the clinical features of exhaustion deaths. The explanations for these deaths were very varied, but congruent with prevailing views of insanity’s basis. There was a growing belief at the time, supported by a relatively restricted range of subsequent scholarship, that such deaths were cardiac in origin. The belief was that the heart was subject to dramatic influences from above which it could not sustain. Limited support for this notion was found. Atheroma in the brain and the heart was prevalent in this young (even by the standards of the day) population. In the following chapter it will be argued this pathological finding may be related to stress. Myocardial changes were also commonplace and were often described in terms of softness and friability and degeneration. Whether these changes were primary or secondary will be debated in the next chapter, but their presence in a young population who died by exhaustion seems likely to be associated with their early and rapid demise.

597 Shulack, “Exhaustion Syndrome,” 466-75.
598 Smith, “‘Visitation by God,’” 110.
600 See e.g. Scull, Museums of Madness, 115.
Margaret Harris and colleagues opine that those patients who died by exhaustion (a category they describe as mysterious) may have simply given up. This perspective is to be found in other historical accounts of this topic. Adland, for example, summarised reports of exhaustive deaths in insanity and concluded that it was “psychogenic illness originating in the need for self-annihilation” and that the sufferers “were determined to die.” 601 However, there is no substantiation of these views other than as a post-hoc belief. On the contrary, what I have discovered much more compellingly in the case notes of the patients studied here, is a picture of patients struggling for survival in the face of overwhelming odds which were not of their making.

Larson commented that “the need for research into the cause of high mortality rate in cases of acute psychoses, which is potentially a recoverable condition, has always been very great” and he expressed the hope that such research would “pave the way to materially lower the mortality” in these cases. 602 Such hopes essentially remained unfulfilled. The antecedents and associations of death with exhaustion in the late Victorian period have become more delineated yet the underlying diagnoses and pathophysiological mechanisms remain largely conjectural.

602 Larson, “Fatal Cases of Acute Manic-Depressive Psychosis,” 982.
Chapter 5.  Vascular Disease in the Insane

5.1 Introduction

This chapter looks in more detail at those patients of the asylum who died of diseases of the heart and vessels to explore questions about the factors associated with the development of vascular diseases in the context of insanity. The chapter also assesses the consequences of the combination of vascular disease and insanity. A sample of patients under the age of 65 whose cause of death was ascribed as vessel or heart disease was identified and the clinical and pathological associations of this ascription examined with a particular focus on the nature of the admission illness. The roles of stressors be they acute or chronic, nutritional status and typology of the insanity were considered with an emphasis on those who died at a notably young age. The nature of the pathology in the heart and vessels and in the brain, and the potential link between them, was investigated and the findings considered in the light of the primary literature and secondary commentaries which have reflected on the issues raised.

This enquiry is both needed and timely. It is needed because the research set out in previous chapters showed that vascular disease was a not infrequent accompaniment of death in the asylum. Approximately one in six of all patients who died were given a vascular cause of death (Chapter 2) and a near identical figure ascribed at post-mortem for those younger patients labelled insane with mania and melancholia (Chapter 3). When post-mortem findings were examined in detail, there was an even higher prevalence of pathological change in the heart and vessels in mania and melancholia with a quarter of those who died of causes other than tuberculosis having enlargement of the heart muscle, a third having fatty deposits (atheroma) in vessels and nearly half having degenerative fatty change in the heart. Exhaustion was labelled as the cause of death for about 10% of the insane in this study. Examination of post-mortem findings in these cases showed an even higher prevalence of these vascular abnormalities which were recorded in three quarters of the exhaustion cases (Chapter 4). This survey is timely because there has been a dearth of historical analysis on vascular disease in insanity, despite its relatively common occurrence, and what there is has been rather superficial. A number of commentators
have mentioned heart disease as a contributor to asylum mortality and a few have speculated on the role of disordered emotion in its genesis but there has been very little consideration of the extent or consequences of pathology in the heart and vessels.\textsuperscript{603} Wallis devotes one and half pages of her seminal volume “\textit{Investigating the Body in the Victorian Asylum}” to the topic.\textsuperscript{604} To redress this balance and to investigate both the associations of vascular diseases and their consequences, this chapter investigates clinical and post-mortem records from patients deemed to have died from vascular disease, whilst acknowledging the limitations of available post-mortem information and the retrospective nature of this enquiry.

Many scholars point out that insanity made its sufferers vulnerable to other diseases, echoing Andrews’ account of Pritchard who, in 1835, had “affirmed the dangerous susceptibility of the insane to congestive diseases of the bowels, liver, heart and intestine” and “lunatics’ peculiar propensity to various disorders profoundly deleterious to organic life.”\textsuperscript{605} In Victorian asylum studies, there is general agreement between historians on inmates’ poor physical health both at admission and during incarceration. For example, in the mid-1870s, 25% of all cases in one Scottish district asylum were either “epileptic or syphilitic;” tuberculosis was found in 20-30%, while heart and lung disease was commonplace, with many dying from heart conditions and strokes.\textsuperscript{606} Deaths from cardiovascular disease and stroke were quite common in each of the three York asylums in the 1880s, even in relatively young patients aged 20 to 40 years.\textsuperscript{607} Wallis describes the commonly held view amongst Victorian alienists that mental phenomena such as irritability were linked to heart disease and that, because the behaviour of asylum patients “appeared to offer an exaggerated version of these states”, “circulatory problems” and “degeneration” of the heart were apt to be prevalent among asylum patients.\textsuperscript{608} However, secondary studies, on the whole, have not tackled the issue and the need for proper comparisons with the community prevalence of such changes so as to judge more fully the validity of such assertions.

\textsuperscript{604} Wallis, \textit{Investigating the Body}, 70-1.
\textsuperscript{605} Andrews, “’Of the Termination of Insanity in Death’,” 131.
\textsuperscript{606} Doody, Beveridge and Johnstone, “Poor and Mad,” 891.
\textsuperscript{607} Renvoize and Beveridge, ”Mental Illness and the Late Victorians,” 23-4.
\textsuperscript{608} Wallis, \textit{Investigating the Body}, 70.
There are a few contemporary accounts on this topic which are relevant. Prominent among these are those of Cecil Beadles, Assistant Medical Officer at Colney Hatch Asylum in London, even though his 1895 report of widespread and frequent changes in the heart and vessels in the insane received little acknowledgement at the time or comment in subsequent histories. The 1885 Goulstonian Lectures on “Insanity in Relation to Aortic and Cardiac Diseases” by Julius Mickle, Medical Superintendent of Grove Hall Asylum, London, had similarly little impact. Mickle described a series of post-mortems in which degenerate muscle of the heart, of pale or dull appearance, was found in all (my italics) cases. In 1901, Drapes suggested that post-mortems of asylum death statistics showed that deaths from heart disease were twice as common as in the general population but a detailed study of the age structure of these respective populations (necessary for calculating an SMR) was not performed. Upon finding arterial degeneration and hypertrophy of the heart in several asylum post-mortems, Thomas Duncan Greenlees, an Ayrshire born alienist working at Garlands Asylum, Carlisle, offered an explanation for mental disease which boiled down to the heart being restricted in its ability to work to its fullest capacity, with the brain receiving less nourishment as a result. However, as Wallis stresses, a control group from the general population is needed before such assertions can be accepted fully. She cites the work of J. Wilkie Burman, Assistant Medical Officer at West Riding Asylum, who, despite initial enthusiasm for the importance of heart disease in insanity, finally concluded that it was “scarcely more common in the West Riding Asylum that in the West Riding of Yorkshire generally.” Another problem lies with the interpretation of these observations. As Blum points out, making inferences about causality from the presence of irregularities is a speculation after the fact. Blum argues that there can be no demonstration that an irregularity causes bizarre behaviour or other consequences since there can be no controlled observation of the onset of either. The problems in ascertaining whether the frequency of cardiovascular deaths and of vascular pathology were different in the asylum population from the general population are a consequence

611 Drapes, “Phthisis and Insanity,” 667-78.
614 J. Wilkie Burman, “Heart Disease and Insanity,” *West Riding Lunatic Asylum Medical Reports* 3 (1873): 255.
of little systematic enquiry into the former in either setting and almost no work on the latter. The
difficulties encountered in both contemporary and modern-day accounts of the prevalence of
vascular disease in the asylum and the frequency of death therefrom, is a reflection (and probable
amplification) of similar difficulties in assessing these parameters in the non-asylum population.
Morgan (a pathologist from Westminster Hospital, London) is clear that, although ischaemic heart
disease was present in the nineteenth century, it is not possible to say how common it was
because of the different descriptors used in life and on death certificates. He concludes, part
tongue in cheek, that all that can reasonably be said is that if the various heart-related causes of
death are lumped together, there was “quite a lot of it about.”616 Vascular diseases became more
commonly used terms in death certificates after the turn of the century but, as Campbell and
Robb-Smith argue, this apparent rise is related both to the increasing longevity of the population
and to modifications in nomenclature and mortality statistics.617 A further complication is that the
number of patients attributed with vascular heart disease in that era may have been confused
with an unknown number of patients who may have had other causes of heart problems, such as
syphilitic aortitis.618 This thesis does not attempt to provide an estimate of whether there was or
was not an increased prevalence of heart disease or artery pathology in the asylum population.
Instead, it seeks to examine what form vascular disease took in the asylum and the relationship
between vascular disease and the features of the insanity.

There are several detailed histories of the development of concepts of diseases of the heart and
circulation, but they are mostly penned by physicians and generally lack analysis of the social or
historical context of the times.619 Principally they are accounts of the main discoveries which
highlight the personal contribution of eminent physicians and pathologists. As Jarcho remarks,
these histories are compendiums rather than analyses and serve mostly as useful reference guides
to medically orientated historians, “those more interested in pathology than in mythology.”620

(1968): 356.
617 Maurice Campbell, "Death Rate from Diseases of the Heart: 1876 to 1959," British Medical Journal 5356 (1963):
618 Evan Jones and Evan Bedford, "Syphilitic Angina Pectoris," British Heart Journal 5 (1943): 107-20; Paul Klemperer,
619 Joshua Otto Leibowitz, The History of Coronary Heart Disease (Berkeley, CA: University of California Press, 1970);
Frederick Arthur Willius and Thomas Jan Dry, A History of the Heart and the Circulation (Philadelphia, PA: Saunders,
620 Saul Jarcho, Review of The History of Coronary Heart Disease, by Joshua Leibowitz, Bulletin of History of Medicine
46 (1972): 413-4.
However, one clear lesson from all these accounts, is the long lag between the development of knowledge and the derivation of concepts in academic centres and their dissemination into routine clinical practice. Jarcho stresses this process was both difficult and slow. The diffusion of such knowledge to alienists in semi-rural asylums is likely to have been even more patchy and drawn-out. An account of the “discoveries” in cardiovascular disease is therefore not germane to addressing the issues outlined above. Rather, it is important to judge the ascribing of causes of death and the pathological reports in asylums against the prevailing teaching and training of the time, while taking into account the underlying views and biases of those performing and reporting the post-mortem.

Before outlining the data accrued on this topic, an account of some specific terminology and what it was thought to represent at the time is necessary to facilitate an understanding of the findings. The most important of these terms is fatty heart and the linked term morbis cordis which was the cause of death ascribed when fatty heart pathology was seen as central to the patient’s demise. In this context, the work of Sir Richard Quain (1816-98) constitutes a good starting point. Quain, an eminent Irish doctor working in London, later as a Royal Physician, described the myocardial changes of “fatty degeneration” in a classic paper of 1850 as when “the myocardium is transformed into soft, fatty tissue, which presents a pale, dirty-yellow tint.” Later, Morgan showed convincingly that the fatty changes that Quain had observed were ischaemic in origin. Quain’s paper included a large series of cases of sudden death. In many of these deaths there were cardiac symptoms with fatty degeneration and coronary artery occlusions being found at post-mortem. Quain found that the clinical features associated with fatty heart included a languid, feeble circulation, breathlessness, syncope, rupture of the heart and sudden death. Quain’s “fatty heart” terminology infiltrated everyday language and became a commonplace and, according to Billing in 1852, “fashionable complaint.” Fatty heart came to be as familiar but also as ominous to the lay public as coronary thrombosis is today. East cites George Eliot’s *Middlemarch* in which Doctor Lydgate, called to a patient with sudden shortage of breath, diagnosed fatty degeneration

622 Jarcho, Review, 413.
624 Morgan, “Some Forms of Undiagnosed Coronary Disease,” 347-9. Morgan examined the clinical and pathological material of 83 cases from Quain’s paper which had been preserved in formalin and convincingly shows that 52 of them had the pathological hallmarks of ischaemic heart disease.
of the heart and warned that death from this disease was likely and likely to be sudden. Bedford delineates how fatty heart represents an epoch in the changing conceptions of myocardial disease and in changing diagnostic habits. He describes fatty heart as having “an evil reputation” and documents the space devoted to it in standard textbooks on heart disease from 1850 onwards as evidence of the importance attached to fatty heart by physicians in this country, Europe and America.

However, despite the widespread awareness of fatty heart, its link to coronary artery atheroma was only appreciated in academic centres. In 1837, Bizot had observed that fatty lesions were found in about one in three deaths under the age of 60 associated with ossification of a coronary artery more commonly on the left side. However, even towards the end of the nineteenth century, Dr Samuel West, a physician at St Bartholomew’s, London, observed that the coronary arteries were frequently overlooked in routine post-mortem examinations. Medical historians have also stressed the limited recognition and/or prevalence of cardiac morbidity and mortality associated with atheroma. As Finlayson and Morris stress, citing Gibson’s contemporary account, despite atheroma being relatively commonplace finding at post-mortems in academic centres, there were very few cases with acute cardiac symptoms. Michaels argues that ischaemic heart disorders became more common towards the end of the nineteenth century, largely as a result of demographic changes, which included a growth in the numbers of people over 45, particularly in the middle and upper classes who were thought to be more prone to these conditions. However, it was not until the early twentieth century that what became called coronary thrombosis (and later myocardial infarction) became both frequently seen and a commonly used term.

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Thus, heart disease was relatively common in the nineteenth century and there was evidence of its presence in the asylum insane. Without attempting to answer the question of how common it was in the asylum, this thesis explores, *inter alia*, both vascular causes of death and vascular pathology amongst the younger functionally insane. This chapter takes a specific approach, examining those deaths in asylums where these vascular problems were, at least at face value, the predominant problem, i.e. those purportedly dying of a vascular cause of death. The sample and their characteristics will first be described followed by an account of the pathological findings in the heart, great vessels and brain. The relationship between clinical factors and vessel pathology will be examined as will how the pathological changes may be interlinked. The findings are to some extent surprising, and therefore their plausibility will be tested by looking at the broader literature which includes studies of both the insane and the non-insane.

5.2 Methods, Findings and Commentary

5.2.1 Characteristics of cases

The periods for study here are the same as that in Chapters 2 and 3 (in Sunnyside, 1892-1901 and in Fair Mile 1895-1906). Selected for scrutiny are those cases of mania or melancholia whose cause of death was deemed to be vascular (i.e. those whose cause of death was ascribed to the heart or vessels). From the samples described in Chapter 3, there were 17 such deaths under the age of 55 in Fair Mile and 10 in Sunnyside. The sample was expanded subsequently by studying all those dying with such causes of death in these periods at or under the age of 65. Post-mortem records were scrutinized, and this led to the addition of 13 cases from Fair Mile. There are therefore a total of 40 such cases examined here. Details of the demography of this sample can be found in Table 5.1.

633 It was intended to carry out a similar exercise within the Sunnyside records, but the Coronavirus pandemic precluded this.
<table>
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<td>4:6</td>
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Table 5-1 Age, gender distribution and clinical label of cases with a vascular cause of death.

The mean age of death of this cohort was in the early fifties. There were 10 with a notably young age of death (at 26, 27 and 34 years with 7 in their early 40s). Therefore, a quarter of this vascular cohort had very premature deaths by the standards of those reaching adolescence (i.e., surviving infancy) in this era. As found with the overall admissions to (and deaths in) the asylum, mania predominated compared to melancholia with a ratio of 2.6:1. There was also a female to male ratio of 2.6:1 which is higher than that found for asylum admissions of that period, for death statistics for these asylums as a whole (as reported in Chapter 2) and for the younger cohort whose post-mortem findings were discussed in Chapter 3 (where the ratio was 2:1). The female preponderance in the vascular deaths was also much greater than that of those who died of exhaustion (Chapter 4) where it was only slight. Of the 33 cases in which it was stated, about half the cases (16) who died of vascular causes were married but the remainder were single or widowed. In terms of occupation, the largest category was manual labourers (12); there were only five cases who could be deemed as skilled. Females showed an interesting profile. 11 cases were described as housewives (mostly of manual labourers) but four females were described as having no job. This demographic profile of the female deaths (unskilled and often alone) fits in with research that shows that sufferers of vascular disease tend to be disadvantaged and often economically and personally stressed.634 Melling, in his study of Exminster Asylum, also highlights this demographic pattern. He comments that an absence of a family member appears to have been an important consideration in the admission of many females who were less likely than men to be from an extended household.635 Walton also shows that lack of kinship was a major driver to asylum incarceration, particularly in economically deprived females.636 Many younger women in

the asylum had been badly treated either by their families or in service and how they were often
deemed to be “incapable of useful employment.”\textsuperscript{637} Unmarried women may have felt
marginalised, lonely, frustrated and unfulfilled possibly leading to insanity.\textsuperscript{638} Life circumstances
were very much to the forefront of thought in Victorian times as documented by MacKinnon. He
describes how it was thought by some that a sinful mode of life was a main aetiological factor in
vascular conditions and that lives full of tension, grief and possibly guilt were very prone to these
conditions.\textsuperscript{639} It is argued below that some of these vascular deaths were precipitated by the
frequency of uncontrollable life events and complex social ills experienced by sufferers and the
evidence suggests that this stress was even more marked for females diagnosed with insanity.

For the majority of these 40 cases, death occurred in their first admission. In the 30 in which the
episode number was made clear, it was the first episode for 20 and the second episode for 9.
However, there was less evidence that these episodes were precipitated by acute stressful events,
as was seen more frequently where death was by exhaustion, nor were the episodes that led to
admission so acute and hectic. A summary of the mental states of this cohort is to be found in
Appendices K and L. In general, the length of episode before death was longer for vascular rather
than exhaustive deaths. Despite being less acute, for the main part admission accounts were of
florid psychosis, with most patients exhibiting well marked delusions, often of a religious nature or
delusions of control by external agencies often accompanied by frequent hallucinations. However,
the psychopathology seen in this cohort was broadly similar to that reported for the patients
described in Chapter 3 who died of non-vascular causes. The melancholic patients all exhibited
clear-cut delusions which were congruent with their mood. What was noticeably absent from the
clinical notes were accounts of vascular symptoms during the terminal episode. No patient
appeared to have had an acute or painful cardiac event. Even those with clear-cut apoplexy
changes at post-mortem did not have what could be described as a clinical stroke documented in
their notes. There were a few who died following a syncopal attack and a few sudden deaths
occurring out of the blue, but these were in a small minority.

\textsuperscript{637} Melling and Forsythe, \textit{The Politics of Madness}, 191.
\textsuperscript{638} Edward Higgs and Amanda Wilkinson, “Women, Occupations and Work in the Victorian Censuses Revisited,”
\textsuperscript{639} Alistair MacKinnon, “The Psychopathology of Ischaemic Heart Disease,” \textit{The Journal of the Royal College of General
Practitioners} 16 (1968): 253. See also Don Carlos Peete, \textit{The Psychosomatic Genesis of Coronary Artery Disease}
(Springfield, IL: C. C. Thomas, 1955).
The question arises whether drugs given to apparently help the patients’ compromised vascular systems may have in fact hastened the demise of these sufferers. Feil has written an insightful history of the treatment of heart disease in the nineteenth century. He observes that, despite fundamental research in some centres on drugs such as nitrates for angina and digitalis for irregular pulse in valvular disease, in clinical practice treatment of heart disease developed slowly and was in a “primitive state” in 1900, akin to that of the late eighteenth century. A few patients in this cohort had medication given to apparently help the heart, like digitalis, and some were given drugs like strychnine to stimulate the heart’s action. These particular drugs have a narrow therapeutic index (meaning that there is only a small gap between beneficial effects and severe adverse effects) and a case could be made that prescribing of these drugs was haphazard in an institution where medical treatment was not the primary focus or experience. However, these drugs were only given occasionally and for short periods usually when the case was already moribund. Their use has the hallmarks of an attempt to overcome the therapeutic nihilism such cases engendered. Many patients were given alcohol, usually beer, which was seen as stimulating to the heart in small amounts. Jones (Superintendent of the LCC Asylum at Claybury) was one of the influences behind this practice. There was no evidence of the use of emetics, but enemas were still frequently used though the effects of them, positive or negative, are hard to discern. It was very difficult to identify the names and doses of many of the medications prescribed. What is available is to be found in Appendices K and L but the issue of the effects, positive or negative, of medications for the heart cannot be explored further here.

As discussed in Chapter 2, one of the main explanatory models offered by scholars is that the frequency of asylum deaths soon after admission was high as a direct consequence of the weak and parlous physical state of those admitted. To examine this issue in this cohort, the length of stay prior to death was divided into five periods and the data are shown in Table 5.2.

641 Jones, “Prognosis in Mental Diseases,” 1578-83.
It can be seen from Table 5.2 that the phenomenon of death very early in admission to both asylums was rare in the vascular cohort. This contrasts with the large cohort with a range of causes of death reported in Chapter 3, where 18% died within four weeks of admission and half of those within a week. The relative scarcity of deaths in the first month from vascular causes also contrasts with deaths with exhaustion where it was a commonplace occurrence (see Chapter 4). Apart from this difference in deaths within the first few weeks, the pattern of deaths in this vascular cohort was similar to that shown in Chapter 3. Overall, two fifths of this cohort died in the first year in the asylum and only a quarter had been resident for more than five years at the time of their demise. Thus, the presence of vascular disease in the context of insanity did not frequently lead to sudden death but also mitigated, at least to some extent, against long asylum stays.

As described in Chapters 3 and 4, an assessment of nutritional status on admission and at death ("General appearance" at post-mortem) was carried out. The body habitus essentially fell into two groups, either emaciated (sometimes qualified with words like “very” or “much” or, less often, “a little” or “rather”) or well nourished. The findings are shown in Table 5.3.
The nutritional status remained the same from admission to death except for one case where the patient was well nourished on admission but emaciated at death. As can be seen, approximately two thirds of these vascular cases were well nourished at death, in sharp contrast to the narrative which usually surrounds the topic of asylum deaths, that death is a consequence of a heady brew of insanity, infection and deprivation, all leading to malnutrition and terminal cachexia. This finding also contrasts with Chapter 3’s finding that nearly two thirds of those dying of non-vascular causes were emaciated at death. This leads to the general conclusion that malnutrition is not an invariable feature of death in insanity and that vascular deaths in insanity have a different pattern and natural history than the remainder of the asylum mortality.

5.2.2 Cause of death

The certified cause of death was taken from the pathological records. A few examples of post-mortem reports from this cohort are reproduced in Appendix A. In the rare instances where two causes were given, only the first was examined. The cases deemed to be vascular were subdivided into five categories based on this information. The breakdown of these causes of death in the two asylums in age categories is shown in Table 5.4.
Table 5-4 Recorded vascular causes of death (placed into 5 sub-categories) in mania and melancholia in both Asylums combined. *Other includes congestion of lungs (2 cases) and 1 case each of cardiac disease, syncope, and pulmonary haemorrhage and gangrene.

Cardiac failure was not reported as a cause of death from Sunnyside Asylum, whereas this label accounted for nearly a quarter of causes of death at Fair Mile Asylum. This difference suggests a divergent approach to the interpretation of post-mortem findings in these establishments. Cardiac failure is not conventionally a pathological cause of death but a clinical syndrome and, in this sense, the Sunnyside post-mortems may be more pathologically correct. Morbis cordis as a cause of death was also much more common in Fair Mile, while apoplexy and valvular heart were relatively more common causes of death in Sunnyside. However, the sample is too small to make much of these differences, other than flagging up something to be looked out for in future studies.

Before going on to examine the individual categories of cause of death and what they can tell us about asylum mortality, the post-mortem reports of this sample were surveyed in more detail to quantify three specific vascular pathology findings: fatty change in the heart muscle (myocardium), hypertrophy of the heart ventricles and atheromatous change in vessels. There were many reports that the heart was soft, and this was commonly accompanied by a qualifier such as “and friable”
or “and fatty.” *Morbis cordis* and/or fatty heart was reported as a cause of death on its own but the pathological changes that underpinned such a cause of death ascription were also found where other causes of death had been ascribed, such as apoplexy and cardiac failure. The pathology reports were scrutinized for these pathological changes irrespective of cause of death ascription and it was found that fatty heart or one of its synonyms was present in 26 of the 40 cases (65%), including 3 from Sunnyside. Some of the analyses below have utilised this combined pathological finding group. There were multiple accounts (n=19, 47%) of the heart being hypertrophied (larger in size). Myocardial hypertrophy is a relatively “hard” sign and its finding generally indicates cardiovascular pathophysiology. Sometimes only the left ventricle was described as hypertrophied and rarely only the right ventricle: these variants have been lumped together. In this era, the arteries within the heart were often described as “atheromatous” or sometimes “calcified” or “calcareous.” Dr Samuel Black suggested, in 1797, that angina pectoris was caused by “ossification of the coronaries” and put forward the reason it was often not so reported was that these arteries laid “buried in the substance of the heart” so the pathological changes might be missed by even the most “accurate dissector.”

Rokitansky noted that when the myocardium showed “dirty yellowish discoloration” and “became loose and flabby” then “very frequently” (my italics) there was “ossification of the coronary arteries.” It was not clear whether the vessel findings here were assessed by the coronary arteries being cut or simply observed from the heart cavity. Atheroma in the aorta (sometimes referred to as “commencing atheroma of the aorta”) was also a frequent finding in this sample. Finally, the basal arteries of the brain (principally the basilar and middle cerebral arteries) can be visualised clearly at post-mortem. The account of them in the post-mortem reports was restricted to either “atheromatous” or normal and this was documented. A category was created to include those where atheroma (or one of the above equivalents) had been reported either in the brain or the heart vessels or in both. It was found that atheroma occurred in 31 of the 40 cases (78%), usually, but not always, in multiple sites. This category was called “any atheroma” and used to carry out further investigation of the clinical and pathological associations of atheroma.

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The cause of death data and the above pathological groupings were examined to compare those dying in mania from those dying in melancholia. The results of this comparison can be seen in Table 5.5 below.

<table>
<thead>
<tr>
<th>Cause of death/Pathological finding</th>
<th>Clinical label</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mania n=29</td>
</tr>
<tr>
<td>Apoplexy</td>
<td>5 (17%)</td>
</tr>
<tr>
<td>Valvular Heart Disease</td>
<td>3 (10%)</td>
</tr>
<tr>
<td>Morbis cordis (fatty heart)</td>
<td>12 (41%)</td>
</tr>
<tr>
<td>Cardiac failure</td>
<td>6 (20%)</td>
</tr>
<tr>
<td>Other</td>
<td>3 (10%)</td>
</tr>
<tr>
<td>Fatty heart pathology (n=26)</td>
<td>18 (62%)</td>
</tr>
<tr>
<td>Any atheroma (n=31)</td>
<td>20 (69%)</td>
</tr>
<tr>
<td>Hypertrophy of heart (n=19)</td>
<td>16 (55%)</td>
</tr>
</tbody>
</table>

Table 5-5 Comparison of mania and melancholia cases with vascular cause of death ascription and presence of specific pathological change in the vascular system.

As can be seen, there were some differences in the proportions of causes of death between the two groups but because the numbers in each of the categories is small, it is unwise to overinterpret these differences. However, what was striking was the frequency of fatty heart changes and atheroma in both mania and melancholia with them being present in about two thirds of all cases. There was a particularly striking association between melancholia and atheroma with the latter being found in all such cases. The association between melancholia and atheroma has long been recognised by contemporary commentators and historians, and has been accorded a wide range of explications, ranging from psychological and behavioural to inherent and
biological. However, it is not clear if atheroma is a cause or an effect of melancholia. Before
discussing what the strikingly high prevalence of these pathological changes (little remarked upon
previously) might tell us about asylum mortality, the findings in the brain, and their link to these
vascular pathologies, will first be demonstrated.

The post-mortem reports from these vascular causes of death were scrutinised using the same
methodology set out in Chapter 3 to identify the most commonly reported brain changes. The
data for both mania and melancholia and for both asylums combined is laid out in Table 5.6 below
and shown in relation to the accounts of vascular pathology recorded. Here, those with atheroma
in cerebral vessels as well as those with any atheroma and/or fatty heart or hypertrophy are
examined in Table 5.6.

It can be seen in Table 5.6 that about half of these patients had atheromatous change reported in brain vessels, about two thirds exhibited fatty changes in the heart and the vast majority (34 of the 40, 85%) had one or other or both abnormalities. Brain softening was seen in about half of the patients, the large majority of whom had fatty heart pathology and/or evidence of atheroma, particularly atheroma in cerebral vessels. This suggests that brain softening may have been caused or worsened by the presence of atheroma in either the heart or brain. Atheromatous change in vessels is likely to be associated with disruption of the blood supply with either ischaemia (shortage of blood) or haemorrhage (bleeding). Several eminent nineteenth-century pathologists wrote about the association between brain softening and changes in vessels and speculated about

<table>
<thead>
<tr>
<th>Pathological change reported in the brain</th>
<th>Number of reports (out of 40 cases)</th>
<th>Pathological change reported in vascular system</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>With atheroma of cerebral vessels (n out of 19 reports)</td>
</tr>
<tr>
<td>Brain softening</td>
<td>21</td>
<td>14</td>
</tr>
<tr>
<td>Shrunken grey matter or cortex</td>
<td>17</td>
<td>14</td>
</tr>
<tr>
<td>Oedema of white and/or grey matter</td>
<td>18</td>
<td>11</td>
</tr>
<tr>
<td>Congestion and/or hyperaemia</td>
<td>13</td>
<td>7</td>
</tr>
</tbody>
</table>

Table 5-6 Pathological findings (using closely equivalent terms) in post-mortem brains of deaths in mania and melancholia who died of a vascular cause and their association with vascular pathology markers.
its probable ischaemic or haemorrhagic origin. A broadly similar pattern of association was seen for shrinkage of the brain’s gyri. However, vessel atheroma and/or fatty heart pathology is neither necessary nor sufficient to cause such changes in the brain, as some cases with atheroma did not show brain softening or shrinkage, while a few cases without any reported atheroma exhibited such pathology in the brain. This relative specificity of atheroma to changes in grey cortex matter (softening, shrinkage) contrasts with the situation with the other reported brain changes, oedema and congestion, where was no strong relationship with atheroma. These findings were just as likely in the cases with and without atheroma and/or fatty heart. This fits in with the view that congestion and oedema (swelling) are vaguer and less reliable findings. This may also lead one to infer that doctors performing these examinations felt under pressure to find and report something, perhaps anything, in cases incarcerated with insanity.

A case which shows many of these features was that of GJ, a 57-year-old gateman who was married with 10 children. He had been “living comfortably” but more recently had not worked for the past six months, though he had never been in the workhouse. He was described as being temperate and of good behaviour but always a worrier. GJ began to feel despondent, refused food and was fearful that he would do his family harm. He tried to kill himself by hanging and on admission was “actively suicidal.” He looked depressed, complained that he had “no stomach” and that his “back passage was blocked by a body that comes down from his chest.” Physically GJ had marked arcus senilis. His heart was described as weak, and his pulse was of “poor tone” and his vessels were said to be thick. For many months, he exhibited poor sleep and limited food intake and was hypochondriacal being “very deluded about his viscera.” GJ had recurrent attacks of bronchitis with “great cardiac embarrassment” before losing strength and dying. At post-mortem he was emaciated. The brain was soft, and the convolutions thinned. The grey matter was hyperaemic and the white matter congested, soft and oedematous. Basal cerebral vessels were atheromatous. The myocardium was soft, pale and fatty and there was atheroma in the first part of the aorta. His left ventricle was dilated and hypertrophied. The lungs and liver showed evidence of congestion. Thus GJ, possibly related to losing his job when he had so many dependents, became severely depressed with evidence of nihilism and psychosis. He had evidence of cardiac

646 Arcus senilis is a lipid infiltration of the peripheral cornea of the eye appearing as a yellowish-white ring around the cornea. Quain regarded it as indicative of fatty degeneration of the heart but not to atherosclerosis in general. It has an association with ischaemic heart disease, particularly in the under 60s.
disease on admission and at death, despite being relatively young, had widespread severe atheroma affecting vessels and the heart at post-mortem with changes in the brain, such as softening and shrinkage, which this study has found were associated with these vascular abnormalities. Several similar case reports, occurring in both melancholia and in mania, can be found in Appendices K and L which provide summaries of the clinical and pathological reports of this group.

Three of the cases in the vascular cohort died of what was called valvular heart disease. All of them exhibited pathological changes similar to those described in textbooks of the time for rheumatic heart disease. Bedford, a cardiologist and historian, has penned an astute history of rheumatic heart disease. Its first pathological description was by Matthew Baillie, John Hunter’s nephew and disciple, in 1799 and that the first case series was published by Wells in 1812. Bouillard, in 1840, demonstrated the direct relationship between preceding rheumatic fever and the subsequent development of rheumatic heart valve pathology. None of these cases here had a history of rheumatic fever documented but this may be easily overlooked. In fact, the only patient in this cohort who did have a history of rheumatic fever did not have lesions on her valves at post-mortem. There is no reason to suppose that this kind of death was any more or less common in the asylum setting than the community.

There were seven cases whose cause of death was ascribed as apoplexy with an average age of death in their early 50s. One case, that of GH, probably died of a violent head injury as described in Chapter 3. The majority had clear cut evidence of a cerebral haemorrhage, all had evidence of atheromatous vessels and most exhibited atheroma in the heart with fatty change in the myocardium as well. Baillie is credited with showing that apoplexy was often related to haemorrhage from damaged arteries which either fed the brain or were inside the brain. He described “bony or earthy material being deposited in the coat of arteries” which thereby “lose a

part of their contractile and distensible powers.”  

Four of the six deaths from apoplexy had marked hypertrophy of the heart. Quain, in 1849, had observed the frequent association of cardiac hypertrophy in patients dying of apoplexy. It is conceivable that systemic hypertension (high blood pressure) was a common mechanism of that link but, of course, Quain could not measure blood pressure and so could not make any link. It is noteworthy that in this series there were several cases, in both the mania and melancholia sufferers, who exhibited softening and shrinking of the brain and hypertrophy of the left ventricle. In the early twentieth century, the German alienists and pathologists Alzheimer and Binswanger named this clinical picture “arteriosclerotic” brain atrophy and others subsequently showed that one cause of these neuropathological changes, often associated with hypertrophy of the left ventricle, was hypertension. Thus, it can be speculated that a component of the brain changes observed in this project may have been a result of elevated blood pressures that, of course, in the periods under study here, would have gone undetected.

5.2.3 Vascular deaths under 45 years of age

There were ten deaths in this category. The youngest was HH, a soldier, who died at the age of 26. He had been promoted to the rank of lance-corporal in his early twenties but at 24 he became suddenly insane and was labelled as having mania. He had insanity in both sides of his family. HH had numerous delusions. He tried to strangle himself and later tied a ligature around his penis on delusional grounds. HH died suddenly and this was ascribed to cardiac failure. At post-mortem he had abnormalities in the cardiac valves and evidence of heart failure so was most probably a case of rheumatic heart disease. The next youngest case was GH who was said to have died of apoplexy at the age of 28 but, as argued in Chapter 3, there is good reason to believe she died of a non-accidental injury and not a stroke. MK died of heart failure aged 34. She had had an attack of typhoid some years earlier but although these symptoms remitted, she remained weak and died of cardiac failure. The post-mortem examination in her case was limited and apart from her heart being dilated nothing else abnormal was noted. The remaining seven cases were all in their earlier 40s and all had evidence of fatty change in the heart and atheroma of coronary and/or cerebral

vessel. All but two were female. While little is known of 2 of these 7, for the remaining 5 there was evidence of stressful events, such as the recent death of a child and a recent birth, and 2 of the females were socially isolated.

Particularly for females, the above analysis shows a potential link between vascular disease in the insane and adverse life circumstances and stress. This brings into focus commentary by various contemporary observers and modern researchers on the interplay between emotions and heart disease in the population at large and in those with a label of mental illness.653 This commentary is important because it brings into sharper relief the zeitgeist that was prevailing at the time both physicians and alienists were performing post-mortems and reflecting on their findings. Leibowitz observes that “strain and stress of life... and mental disposition” have been repeatedly thought to be associated with coronary artery disease over the last two millennia.654 Fay Alberti, in her account of the narratives of the relationship between the heart, the brain and the soul, highlights a shift of the heart from being considered the centre and site of emotion to its identity as “the barometer (rather than the instrument) of emotional experiences.”655 Kirstie Blair argues, that the heart lends itself such interpretations “constantly blurring the boundaries between cultural supposition and apparent medical fact.”656 Ryle and Russell, in their 1949 history of this topic, show that the concept of stress’s association with heart disease began to become firmly rooted in the nineteenth century. They note that Heberden, who first published a description of angina in 1802, mooted that "disturbance of mind" was not to be ignored as a possible aetiologial factor in coronary disease.657 Kowal shows that at the beginning of the nineteenth century the French physician Corvisart emphasised that prolonged states of tension (“ever-renewing causes”) was pathogenic to the heart, but how the focus subsequently changed to the role of acute emotional states like anger in the genesis of heart disease.658 Despite obvious cardiac morphological changes, the conviction arose in the minds of many physicians that heart disease was often the result of an emotional disturbance. For example, Latham, from St

654 Leibowitz, The History of Coronary Heart Disease, 165.
656 Kirstie Blair” 'Proved on the Pulses': Heart Disease in Victorian Culture, 1830–1860,” In Framing and Imagining Disease in Cultural History, ed. George Rousseau et al. (Basingstoke: Palgrave Macmillan, 2003), 285.
Bartholomew’s Hospital in London, was clear that “death has followed mental excitement more frequently that bodily excitement.”

Kowal describes how the majority of clinicians “selected rage as the most important emotional state connected with angina pectoris, although the elated state of joy was not neglected” and how anger was seen the most dangerous precipitating factor of heart symptoms and sudden death. However, Leibowitz identifies a later shift so that “the most acceptable and weighed opinions are those who attached less importance to sudden excitement or anger but to “successive... stress situations”. He comments on the increasing influence of the views of Corvisart. Leibowitz concludes that the bulk of the literature of the nineteenth century shows that coronary events could occur where no evidence of stress was evident but that it appeared more frequently in those with chronic and maladaptive stress reactions, usually when chronic stress or adversity had been evident for some time. MacKinnon underlines the Victorian view that many with heart disease were depressive who seem to live driven lives (“unable to stand and stare”) and exhibited “patterns in self-destruction.”

These conclusions chime with other commentators’ views on the topic and the current findings. Levine-Clark delineates how asylum case histories often reveal that indigent women had experienced “dysfunctional domesticity,” with evidence of many pressures, as well as poverty, grief over the death of friends and family, or love and marital relationships gone wrong, sometimes violently. The presence of these factors is also commented on by other scholars, such as Melling and Cathy Smith.

All this evidence supports the conclusion reached here that a vascular disease death was more common in those patients, particularly female ones, whose insanity was in the context of chronic domestic and economic disadvantage.

Despite this evidence, Ryle and Russell’s survey of the topic concludes that the following factors combined to increase the risk of heart disease: -

“(a) maleness on account of the occupations which it entails; (b) the exacting character of sustained mental work accompanying intellectual occupations and posts involving heavy responsibilities; (c) ageing, through mounting cares and the longer exposure to stress; (d) the emotional tensions that frequently, on the one hand, accompany business, professional and intellectual life and

659 Peter Latham, Lectures on Subjects Connected with Clinical Medicine (London: Longmans, 1846), 413.
interruptions to it, and, on the other, are expressions of it and (e) the inheritance of an ambitious or conscientious personality pattern.”

It is noteworthy that very few of these factors, many of which applied mostly to the middle classes, were discernible in the very different cohort under study here. This may indicate that heart disease in the mentally ill was not considered in the various studies summarised by Ryle and Russell. There is some common ground in that Ryle and Russel cite repeated accounts of emotional disturbance or prolonged anxiety as having a continuing adverse influence on the arteries and the heart. They conclude that “sustained mental over-activity is another form of ‘disturbance of mind’,” which must be considered as capable of affecting the coronary circulation and eventually of causing vascular damage. Protracted emotional dysregulation and psychological stress factors, exacting and distressing living conditions and socio-economic deprivation appear likely to be operating in this and other asylum cohorts.

The relationship between emotional dysregulation and vascular disease dominates the history of this topic. However, some scholars place additional emphasis on a range of other factors linked to diet and nutrition. Acierno, for example, observes that there was a prevalent belief that structural vascular changes were associated with chronic malnutrition during the nineteenth century but shows that it was not until the twentieth century that these changes were characterised. In brief, as demonstrated by Gallavardin in 1900, malnutrition and cachexia are associated with both dilation and hypertrophy of the heart and with minute patchy fibrosis and fatty degeneration of the myocardium. However, as shown in Table 5.3, only a relatively small proportion of the patients in this cohort (or indeed the larger cohort described in Chapter 3) were emaciated and the fatty degeneration appeared far from patchy. Thus, malnutrition is unlikely to account in a significant way for the fatty changes seen here. Campbell argues, based on tailoring records, that obesity was more prevalent than has generally been considered in the nineteenth century, particularly in the middle classes. Obesity (recorded only in a vanishingly small number of patients in this study) was also associated with fatty infiltration of the heart but a range of pathologists, including Laennec in 1838, Quain in 1850 and Herxheimer in 1901, repeatedly

664 Ibid, 387.
666 Sarah Campbell, "Waists, Health and History: Obesity in Nineteenth Century Britain" (PhD diss., Oxford University, 2014).
observed that the fatty accumulations in the hearts of the obese were pathologically different in appearance from fatty degeneration of the myocardium. According to Acierno, a similar situation to that of both malnutrition and obesity applies to alcohol excess but he supplies no supporting evidence for this claim. Bedford observes that alcohol was often mentioned as a cause of fatty heart and that the condition was common in publicans and gin drinkers but there appears to have been no specific study of this carried out. Moreover, intemperance was very rare in this cohort so alcohol misuse is unlikely to be a major confounder of these findings.

5.2.4 Current findings in the context of the literature

The finding of widespread atheroma and vessel disease in the cadavers of young patients with insanity with apparent links to pathology in the brain and to the patient’s untimely demise might be considered as an unusual or surprising finding and perhaps one that lacks plausibility in the context of the apparent absence of conventional symptomatology associated with vessel disease. For these reasons the history of the understanding of atheroma and its symptoms in the community, and what little there is of this in mental illness, will be explored to put these findings in context. Thompson observes that atherosclerosis is a disease that has plagued mankind for millennia with its pathological hallmarks being identified retrospectively in preindustrial populations suggesting that the disease is an inherent component of human ageing and not necessarily linked to specific diets and lifestyles. Since post-mortem examinations began to be performed more frequently in the eighteenth century, the specific anatomical findings of atheroma were recognized and the first published use of the term to describe changes in vessels was by the Swiss anatomist and physiologist Albrecht von Haller (1708-1777, Professor at Gottingen) who used it in a description of an aneurysm of the aorta in 1755. John Morgagni (1682-1771) of Bologna and Padua, seen by many to be the founder of pathological anatomy, demonstrated sclerosis of the coronary arteries and called attention to patchy fibrous regions in the myocardium. He also denoted the marked fatty myocardial changes which later were shown to represent myocardial ischaemia. The French surgeon and pathologist Jean Lobstein introduced

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668 Bedford, "The Story of Fatty Heart," 27.
670 Leibowitz, *The History of Coronary Heart Disease*, 77-81.
the term “arteriosclerosis” in his unfinished *Traité d’Anatomie Pathologique*, a four-volume treatise on pathological anatomy, published in 1829, to describe arteries with atheroma that had hardened.671 In the mid-nineteenth century, the noted pathologist Rudolf Virchow used the term atheroma to describe visible swellings of the arterial wall and suggested, in 1860, that inflammation played a primary role in their development.672 Another school of pathology, that of Carl von Rokitansky in Vienna, Austria, also described cellular inflammatory changes in vessels with atheroma but considered these changes were blood borne. Virchow revolutionized medical thinking about vascular disease by firmly putting the emphasis on changes in the vessel wall rather than in the blood (even if he had to found his own journal before his papers on this topic could be published). He strongly criticized Rokitansky and the Viennese Medical School for their dogmatism and support of outdated humoralism (the “crass theory of the Viennese school”). Rokitansky must have come to see that his theory did not stand up: the humoral disease theory included in the first edition of Rokitansky’s pathology textbook *A Manual of Pathological Anatomy* was eliminated entirely from the second edition.673 The historian Schiller called this a victory of “solidism over humoralism.”674 To describe all these changes in the arterial wall, Virchow revived the term “arteriosclerosis”, first used by Lobstein in 1829.

Despite these developments in the understanding of atheroma, eighteenth- and nineteenth-century accounts of the clinical manifestations associated with it were much less clear cut. Notably, a discontinuity between the presence of atheroma and arteriosclerotic changes in the vessels and the presence of clinical ischaemic heart disease was evident for all of this time. Despite new pathological insights in the eighteenth and early nineteenth centuries, clinicians were ignorant of myocardial necrosis secondary to obstructive coronary artery disease and more preoccupied with fatty change and degeneration of the heart (which was often attributed to chronic myocarditis). Quain was foremost among the exponents of “fatty degeneration of the heart” and this concept dominated thinking for the next few decades. He stated that these changes corresponded to the region supplied by a coronary artery which was “ossified” but he did not point out any mechanistic link between his two key findings. For reasons that are unclear but

671 Jean Frederic Lobstein, *Traité d’Anatomie Pathologique* (Paris: Levrault, 1829). The synonymous term atherosclerosis also came to be used frequently after this time.
may be related to his personality, Quain also did not acknowledge others’ work on this theme.\textsuperscript{675} Finally, in 1880, Carl Weigert definitively described the condition of myocardial infarction and its correlation with disease of the coronary arteries which caused ischaemia and fatty myocardial change. He therefore recognized the aetiological role of coronary arteriosclerosis in a clinical syndrome, and he distinguished the effects of myocardial ischaemia, fibrosis and fatty degeneration linked to gradual and chronic ischaemia from necrosis caused by abrupt ischaemia. He also laid to rest the notion that the fashionable chronic myocarditis was the cause of the myocardial fatty degeneration, an idea that had held sway for several decades of the later part of the nineteenth century.\textsuperscript{676}

Despite the research summarized above, heart angina of effort was rarely mentioned in the late nineteenth century. George Balfour (Physician at the Edinburgh Royal Infirmary) had seen only two cases in Edinburgh Royal Infirmary and scarcely mentioned the condition in an introductory analysis of 200 cases of heart disease in 1876.\textsuperscript{677} The famous physician and pathologist Osler had not seen a single patient with heart angina before becoming a Fellow of the Royal College of Physicians of London in 1910.\textsuperscript{678} The absence of many descriptions of angina or acute heart pain leading to sudden death in the nineteenth century, could be because it was infrequent, or else because physicians lacked the clinical acumen necessary to recognize it. Most historians of cardiovascular disease consider the former explanation to be much more likely. Michaels argues particularly persuasively on this point, citing numerous well written descriptors of other conditions from earlier periods.\textsuperscript{679} One of Edinburgh Medical School’s renowned physicians, George Alexander Gibson (1854-1913), published his widely acclaimed textbook \textit{Diseases of the Heart and Aorta} in 1898 which encapsulated many of the concepts and advances of the time and similarly downplayed angina and myocardial ischaemia.\textsuperscript{680} It is likely that Gibson’s teachings were influential for many of the alienists who carried out the post-mortems described in this study, as


\textsuperscript{677} George Balfour, \textit{Diseases of the Heart and Aorta} (Philadelphia, PA: Lindsay & Blakiston 1876).

\textsuperscript{678} William Osler, \textit{Lectures on Angina Pectoris and Allied States} (New York: Appleton, 1897).


\textsuperscript{680} Gibson, \textit{Diseases of the Heart and Aorta}, 652-744.
many of them trained in Edinburgh or Glasgow. Between 1901 and 1910, deaths in England and Wales attributed to angina pectoris averaged 842 a year despite it by then being described in standard textbooks of the time.\(^{681}\) Thereafter, however, there was a rapid rise in the clinical manifestations of coronary atheroma (angina and coronary thrombosis).\(^{682}\) Notwithstanding the contribution of many clinician scientists, it was James Herrick (1861-1954), Professor of Medicine at Rush Medical College, who is credited with the classic clinical and pathological description of coronary thrombosis in 1912 and to whose name the “discovery” is attributed.\(^{683}\) A few years later, he added information to the picture from electro-cardiological records. Toward the end of his career, Herrick himself became a well cited historian of the topic.\(^{684}\)

These shifts in nomenclature and in the prevalence of both the pathology and clinical manifestations of heart and vessel disease are relevant to understanding my findings of widespread vessel atheroma in the insane in the absence of manifest cardiovascular symptomatology. An explication for this discontinuity is to be found in the work of Morris and Finlayson.\(^{685}\) Morris (a physician from the Social Medicine Research Unit of Medical Research Council) demonstrates that symptomatic coronary artery disease, along with related diagnoses like coronary thrombosis, coronary occlusion, and myocardial infarction, emerged at the time of the first World War and that the SMR of these conditions follows a steep rise since that time. This is an important finding which has important connotations. As Morris points out “if the increase is real, in whole or in part, it presents a crucial problem for social medicine... for causes may be discoverable in changing ways of living.”\(^{686}\) He emphasizes that whilst occlusion and coronary thrombosis have become common during the twentieth century, there was no evidence of any corresponding increase in the underlying coronary atheroma which was a common finding in post-mortem material from the nineteenth century. Morris based this conclusion partly on the work of the pathologist and historian of pathology Rodney Finlayson. Finlayson carried out an historical survey of post-mortem reports from St Bartholomew's Hospital in the City of London dating back


\(^{682}\) Leon Michaels, "Aetiology of Coronary Artery Disease," 258-60.


\(^{685}\) Finlayson, "Ischaemic Heart Disease," 151-168; Morris, "Recent History of Coronary Disease," 1-7.

\(^{686}\) Morris, "Recent History of Coronary Disease," 1.
to 1868, supplemented by records of post-mortems performed at the London City mortuary at the direction of the coroner. Ischaemic heart disease had been labelled under several earlier morphological diagnoses and Finlayson cites Steven’s list of “calcareous atheroma, myomalacia cordis, chronic myocarditis, fibroid disease of the heart, degeneration of the muscles of the heart, fatty heart, cardiac necrosis, cardiac abscess, rent or rupture of the heart” as some of the options. Finlayson finds that the prevalence of severe coronary atheroma had not increased, indeed had slightly diminished, over the period of his study (1868-1982) but that coronary artery symptoms and deaths had rocketed from about the 1910s onwards. This brings into question the “almost tacit assumption” that a surge in the intensity of atherosclerosis and its causative factors was responsible for the massive increase in clinical coronary heart disease during the twentieth century. The prevalence of severe atherosclerosis of the aorta appeared to show little change over the period of Finlayson’s study (1868-1982) although this analysis presupposes that the term "severe or very severe atheroma" meant much the same to a prosecutor of 1890s post-mortems as it did to their more modern counterpart. MacKinnon, a general practitioner, however supports Finlayson’s conclusion based on a study of coroner’s records for Ripon and Kirkby from 1855-1926 and 1981-83. He concludes that the evidence suggests “as far as it is possible to come to a confident finding in a retrospective assessment of this kind” that acute coronary deaths are influenced by factors different from those influencing coronary artery atheroma. Gorringe, in a masterly, if discursive, overview of this topic, comments that if the concept of separating acute heart attacks from coronary atheroma is accepted, some other facts that have appeared contradictory can be reconciled. For example, the conclusion of Rose and Marmot that historically mortality from ischaemic heart disease was positively correlated with poverty, whereas atheroma was positively correlated with affluence and the wealthy’s ingestion of rich foods,
becomes less paradoxical in the light of this dissociation.\textsuperscript{691} It is thus highly plausible that atheroma was commonplace before the 1900s and yet also to agree with Michael’s contention that “angina pectoris first made its appearance in the late eighteenth century and remained exceedingly rare for the next 150 years with coronary thrombosis being virtually non-existent.” All this fits with my findings, that while atheroma was common in the insane, specific symptoms related to the heart or vessel disease were rare.

Some Victorian and Edwardian alienists explored the vascular system of the insane at post mortem in some detail. While they were very much lone voices, their work uncovered remarkably similar findings to my own and supports the idea that widespread arterial disease is a feature of a significant number of the insane, without other apparent risk factors such as smoking and obesity, if it is specifically looked for. In 1895, Beadles observed widespread clinical signs of arterial degeneration that he considered were manifest in “a large percentage of the insane” but his report received little comment at the time or acknowledgement in subsequent histories.\textsuperscript{692} Beadles had Balfour’s strictures in mind, as he quotes the latter’s remark on the condition of the brain in insanity, that “little dependence can be placed upon records... seeing that what maybe to one observer a deviation from the normal standard will to another be perfectly healthy.”\textsuperscript{693} Nonetheless, he found frequent “prominent and rigid arteries” and signs of heart hypertrophy and dilatation at post-mortem which he considered led to “the feeble pulse, tachycardia, and syncopal attacks to which lunatics are liable.” These signs and symptoms were also present in my study (see 4.2.5). Similarly, as found in the current study, the myocardium in many of these cases was soft and flabby, “with signs of degeneration” and the aorta often displayed “early or advanced atheroma.” He found these features, particularly among recent and acute cases of insanity, as also found here. Beadles observed that while heart disease was a more frequent accompaniment of mania than of melancholia, the proportion of cases of cardiac functional disorder was greater in the latter condition. Both these findings were replicated in the current study. Beadles commented that melancholics with heart disease often exhibited “strange delusions, such as the workings of an unseen agency, as electricity, within them.” Interestingly, four of the current vascular cohort exhibited delusions about electricity although all of them were labelled as suffering from mania. A further parallel between Beadle’s study and the current one is that he found atheroma “where


\textsuperscript{693} Balfour, “Pathological Appearances Observed in the Brains of the Insane,” 49-60.
death has occurred early from exhaustion of acute mania or melancholia,” presaging the findings in the exhausted cohort reported in Chapter 4. Furthermore, Beadles found that the arteries at the base of the brain often showed evidence of degeneration: “Their walls are often thickened or opaque, and frequently rigid with earthy deposits.” 694 He found such changes in just over half cases. It is noteworthy that Frederick St. John Bullen, in a scarcely quoted analysis of the morbid changes exhibited in 1,565 brains of lunatics, found similar changes in basal brain vessels, albeit at the lower rate of a quarter of the total, a figure which mirrors, almost exactly, the frequency found in the current study. 695 Beadles reflected on whether the changes he found were primary to or secondary from insanity. He commented approvingly on a paper by Fred M. T. Skae (Assistant Physician and Pathologist, Stirling District Asylum, Larbert) who believed that the disease of the heart, blood, and blood-vessels which he found in the insane give rise “to some obstacle in the proper supply of the blood to the brain, cause deficient oxidation and result in a fatty degeneration of the cells followed by vacuolation.” 696 Beadles also concluded that the “diseased condition of the arterial system precedes the nerve degeneration and does not result from it.” On the other hand, he did not discount the idea that the vessel changes might be secondary to “the strain thrown upon the heart and vessels from the restlessness, excitement, and violent exercise of the patients.” 697 Ultimately, however, Beadles came down on the side of the prevalent toxaeemia theories of insanity, concluding that there was “poisoning” of the nerve-centres from a failure of the heart and kidneys to remove “effete matters” (he cited “alcohol and syphilitic poison” as potential factors) from the blood. 698

Thus, the largely ignored findings of Beadles resonates closely with that of the current study. The work of Mickle also provides an interesting comparative lens onto the current study. Mickle was highly thought of according to Mott, an eminent pathologist, who wrote Mickle’s obituary in 1918 in which he characterised him as a “pioneer.” 699 In his Goulstonian Lectures, Mickle described a series of asylum post-mortems in which degenerate muscle of the heart, of pale or dull appearance, was found in all (my italics) cases. 700 The coronary arteries, particularly the left one,

were noted, in two-thirds to be “more or less atheroma, or whitish, or whitish-yellow opaque thickenings, or nodules and calcified patches”, again paralleling the findings here. Mickle commented that several patients showed delusions of persecution or delusions of bodily injuries. In some, the alleged injuries were of an extraordinary kind, like tortures which the patients attributed to the malpraxis or malevolence of medical men. Of course, one might wonder whether or not this was a misattribution. He also described four cases of melancholia with these vascular changes who exhibited “delusions of evil impending or poisoned food and with self-abasement or refusal of food,” thus giving a description very similar to that seen in the current cohort (see Appendices K and L and Chapter 4). The parallels with the current findings are strong but important divergences in both findings and their interpretation emerge. Mickle also described similar changes to the above in four cases of dementia, and in three cases in the demented stage of general paralysis, which raises issues about the specificity of his findings. Mickle also asserted that “fatty heart is not always an unmixed evil; indeed, in some cases it may be a preservative lesion, for when the arteries are diseased it may be safer for the subject to have a feebly-acting heart than a normally strong and vigorous one.” He proudly mentioned that Sir William Jenner had taught this same point, that a fatty heart diminished the risk of apoplexy but both Mickle and Jenner’s conclusions are out of step with my findings and my synthesis. In summary, while there is a close approximation between the post-mortem vascular pathological changes, and their clinical associations, in the insane in these Victorian studies and the current retrospective one, what remains elusive is the mechanism and direction of causality of these findings.

In this study, an association between cerebral softening and brain shrinkage and both atheroma of vessels or atheroma combined with fatty disease of the heart was found. This association was recognised early in the nineteenth century by pathologists, but this association has not been explored in the insane and has been little attended to in the secondary literature. A link between these brain changes and hypertrophy of the heart was also demonstrated in the vascular cohort. These associations are even stronger than those discussed in Chapter 3, where the

701 Ibid, 580.
702 Mickle failed to mention that, according to Bedford’s account, Jenner was met with “shouts of derisive laughter” at the Pathological Society of London when he put this contention forward, (Bedford, "The Story of Fatty Heart," 27).
703 Marc De Hert, Johan Detraux, and Davy Vancampfort, "The Intriguing Relationship between Coronary Heart Disease and Mental Disorders," Dialogues in Clinical Neuroscience 20 (2022): 31-40.
development of theories about cerebral softening and brain shrinkage and the possible role of disrupted blood supply in their genesis were explored. These arguments will not be rehearsed again but it is important to note that, whatever the direction of causality between these sets of variables is, there were significant impacts on sufferers when both these sets of problems co-existed. The current results also highlight that vascular disease was not generally taken into account when alienists wrote about the brain in the Victorian period under consideration here, nor has it been by historians looking at their work. Thus, at the very least, vascular disease may be a variable that accounts for a lot of the discrepancies within and between such accounts.

An association that may have been part of the reason that young insane were prone to vessel and heart disease is the link between infection and heart disease. This link is even more evident in those with deprivation and stress, both factors common in this cohort.705 Not only were the insane more likely to suffer economic hardship, but commentators also show that they were more prone to infections, particularly those with chronic debilitating conditions. Leonard Smith describes the proneness of asylum inmates to infectious diseases which, as Ernst points out, were all conditions that particularly affect those already suffering from a fragile state of health because of other illnesses, including their insanity.706 Szreter argues that industrialization and rapid economic growth in the late Victorian period were disruptions which caused social deprivation leading to disease, often infectious, and death for the most unfortunate and marginalized individuals.707 However, we have to be cautious in such interpretations and bear in mind McCarthy’s stricture that many investigations into the lives of the poor are a “genre-crossing blend of statistics and sentiment, empiricism and emotionalism.”708 Deprivation per se is an unlikely specific cause of the high prevalence of vascular lesions in the current cohort. Although many cases were in economic hardship, a substantial number were in reasonable circumstances, and some were relatively comfortable. The combination of stress and recurrent infection provides a more plausible explicatory model, however. An explanation of vessel atherosclerosis and related heart abnormalities, that first Virchow speculated about, and that some other nineteenth-century pathologists accrued evidence for (as Krishnaswamy and others have summarised), is that it was

707 Szreter, "Industrialization and Health," 75-86.
the consequence of an inflammatory process, which may have been, in turn, a consequence of frequent and unrestrained infections and/or stress. Inflammation is thus a particularly plausible mechanism for vessel pathology in the insane, as recurrent infections were common, particularly in overcrowded asylums, whilst, at the same time, such sufferers often endured social disadvantage, the stress of illness and then the loss of agency that that entailed.

5.3 Conclusions

Chapters 2-4 of this thesis have all shown that, in addition to the toll that infections took on the insane in the asylum, vascular disease in the form of atheroma in the vessels, particularly in the heart, was prevalent. It was found amongst those with all sorts of conditions in (Chapter 2), in those patients with mania and melancholia who died prematurely (Chapter 3) and in those who died with exhaustion (Chapter 4). The cohort studied in this chapter had vascular disease as the cause of death, so presented an opportunity to examine this combination of conditions in a more isolated way, in purer culture. The cohort was, as might be expected in a retrospective enquiry into an asylum population, a bit of a rag-bag. Some died of what appears to be rheumatic heart disease and others of problems that were unclear and uncertain, perhaps then, certainly now. However, the vast majority had atheroma in vessels of the heart and/or brain and/or the changes of fatty heart, which was considered by late nineteenth-century authorities to be ischaemic in origin and certainly so by subsequent commentators. Some died of apoplexy, seemingly related to vessel disease with an unknown contribution from undetected high blood pressure. One oddity here is the lack of symptoms suggestive of a stroke in the case notes, in cases where the pathology of apoplexy was clear cut at post-mortem. Others became weak or died of syncope and this was a common outcome of a degenerative process in the myocardium whose hallmark was fatty deposits whose origin, as postulated at the time and shown in subsequent studies, was ischaemic. These inmates did not have the signs and symptoms of an acute heart attack but, as I have shown, such clinical events were rare at the time of this study, not starting to become the epidemic they became until at least ten years later.

It is argued that the atheroma prevalent in the insane was brought on, in variable fashion and to variable degrees, by some of the problems of insanity, in particular uncontrollable stress, chronic domestic and socio-economic disadvantages and the increased likelihood of infection, but not directly by others such as malnutrition, emaciation or intemperance. Tuberculosis and, to a lesser extent, other infections, were major killers of the young insane in asylums in their own right but so too was vessel disease. Vessel disease was not as prevalent as infections and it was certainly not as easily visible to either alienists, the public or indeed to historians. It is argued that this, often overlooked, vascular disease is linked to shrinkage and softening of the cerebrum in insanity and partly explains, or, at least, contributes to the early and frequent demise of the insane in the asylum, its female patients in particular.
Chapter 6. Conclusions

This thesis has contributed significant new perspectives to existing scholarship on asylum mortality. It has also lent partial endorsement to some of the conclusions of other studies, whilst at times offering important modifications, qualifications and adjustments. Key questions for future research are also posed. In her 2012 article on deaths in Victorian asylums, Cathy Smith remarks that such “deaths in numbers posed... questions” and that “the continued susceptibility of insane patients to death could neither be fully explained by medical science nor, in the end, significantly reduced by medical or state provision.” 710 These questions remain outstanding, but the current work, in my view, provides mechanisms which have explanatory value and raises new hypotheses to test.

Smith also highlights how statistics on mortality rates became one of the key metrics by which asylums came to be judged as failing by the Victorian public, viewed as part of an increasingly dismal prognosis for the insane. In his earlier influential writings, Scull enlarged on this negative view, contending that deaths early in admission showed that dying people were sent to the asylum to be nursed to their death and that asylums had become “the resting place for the broken-down and physically decrepit.” 711 Other commentators have perceived asylum mortality in a more mitigatory light, more sympathetic to the challenges asylum practitioners faced. Some have been more willing, as this thesis tends to argue, to see mortalities as in part a consequence of florid, untreated mental illness and something that was unfortunate but unavoidable. For example, Melling’s survey of Exminster Asylum finds that men had a high death rate, with many dying prematurely, despite being on the whole in good or fair health on admission. 712 However, whichever of these viewpoints has the more credence and support, most observers would agree with Smith’s plea for further evidential underpinning and enquiry on this topic and this case study of two asylums’ mortality profile has aimed to provide that. My doctoral research was undertaken to analyse and explicate patterns and causes of death in Victorian asylums dispassionately,

710 Smith, “‘Visitation by God’,” 113.
711 Scull, Museums of Madness, 192.
released, as Murphy puts it, “from the imperative of chasing Foucault’s shadow,” but mindful of the social and economic issues of the time and the place.  

Historians of asylum mortality have put forward several theories as to the cause of the increased and premature mortality of the asylum insane. Some of these have been supported by this work but others cast into doubt.  

Firstly, it was found that it was imperative to drill down into the mortality data to reduce its heterogeneity, a strategy that has been rarely followed in previous studies. The average age at death in these Victorian asylums was in the mid-50s. However, this is a potentially misleading number, made up as it is of deaths in disparate conditions – the final average figure dependent on the makeup of the sample at a particular juncture and locality. In this study, the average age at death was shown to be derived from three components: i) a population made up of those with disorders where insanity and/or behavioural disturbances were secondary to an underlying predominantly neurological disorder such as GPI, epilepsy or idiocy, who had a young average of death in their 40s; ii) an older group, usually called dementia but sometimes senile, who had a much more advanced age at death; and iii) those who were deemed to be primarily suffering from insanity and had an average age of death in their mid-50s. There were very different clinical and pathological associations of demise in each grouping. It is argued here that future work should ensure that these groups are studied separately and comparatively, rather than examining asylum deaths overall which most previous studies have done. This study focusses on the third group and demonstrates that, on average, these sufferers had reduced longevity of about 15 years, compared to an estimate for the rest of the population, confirming this finding which has been made by both primary and secondary sources. Since this figure is an average, a substantial number of the insane died much younger than their non-insane counterparts. This thesis confirms and adds substance to the observation that mortality in insanity was much more likely to occur in the early weeks and months of admission. To explain this observation, most historians consider that poor physical health and emaciation on admission to the asylum

716 Jones, “Prognosis in Mental Diseases,” 1578-83; Cox, Marland and York, “Emaciated, Exhausted, and Excited,” 500-24; Melling and Forsythe, The Politics of Madness.
were the most powerful factors. This is perhaps best exemplified by Scull’s contention in *Museums of Madness* outlined above.\textsuperscript{717} Szreter posits that the dramatic transformations associated with industrialization in the Victorian era impacted on health because of the intensely disruptive nature of this change, which caused social deprivation leading to disease and death for the most unfortunate and marginalized individuals, of which the pauper insane are a paradigmatic example.\textsuperscript{718} However, while this latter contention may have validity on a population-wide basis, it does not have significant explanatory power in the asylums analysed in this thesis. The current study showed that a good proportion of those admitted into these semi-rural asylums who subsequently died had or had previously good jobs, were in reasonable physical shape and were well nourished. These observations chime in more with the findings of Walton on Lancashire asylums and Melling’s wide-ranging studies of Devon asylums, than with those of Szreter or Scull (who has sometimes characterized asylum inmates as a heavily economically compromised, conveniently discarded and displaced, societal flotsam and jetsam).\textsuperscript{719} A different view expressed by some historians is that premature mortality was a direct consequence of overcrowding and the poor sanitary conditions within asylums which produced infections and epidemics that were mainly responsible for the frequent early demise of many inmates.\textsuperscript{720} However, this thesis argues that asylum mortality is not directly or fundamentally yoked to such environmental factors. Evidence from this study and other work demonstrates that there is no necessary association between the total numbers in an asylum and its mortality rate.\textsuperscript{721} The datasets used here do not support the contention that mortality of the insane was a direct consequence of either overcrowding or malnutrition or, at least, that neither operated in a major way.

So, if environmental factors, both outside and inside the asylum, are not the direct or main causes of the deaths of the insane in asylums, what other factors seem to have been operating? It is demonstrated here that the severity of the mental illness and the immediate consequences of that florid state were the key drivers of early and premature death by inducing a vulnerability to disease. For example, respiratory infections, particularly tuberculosis, were a common cause of demise, especially in melancholia and, for the most part, appeared to be secondary to severe

\textsuperscript{717} Scull, *Museums of Madness*.
\textsuperscript{718} Szreter, “Economic Growth, Disruption, Deprivation, Disease, and Death,” 693-728.
Insanity. It is argued that mortality in mania and melancholia was linked to the uncontrollable stress of illness and the consequent incarceration with associated loss of agency. Despite the efforts of staff to get people discharged or to isolate them once infected, the asylum was undoubtedly an unhealthy breeding ground for highly infectious conditions such as tuberculosis which led to the demise of about 40% of patients and most of which, I argue, was contracted in the asylum. My findings are consonant with those of Ernst who, echoing Szreter, describes tuberculosis as a "scavenger" disease which significantly preyed on individuals in a weakened state as a result of their insanity. For some patients in this study the final fatal illness followed a sudden emotional shock, an association much vaunted by Victorians and by subsequent commentators, but such circumstances were, in practice, rare. For most, a wide range of potentially stressful factors, be they medical, economic or social, seemed to be operating, often in combination and chronically, to make the insane more vulnerable to severe diseases and death.

The data from this study confirmed the findings of other studies that suicide was not the cause of excess premature mortality with only two cases being identified in this large survey. Deaths from violence, or mistreatment, perpetrated by staff, both medical and nursing, have also been put forward as plausible explanations of the high death rate in asylums. This notion may have had some validity in the eighteenth century or earlier part of the nineteenth century, but reports of violence in asylums greatly diminished in the latter half of the century as a consequence of the greater focus on moral management, the impact of adverse public and professional opinion and the significant amount of bureaucratisation of asylums, so that every untoward incident and use of restraint had to be detailed. In this study only 2 out of 172 deaths in the mania and melancholia sample were identified where violence from staff played a key part, one fairly clear cut, the other more speculative. The former case raised alarm and ultimately fruitless enquiry; the second was not followed up despite a clear flag at post-mortem. What these cases do seem to represent however is a window into a culture where such incidents were downplayed, if not covered up. As discussed in more detail in Chapter 3, there is a possibility that there was some collusion between

724 Shepherd and Wright, “Madness, Suicide and the Victorian Asylum,” 175-96
726 Arieno, Victorian Lunatics, 97-114.
the medical assistants performing post-mortems and the attendant staff (with the exposé of death from abuse being in neither’s interest), but - in the absence of more telling concrete evidence - this must remain conjectural.

This thesis has looked in detail at the causes of death in young patients with mania and melancholia and relied, almost uniquely, on post-mortem records. As my analysis of previous scholarship has clearly shown, these are underused resources but of course far from a gold standard evidentially. Causes of death remain uncertain, mired on shaky ground and subject to many vagaries of time and place and of the individual recording it. Setting this caveat aside for a moment, it was found that two thirds of such patients died from infections and two thirds of those were from tuberculosis. The infections were mainly respiratory rather than gastrointestinal. Epidemics of the latter had ravaged society and institutions in the earlier parts of the century but by the time of this study were largely confined to mini-outbreaks. Almost certainly, such fatal respiratory infections were a consequence of vulnerability induced by the weakened state brought on by mental illness and/or represent a predilection to infection in those with mental illness. Similar high death rates from infection have been documented in severe mental illness in 1920s India. This association is also evident in developing countries and in displaced populations such as refugees. These populations are prone to deprivation and limited access to health care, both of which were realities for the insane of Victorian Britain. The precise extent to which incarceration for mental illness amplified the risk of infections in the insane is unknowable for some respiratory infections but seems to be a factor in the case of tuberculosis. The high numbers of deaths from tuberculosis in a young population are a reminder of the horrors of this scourge and this observation should counter the often-romanticized image of this condition found in some literature from that period. More prosaically, surveys of the prevalence of tuberculosis must take age into account for, as confirmed by this study, it was very much a condition of the young adult and the early middle aged.

Many of the distinctive and original findings of this thesis relate to those sufferers who died of exhaustion and those who died of heart disease, which together accounted for over one third of

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all the asylum deaths, both showing a predilection for younger patients. Addressing exhaustion deaths first, this cause of death has been put forward by some historians as a way for asylum staff to explain away deaths for which no explanation was evident. One of the reasons that post-mortem s were carried out assiduously in asylums was concern about whether malpractice, either by omission or commission, accounted for many asylum deaths. Declaring death by exhaustion, in providing an apparent or plausible medical cause of death, has been commonly considered by scholars as one way for asylum clinical and administrative staff to evade enquiries into potential malpractice and/or negligence.\(^{730}\) Insufficient clinical or pathological evidence was found to substantiate these notions, however. Nonetheless, it must be kept in mind that the information on which this conclusion rests is conditioned by a range of vested professional interests, and also mainly derived from sources in which the patient’s voice is remote, or more often entirely absent.

This study has confirmed the acute and hectic nature of the insanity for many of the patients who succumbed to exhaustion. For the majority, it was their first episode that led to death and several of these cases were apparently well only a few weeks before succumbing. On the other hand, this thesis did not confirm that, for the majority of cases, an acute stressful event had precipitated these fatal illnesses, as posited by some commentators. Some cases were identified where an acute stress was apparent at the onset of the fatal final illness but this was not at sufficient frequency to establish cause and effect.\(^{731}\) The weakened premorbid status of exhaustion cases has been emphasised by some reviewers but it is noteworthy that the majority of cases here and in some other asylum studies were not enfeebled at admission, and, indeed, as Derby also shows, many were well nourished.\(^{732}\) This finding should be seen in the context of general improvements in public health in the study period. Conversely, a few patients were described as having an acute illness with sudden onset and yet were emaciated or poorly nourished on admission, pointing to the likelihood of longstanding difficulties, be they economic, physical or mental. These observations demonstrate that cases need to be looked at in the round and generalizations based on cross-sectional observations can lead to inaccurate and misleading conclusions.


\(^{731}\) Shulack, “Exhaustion Syndrome in Excited Psychotic Patients,” 466-75; Smith, “‘Visitation by God’,” 109-110.

\(^{732}\) Smith, “‘Visitation by God’,” 110; Cox, Marland and York, “Emaciated, Exhausted, and Excited,” 509-12; Derby, “Manic-Depressive ‘Exhaustion’ Deaths,” 436-49.
Other explanations for exhaustion deaths put forward by contemporary observers in the Victorian period were often congruent with prevailing views of the basis of insanity. The belief that death by exhaustion occurred because the heart was subject to dramatic influences from the brain and the mind which it could not sustain was linked to notions of the heart’s underlying fragility as a consequence of some mix of degeneration (in the sense that the insane were of inferior stock), toxaemia and neurasthenia.\textsuperscript{733} Similarly, myocardial changes of softness and friability were commonplace in such cases, terms which historians have associated with strong undercurrents of degeneration theory which was held by many alienists at that time. However, the research conducted here shows that while there was indeed degeneration, this was fatty degeneration of ischaemic origin. Another explanatory thread was that patients who died by exhaustion had simply given up the struggle – a form of death quite commonly depicted in literature in the nineteenth century. This narrative is also found in historians’ accounts, such as those of Harris and Adland who conclude that some of these patients had a “blind drive for annihilation” or “were determined to die.”\textsuperscript{734} However, this thesis has found no evidence for any of these views: they seem to be post hoc assumptions put forward to explain inexplicable deaths.

This thesis has also demonstrated that another, previously little discussed, but highly significant pathological factor impacted on these patients and compromised their longevity, namely arterial disease. Only a very small number of alienists at the time and fewer subsequent asylum historians have discussed the issue of vessel disease and its implications. Analysis of post-mortem evidence showed that between a third and a half of young cases of mania and melancholia who died either with or without exhaustion exhibited clear evidence of atheroma in vessels, particularly those of the heart. Some of these patients died of stroke, others became weak or died of syncope. These are all common outcomes of a pathological process in vessels or in the myocardium, whose hallmark was fatty deposits known as atheroma. The outcome of atheroma in cardiac vessels was fatty heart or morbis cordis, which was considered by some at the time to be ischaemic in origin and so confirmed by subsequent commentators.\textsuperscript{735} This study makes the key observation, found in

\textsuperscript{734} Harris et al, “The Morbidity and Mortality linked to Melancholia,” 3-14; Adland, “Review, Case Studies,” 66.
\textsuperscript{735} Michaels, “Aetiology of Coronary Artery Disease,” 258-64; Morgan. “Some Forms of Undiagnosed Coronary Disease,” 347-9.
two cohorts, that atheroma was associated with evidence of important changes in the brain in the insane such as softening and shrinkage.

The tendency to develop disease of the arteries is thought be either biological or environmental or a combination of both. It is suggested that the atheroma in the vessels of some of the insane was associated, in variable fashion and to a variable degree, with the insanity itself. It is further mooted, in line with some other commentators’ ideas on this issue, that the vessel abnormalities were causally linked to the uncontrollable stress and increased likelihood of infection that were both causes and consequences of insanity. Tuberculosis and, to a lesser extent, other infections, were major killers of the insane in asylums but so too, I argue, was vessel disease. Vessel disease was not as prevalent as infections and it was certainly not as easily visible to either alienists, the public or historians but it is concluded that this, often overlooked, vascular disease is another key explicatory factor underlying the early and frequent demise of the asylum insane.

Our current understanding of the second area of enquiry this thesis addresses, the performance of post-mortems in Victorian asylums - why they happened and what they achieved - is rather patchy, incomplete and under-researched, apart from a few notable exceptions. It is maintained in this thesis that, overall, asylum post-mortems were carried out on a routine, almost industrial scale, basis because they were so mandated by watchdogs and, to some extent, by a perceived need to assuage public anxieties. It is also argued that they served a range of (limited) clinical or academic purposes. There is no evidence that the LC relented in their demands on mandating post-mortems during the Victorian or Edwardian periods. One surmises that, once initiated, the Commissioners found it hard to stand back and reflect and allow any opt out from the process.

One of the objectives of the mandated high post-mortem rate was to throw light on the question of why people with mania and melancholia died frequently and, overall, at a young age. However, there is little evidence that they provided much illumination on this point, the main exception being that post-mortems at this time helped understanding of neurological conditions that could cause insanity like GPI. The high post-mortem rate in asylums should nonetheless also be seen as part of the development of objective scientific enquiry during this era, albeit one hampered by

doubts over the reliability and meanings of cause of death. Unfortunately, the asylum, with its pervasive underpinning agendas, was relatively unfertile ground for such an endeavour.

Supervising authorities saw post-mortems as a method to help identify and, more crucially, prevent violence and non-accidental injury in asylums. In this context, Andrews summarises how the conduct of a post-mortem examination functioned, in part, to appease familial and wider public and political anxiety and the demands for overarching official oversight. At the juncture when post-mortems were effectively mandated, it was perceived that there was still a lot of abuse being perpetrated in institutions. Furthermore, a lot of issues, for example the genesis of fractured ribs frequently found in the insane, were unresolved. Accounts of abuse and scandal lessened across the nineteenth century but whether post-mortem practice had any part to play in this process is speculative. Historians have drawn attention to the fact that the medical assistants of the asylums performed the post-mortems mitigated against them being impartial assessments. There may well have been unspoken accords between medical assistants and attendants so that those performing post-mortems were disinclined to find changes that may have stemmed from abuse or neglect. What stands out from the current study, is that signs of violence were identified in the post-mortems of just two cases from Fair Mile, yet no action came from those findings either on the part of the coroner or in the reports from Visitors and Commissioners. The coroner at Fair Mile seems to have been particularly quick to wave cases through which also reduced scrutiny. So, while the post-mortem itself may have been carried out with care and diligence and, potentially, an open enquiring mind, the processes for independent scrutiny thereafter seemed to be lacking. Alternatively, it could be that because post-mortems were effectively routine, they acted as a preventative measure. Post-mortems had an aura about them that they could reveal the truth and this idea was likely to be held by the attendants and may have given them pause for thought and curbed malpractice. If there was such an effect it is likely to play a smaller role than the detailed recording of untoward incidents (including the monitoring of restraint). The work of Hervey and Andrews respectively examining the English and Scottish Commissioners shows that the Commission could and would intervene when an asylum’s mortality rate was particularly high.

740 Burney, Bodies of Evidence; Cullen, “Post-mortem in the Victorian Asylum,” 284.
(though in those cases there were other markers of a poor quality of care). Whether the Commissioners’ scrutiny of post-mortem records had much impact is, however, questionable and no documentary evidence to that effect was found. In summary, there is only weak evidence that the performance of routine post-mortems played a part in reducing asylum abuse and violence.

A related objective of routine post-mortems was to protect asylum staff against accusations of malpractice and brutality, acting, in Andrew’s words, as “insurance against lawsuits.” Against this yardstick, the routine post-mortem appears to have achieved its aim. For example, the post-mortem exercise usefully reduced potential opprobrium for the staff in two cases where patients had choked to death, an outcome not infrequently seen in insanity in the absence of others’ involvement.

Another key narrative underpinning the pressure to perform post-mortems was the potential to uncover the cause of death and identify organic conditions that were linked to insanity, and which might worsen its outcome. The specific detail in the post-mortem reports analysed in this thesis seems, for the most part, to be consonant with the ascribed cause of death. However, there was frequent usage of vague terms particularly when describing brain changes: for example, congestion in the brain was the sole finding in several cases. This term was in popular use and was promoted by some influential practitioners and it is hard to avoid the suspicion that congestion, a term with no clear definition or boundaries, was reported because of an expectation of finding something, anything, in the brain of a highly disturbed individual. However, even congestion was not ubiquitous, and two out of three post-mortem reports did not mention it. The causes of death ascribed after a post-mortem seemed internally consistent but as other commentators point out, were homogenous. No unusual or unexpected findings were made which is out of step with normal post-mortem practice. It seems that the medical officers were happy to pigeonhole cases into accepted, and acceptable, causes of death. Another issue which bears on this question is that there was considerable similarity in the causes of death and their frequency between cases who had received and those who did not receive a post-mortem. This finding could be an indicator that the regular practice of post-mortems was useful in improving cause of death

744 Cullen, “Post-mortem in the Victorian Asylum,” 284.
reporting for those cases who did not have a post-mortem but it could also point to the relative futility of this complex, laborious procedure.

Arguably the most important driver for carrying out post-mortems was to provide the material and data for research into insanity, to enhance scientific benchwork praxis in the asylum and to disseminate pathological knowledge of insanity. It seems, however, that this complex labour intensive and potentially distressing procedure in these non-academic settings contributed little to the understanding and knowledge of processes underlying insanity. There is no evidence that the findings of the post-mortems carried out at the asylums studied here were ever part of a public output other than a footnote in the LC’s Annual Report. Findings from post-mortems in other asylums in this late Victorian period that were of value were made but they originated from the study of a small number of cases in specialist centres, usually involving an expert from the growing discipline of Pathology and its developing laboratory style setting. Of course, this negative outcome must be seen in the context that even pathological experts struggled to find anything consistently abnormal in the brains and bodies of the insane.745 Thus, the massive amount of work in the asylum post-mortem, for example in weighing the organs or carefully dissecting and examining the meninges, led to precious little. A saving grace, however, is that the medical staff and some relatives may have been encouraged that something was being done to unravel the knotty mysteries of insanity and that negative results have value. Post-mortem pathology at some of the leading British research asylums and psychiatric medical schools, including West Riding, Claybury and Edinburgh, seems to have had a more significant and wider-reaching influence than that produced by less prestigious and well-resourced asylums, including the two surveyed here.

The pathological activities described here should moreover be seen in the broader context of post-mortem research at this time. Nineteenth-century post-mortem studies had fostered a lesion-based notion of insanity. There was a widespread conviction that post-mortem pathology that had so impressively delineated some medical conditions in the first half of the nineteenth century, would soon illuminate the so-called functional disorders of mania and melancholia. Many concurred with Gray’s view that post-mortems potentially were a method that could diminish speculations which lacked some scientific basis.746 By the mid- to later parts of the century,

746 Gray, “General Paresis or Complete Progressive Paralysis,” 66.
however, with the evident lack of a conclusive model emerging, there had been an assimilation of the lesion-based narrative with other theories such as germ theory, and a growing interest in the impact of heredity and evolution. Rosenberg remarks however that the subsequent development of the biomedical sciences of histology, biochemistry and physiology provided a “compelling and seemingly objective store of tools, procedures, models... that promised to delineate disease in newly precise, measurable... terms.” These scientific developments were seen as a way of linking “biology and behaviour, mind and body, past and present,” providing a “reassuringly somatic mechanism with which to explain a variety of unsettling emotions and problematic behaviours.”747 These trends lead one to conclude that the performance of post-mortems in asylums in the late nineteenth century was more than just a process to avoid being censored but one which was imbued with a sense of hope and optimism. The post-mortem procedure also provided a training opportunity for young doctors and inculcated them into new ways of thinking about mental illness from a scientific perspective. To some extent, the post-mortem process dovetailed with the scientific ambitions of new generations of mental medicine practitioners keen to raise the status of the profession and to reassert mental medicine’s expertise. Somatic, neurophysiological and brain-centred models of insanity became more prominent and this was enhanced as moral management was now seen to be failing and asylums becoming fuller with more and more patients, many difficult to cure.

What does this thesis tell us about those performing post-mortems? As with other accounts of this topic, it has proved difficult to uncover meaningful data about post-mortem attendants and their arts, dark or otherwise. This work throws some light on the doctors carrying out these post-mortems and some of the conflicting perceptions of them. Primarily junior medical officers performed the post-mortems in the asylums studied here but this was something for which their medical training had prepared them. Nystrom comments that for the nineteenth-century medical student, gross anatomy courses represented the most significant component of their education; anatomical and pathological knowledge was considered to be the basis of medical professionalism and a benchmark of their social identity.748 The majority of the doctors performing post-mortems in the two series discussed here were graduates of either Glasgow or Edinburgh Universities,


242
which, as described in Chapter 2, were both established centres for pathology with strong reputations in that field. This may account for the apparent good quality of the post-mortem records studied here and, arguably, their consistency and homogeneity (though this would ideally be tested by looking at the reports from other institutions). Ray observes that the frequent occurrence of death in asylums provided support for the development of this aspect of doctors’ professional practice and concludes that “the asylum death-rate did not undermine the medical and sick-role construction of the asylum inmate, but in some respects re-enforced it.”

There was nothing uncovered in this study to detract from the sway of that conclusion. Cullen suggests that post-mortems were, in large part, to satisfy medical officers’ curiosity and scientific ambitions. However, as discussed in more detail in Chapter 3, I find this contention difficult to support fully based as it is on her questionable interpretation of the use of some non-medical language employed in post-mortem reports. According to Buklijas, the role of “pathologist” in the asylum was usually allocated to the youngest medical officer(s) who had the least clinical work. Some juniors benefitted from this activity and got training in scientific methods and a few got case reports published but Buklijas shows how the latter outcome was patchily distributed round only a few centres. Louise Hide observes the precarious position of medical officers who spent years in personal, professional and often geographical isolation, waiting for a position as a Medical Superintendent; “They toiled endless days of asylum life, waiting a good part of their adulthood for the most senior position.”

The research undertaken here on post-mortems supports Hide’s viewpoint. William Murdoch performed most of the post-mortems in the period from his appointment as Assistant Medical Officer in 1881 until his promotion to Physician Superintendent in 1892. From 1894 until his similar appointment to the top job in 1918, Dr E. L. Dunn also carried out a large part of these activities. Post-mortems were a duty for junior doctors and one with limited benefits for the majority and, at best, delayed gratification for a few.

Overall, the findings of this enquiry into asylum post-mortems reveals that, for the most part, they were carried out because they were a prescribed and increasingly expected feature of asylmumd. In practice, they served little purpose and illuminated little, though they may well have engendered feelings of hope and of control in an environment where these feelings were in short

750 Cullen, “Post-mortem in the Victorian Asylum,” 286-7.
751 Buklijas, “The Laboratory and the Asylum,” 311–25.
752 Louise Hide, Gender and Class in English Asylums, 1890-1914 (Cham: Springer, 2014), 55.
supply. This leaves the question of whether the material from post-mortem series like these provides insights for a medical historian. I argue that it does, albeit in a limited way. However, a reliance on records which as Andrews points out can be “innately jaundiced” (although whether this applies to post-mortem reports has been little studied) serves to potentially limit the worth of this strategy.\(^{753}\) Notwithstanding the caveats above, some important findings were made. People with mania and melancholia in this study were dying, on average, 15 years younger than the general population and infections were associated with death of two thirds of the patients, information likely to be both accurate and informative. The finding of vessel changes in many of the remainder of this young population and the linking of those changes to findings in the brain provides information which helps explain the premature mortality of these forms of insanity. It is argued here that the insane appear not just to exhibit a predilection for severe, often fatal, infections, but also to suffer from vascular disease as a consequence of the combination of deprivation, and/or stress, and/or infections, with an inflammatory process being a probable final common pathway to morbidity and mortality.

The implications of this study for future work are now considered. Since the methodology of this thesis is unique (at least as far as I am aware), the main future research strategy would be to attempt a replication. This approach would answer several questions such as how generalisable and robust these findings were. It would be ideal to study other asylums, both rural and urban, academic and non-academic, UK versus international (perhaps colonial and non-colonial) and to look at other time windows to pick up secular trends. However, if one was going to go to this effort (and it is very considerable), it might be worth approaching such a project differently. Firstly, the research team should be multi-disciplinary as McCarthy and colleagues have persuasively argued for.\(^{754}\) At a minimum, the team should include a pathologist, a psychiatrist, a historian and someone with expertise in historical demography and/or epidemiology. Secondly, a variety of overlapping approaches should be taken, for example a large survey of causes of death in death certificates, perhaps on digitised records, combined with drilling down in detail into a small number of cases. Coroner’s records also need to be perused alongside such data sets. I had access to coroners’ reports but not in a format that allowed searching and it was deemed that it would

\(^{753}\) Andrews, “Case Notes, Case Histories,” 267.
\(^{754}\) McCarthy et al., “Lives in the Asylum Record,” 358-79.
not be a productive use of time to wade through them looking for my few cases of interest. Following up this thesis’s approach should also involve further attempts at finding suitable control populations. This is a particularly important for assessing the true rate of vascular disease and vessel pathology in the insane. In my view, appropriate control settings would have to be from a basket of different settings to the asylum to be able fully to draw meaningful comparisons. Finally, the design of a further study would benefit greatly from being discussed and debated widely, both within a putative research team and with the wider historical and scientific community.

A few final reflections. Asylum studies have ranged in methodologies and styles. According to Murphy, Scull’s “deeply researched and provocative” 1979 account of the growth of public asylums in nineteenth-century England, Museums of Madness, attributed the expansion of asylumdom to “the emerging commercial market economy and the consequent extrusion of inconvenient non-working people from the mainstream of family and community life.” Scull interpreted the growing interest in madness by specialist mad-doctors as an unattractive bid for power and status by a group of financially insecure members of a profession still on the threshold of respectability. Scull acknowledged that his work was stimulated in part by Foucault’s essays on power relations in Histoire de la Folie (and its early English abridgements as Madness and Civilisation), which Murphy calls “brilliant but flawed,” and he softened some of his critique in later publications. At the same time, according to Murphy, Porter reasserted the value of a pragmatic analysis of events in the asylum in the context of a broader cultural approach and it is in this latter spirit that the current research was carried out.

Luckin remarks that researchers whose “primary training has been medical” may oversimplify findings and take insufficient account of the social and economic context. Wrightson opines that the “inclinations of modern clinicians sit uneasily in an environment where historians are only too well aware of the complexities, contingencies (and) ironies of historical change.”

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757 Murphy, “The Administration of Insanity,” 336; Michel Foucault, History of Madness, Translated by Jonathan Murphy and Jean Khalfa (London/New York: Routledge, 2006).
758 Luckin, “Death and Survival in the City,” 53-61.
observes wryly that “doing history is easier for clinicians (and bad history simpler still) than is psychiatry for historians,” advocating that rather than privileging a special class of medical facts, historians of psychiatry should study all sorts of evidence. Micale and Porter, along with a range of other historians, have criticised some clinically authored psychiatric histories as often overly defensive of professional interests, and classically “presentist, progressivist, and tenaciously internal.” However, both these scholars have collaborated pretty frequently in joint research and writing endeavours with clinician historians, while McCarthy comments more positively that collaboration and inter-disciplinary research in asylum studies are both valuable and wanting. McGovern suggests that historians must explore the inner history of asylums to discover more about mortality within in them. I hope my investigations of the individual causes of death in detail is a contribution in the spirit of these latter sentiments and that I have largely avoided the pitfalls laid out by the former commentators.

Commenting in 1855 on mortality figures, Davey opined that “… the insane are, as a body, not only susceptible to the ordinary endemic and epidemic disorders, as fevers, cholera, diarrhoea... they are especially liable to be affected by every kind of morbid influence, however and wherever developed.” Exactly 50 years later, in 1905, Jones similarly concluded that “we are... justified in looking upon insanity as a diseased state which shortens life, and upon the insane as a class which is marked by considerable physical inferiority and lowered vital resistance to disease.” In her 2012 commentary on Victorian and more modern asylum mortality, Cathy Smith puts it thus: “The continued susceptibility of insane patients to death could neither be fully explained by medical science nor, in the end, significantly reduced by medical or state provision.” I am optimistic that the enquiry carried out here and the evidence accrued provides a basis for understanding not just how these issues were perceived at these various times but generates models which have explicatory value.

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764 Jones, “Prognosis in Mental Diseases,” 1579.
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   1. Male D/H10 D2/1/1-19 1870-1905
   2. Female D/H10 D2/2/1 1871-1905

D. Register of Admissions
   1. D/H10 D1/1/1/1 1870-1879
   2. D/H10 D1/1/1/2 1879-1885
   3. D/H10 D1/1/1/3 1886-1890
   4. D/H10 D1/1/1/4 1891-1895
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Total proforma shown along with close-up of points of note. Cases are from the under 55 group of patients with mania or melancholia (Chapter 3)

Case 1 Post-mortem report on a 48-year-old labourer dying of “Exhaustion from acute mania” 6 days after admission.

Detail of Thorax report

766 Post-mortem proforma from Sunnyside Asylum were almost identical. It was intended to show photographs of examples from there but the Covid-19 pandemic of 2020 precluded this.
Points of note:- R ventricle thin. Heart muscle pale and friable.

Case 2 Post-mortem report of 40-year-old bricklayer dying of “Exhaustion from acute mania” 4 days after admission.
Detail of report of Brain and Thorax


Case 3 Post-mortem report from case GH (see Chapter 3)
Points of note:- No “signs or marks of violence”. Bruising in the scalp which “appear to have been in existence for three to six” days. No fracture of skull which was noted to be “extremely thin”. Large haemorrhage in occipital and parietal areas on both sides”. Brain, including blood vessels, otherwise healthy.
Case 4 Post-mortem report on 53-year-old housewife with delusional insanity who died of “Fatty Heart”

Detail of heart and vessels report.

Points of note:- Myocardium (heart muscle) is “soft and flabby showing fatty change”. Ventricles hypertrophied. “Commencing atheroma of aortic valve”.

282
Case 5 Post-mortem report from 47-year-old female dying of “Exhaustion from Mania”.

Detail of thorax report

Points of note:- Myocardium (heart muscle) is “soft and degenerated”. Atheroma of mitral and aortic valves.
Case 6 Post-mortem report from 42-year-old antique dealer dying of Pneumonia.

Detail of Head and Thorax report

Appendix B. Causes of death in main clinical subgroups (Mania, Melancholia, Dementia, Epilepsy, Idiocy and Other) across a 10-year period (1896-1905) in the Berkshire Asylum.

Causes of death as recorded in the Admissions and Deaths Register. Numbers and age at death (in years, Average or Mean ± Standard deviation) is also noted for each of these groupings and for the cause of death subgroups (Exhaustion, Brain conditions, vascular disease, infection and other).
<table>
<thead>
<tr>
<th>Clinical Subgroup (Ave age at death)</th>
<th>Cause of death</th>
<th>Ages at Death (years)</th>
<th>Cause of death sub-group</th>
<th>n</th>
<th>Number of cases and age at death of Subgroup (years Mean ± SD)</th>
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</thead>
<tbody>
<tr>
<td>Mania with Senile Mania shown with a A *</td>
<td>Cerebral softening</td>
<td>79, x, 55, 64, 71, 63, 66, 40, 70, 63*, 68, 42, 78, 60</td>
<td>Brain Conditions</td>
<td>14</td>
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<td>Mania without senile mania n=139 (55 years)</td>
<td>Senile Decay</td>
<td>73, 70*, 78*, 85*, 85, 76, 78, 83, 70, 70, 79, 70, 68, 79, 76*, 66, 75, 72, 71, 81</td>
<td>Vascular disease n=36 58±14</td>
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<tr>
<td></td>
<td>Cerebral Atrophy</td>
<td>60, 47, 68</td>
<td>Vascular disease</td>
<td>17</td>
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<tr>
<td></td>
<td>Senility</td>
<td>74*, 82*</td>
<td>Infections</td>
<td>17</td>
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<td></td>
<td>Morbis Cordis</td>
<td>71, 80, 66, 70, 64, 71*, 43, 74, 58, 53, 63, 67*, 72, 50, 53, 49, 58</td>
<td>Infections n=54 47±15</td>
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<td></td>
<td>Heart Disease</td>
<td>63, 41, 74, 41, 34, 60, 67, 59</td>
<td>Infections</td>
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<td></td>
<td>Cardiac failure</td>
<td>26, 34, 53, 61, 78</td>
<td>Infections</td>
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<td></td>
<td>Apoplexy</td>
<td>76, 74*, 27, 54, 68</td>
<td>Infections</td>
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<td></td>
<td>Syncope</td>
<td>45</td>
<td>Infections</td>
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<td></td>
<td>Phthisis Pneumonale</td>
<td>42, 34, x, 46, 23, 48, 40, 32, 51, 40, 38, 33, 48, 32, 36, 53, 39, 26, 45, 30, 47, 49, 22, 31</td>
<td>Infections</td>
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<td>Tuberculosis</td>
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<td>Infections</td>
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<td>Pneumonia</td>
<td>73, 76, 66, 70, 62*, 69, 69, 52, 49, 52, 28, 73*, 44, 59</td>
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<td>Pyelonephritis</td>
<td>49, 59</td>
<td>Infections</td>
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<td>Enteric fever /Colitis</td>
<td>24, 38, 34, 55, 39</td>
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<td>Dysentery</td>
<td>47, 65*, 71, 52</td>
<td>Infections</td>
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<td>Condition</td>
<td>Group without senile melancholia (56 years)</td>
<td>Exhaustion</td>
<td>Cerebral softening</td>
<td>Senile Decay</td>
<td>Senility</td>
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<tr>
<td>Abscess of liver</td>
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<td>Meningitis</td>
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<td>62</td>
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</tr>
<tr>
<td>Cancer</td>
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</table>
( Rectum, duodenum, uterus(2), pancreas, liver, sarcoma) |                                              |            |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Volvulus                          |                                          | 53         |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Peritonitis                       |                                          | 63         |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Suffocation                       |                                          | 33         |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| MELAN-CHOLIA                      | n=70 (57 years)                          |            |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Those with senile melancholia     |                                          |            |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| shown with a *                    |                                          |            |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Exhaustion                        |                                          | 60,58,54,44,50,64,44 |       |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Cerebral softening                |                                          | 62,60,60,60,61,61,63,47,63 |       |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Senile Decay                      |                                          | 72,72,70*,75,81* |       |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Senility                          |                                          | 63,70,88   |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Morbis Cordis                     |                                          | 74,66,45,74,59,65*,62 |       |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Heart Disease                     |                                          | 71,66,66,54 |       |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Cardiac failure                   |                                          | 59,59,47   |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Apoplexy                          |                                          | 72,60      |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Lung gangrene                     |                                          | 57         |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Phthisis Pneumonale               |                                          | 36,24,52,36,56,25,38,40 |       |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Pneumonia                          |                                          | 42,55,44,56,78,66,55,40,37,78*,68 |       |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Enteric fever /Colitis            |                                          | 49         |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Dysentery                          |                                          | 63*,38,57  |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Meningitis                        |                                          | 59         |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Empyema                           |                                          | 53         |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |
| Cancer (bladder)                  |                                          | 55         |                   |              |          |              |              |                |          |              |                   |           |                      |           |            |         |                 |        |

Exhaustion group:
- Exhaustion: 7 cases, mean 53 ± 7

Brain conditions group:
- Brain Conditions: 16 cases, mean 66 ± 10

Vascular disease:
- Vascular disease: 17 cases, mean 62 ± 8

Infections:
- Infections: 25 cases, mean 50 ± 14

Others:
- Others: 10 cases, mean 56 ± 14
<table>
<thead>
<tr>
<th>Condition</th>
<th>Code</th>
<th>n</th>
<th>±</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shock after operation</td>
<td>59</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Peritonitis</td>
<td>55</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Spinal Atrophy</td>
<td>58</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Bright’s Disease</td>
<td>62</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Exhaustion</td>
<td>76‡</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Cerebral softening</td>
<td>60,67,78,84,63,65,56,65,64,64‡,68,50,66,67,45</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Senile Decay</td>
<td>80,84,76,83,81,70,75,82,87,77,89,77,77,82,73,72,77,72,73,85,70,73,78,86,73,79,74,70,80,75,73,68,84,76,68,77,77‡,79,76,71,87,80,81,89,79,79,81,64,76,70,77,69,58,85,88,74,78,75,76‡,76,84,78‡,89,77,75</td>
<td>65</td>
<td></td>
</tr>
<tr>
<td>Cerebral Atrophy</td>
<td>75,66,67,52,68</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Senility</td>
<td>79,68,83</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Morbis Cordis</td>
<td>49,76,64,61,56‡,74,68,56,77</td>
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</tr>
<tr>
<td>Heart Disease</td>
<td>71,68‡,62‡</td>
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</tr>
<tr>
<td>Cardiac failure</td>
<td>64,63,42,46,64</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Apoplexy</td>
<td>52,48,55,72‡</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Phthisis Pneumonale</td>
<td>52,26‡,41,56,59,56,41‡,27,22‡,27,57‡,62,56,54,41,37,54,69,62,66,41</td>
<td>21</td>
<td></td>
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<tr>
<td>Tuberculosis</td>
<td>37</td>
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<tr>
<td>Pneumonia</td>
<td>60,33,63,73,72,69,28,49,76,29,56,70,62,77,64,65‡,68,66‡,63,70,66,49,27</td>
<td>23</td>
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</tr>
<tr>
<td>Bronchitis</td>
<td>X</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Enteric fever/Colitis</td>
<td>38</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Dysentery</td>
<td>31‡,49,80,82</td>
<td>4</td>
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</tr>
<tr>
<td>Primary DEMENTIA</td>
<td>n=169</td>
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<td></td>
</tr>
<tr>
<td>(66 years)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Those with secondary dementia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>shown with a ‡</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Exhaustion                      | n=1 76|     |     |

Brain Conditions                | n=88 74±9|     |     |

Vascular disease                 | n=21 61±10|     |     |

Infections                      | n=52 54±16|     |     |
<table>
<thead>
<tr>
<th>Condition</th>
<th>Frequency</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Empyema</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cancer (Bladder, stomach, sarcoma)</td>
<td>50, 82, 50</td>
<td></td>
</tr>
<tr>
<td>Subdural</td>
<td>65</td>
<td></td>
</tr>
<tr>
<td>Unknown</td>
<td>49, 80, 82</td>
<td></td>
</tr>
<tr>
<td>Others</td>
<td>n=7</td>
<td>65±15</td>
</tr>
<tr>
<td>Exhaustion</td>
<td>29, 23, 70</td>
<td></td>
</tr>
<tr>
<td>Cerebral softening</td>
<td>55, 56, 60</td>
<td></td>
</tr>
<tr>
<td>Senile Decay</td>
<td>66, 72, 77, 70, 70, 71</td>
<td></td>
</tr>
<tr>
<td>Cerebral Atrophy</td>
<td>50, 48, x</td>
<td></td>
</tr>
<tr>
<td>Morbis Cordis</td>
<td>65, 43</td>
<td></td>
</tr>
<tr>
<td>Cardiac failure</td>
<td>50, 28, 15</td>
<td></td>
</tr>
<tr>
<td>Apoplexy (check)</td>
<td>39, 47</td>
<td></td>
</tr>
<tr>
<td>Phthisis Pneumonale</td>
<td>26, 32, 18, 38, 16, 27, 13, 19, 17, 48, x, 16, 46, 17, 22, 19, 17, 33, 32</td>
<td>19</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Pneumonia</td>
<td>34, 20, 28, 48, 78, 46, 51, 50, 22, 48</td>
<td>10</td>
</tr>
<tr>
<td>Enteric fever/Colitis</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>Pyelonephritis</td>
<td>49</td>
<td></td>
</tr>
<tr>
<td>Dysentery</td>
<td>52, 47</td>
<td></td>
</tr>
<tr>
<td>Marasmus</td>
<td>16, 29, 13, 17</td>
<td>4</td>
</tr>
<tr>
<td>Epilepsy</td>
<td>64, 39</td>
<td></td>
</tr>
<tr>
<td>Status epilepticus</td>
<td>54, 15</td>
<td></td>
</tr>
<tr>
<td>Peritonitis</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>23</td>
<td></td>
</tr>
</tbody>
</table>

**Others**

n=7 65±15

**Exhaustion**

n=3 40±21

**Brain Conditions**

n=12 63±9

**Vascular disease**

n=7 41±15

**Infections**

n=34 32±16

**Others**

n=16 32±16
<table>
<thead>
<tr>
<th>Condition</th>
<th>Patients</th>
<th>Codes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bright’s disease</td>
<td>42</td>
<td>1</td>
</tr>
<tr>
<td>Cancer (bowel)</td>
<td>44</td>
<td>1</td>
</tr>
<tr>
<td>Shock</td>
<td>43</td>
<td>1</td>
</tr>
<tr>
<td>Ataxia</td>
<td>51</td>
<td>1</td>
</tr>
<tr>
<td>Unknown</td>
<td>20,17</td>
<td>2</td>
</tr>
<tr>
<td><strong>EPILEPSY</strong></td>
<td><strong>52</strong></td>
<td><strong>11</strong></td>
</tr>
</tbody>
</table>
| Exhaustion                      | 52,43,24,22,26,59,23,21,55,24,29 | 11 Exhaustion n=11 34±14  
| Cerebral softening              | 71,53,66 | 3                                                                    |
| Senile Decay                    | 85,84,79,49,61 | 5                                                                    |
| Cerebral Atrophy                | 60       | 1                                                                    |
| Morbis Cordis                   | 72       | 1                                                                    |
| Cardiac failure                 | 56,64,29, x,19,69 | 6                                                                    |
| Apoplexy (check)                | 56,57    | 2                                                                    |
| Phthisis Pneumonale             | 55,61,32,26,56,31 | 6                                                                    |
| Pneumonia                       | 33,31,43,59,48,41,53 | 7                                                                    |
| Enteric fever /Colitis          | 34       | 1                                                                    |
| Dysentery                       | 41, 67   | 2                                                                    |
| Epilepsy                        | 27,29,35,39,43,38 | 6                                                                    |
| Status epilepticus              | 15,37,23,35,36,31 | 6                                                                    |
| Epileptic dementia              | 38,70    | 2                                                                    |
| Emphysema                       | 14       | 1                                                                    |
| Ca (Jaw)                        | 42       | 1                                                                    |

**n=61** (45 years)
Appendix C. Causes of death from post-mortem reports of those aged 55 or under at death with diagnosis of Mania or Melancholia in Berkshire and Sunnyside Asylums

<table>
<thead>
<tr>
<th>Cause of death of death category</th>
<th>Examples (in order of frequency in each sub-group)</th>
</tr>
</thead>
</table>
| INFECTIONS                      | Tuberculosis (TB) - Phthisis pneumonale, acute TB, TB, Acute miliary TB  
                                   | Pneumonia - Bronchopneumonia, Acute Pneumonia, Empyema, Pleurisy, Bronchitis  
                                   | Gastro-intestinal Infections - Enteric fever, Dysentery.  
                                   | Other Infections - Meningitis, Septicaemia                                                                                                                                                                                                  |
| OTHER                           | Various cancers.  
                                   | Intestinal obstruction, Volvulus, Perforation, Peritonitis, Rupture of bowel, Ulcerative colitis, Hydatid cysts.  
                                   | Choking  
                                   | Suicide – Asphyxia, Laceration of throat.                                                                                                                                                                                                   |
| VASCULAR                        | Heart disease, Heart failure, Morbis Cordis, Valvular disease of heart, Apoplexy, Cerebral haemorrhage, Rupture of aneurysm, Congestion of lungs.                                                                                                          |
| BRAIN CONDITIONS                | Brain atrophy, Cerebral softening, Senile decay, Brain congestion, Senility                                                                                                                                                                   |
| EXHAUSTION                      | Exhaustion from mania, Exhaustion from melancholia                                                                                                                                                                                            |
Appendix D. Post-mortem reports in cases with melancholia or mania
dying at or under the age of 55 in Sunnyside, 1892-1901.

Abbreviations:- ME= Melancholia   MA= Mania

*Length of stay categories:-  A – less than a week, B – 1-4 weeks, C – 4 weeks - 12 months, D – 1-5 years, E – more than 5 years.

<table>
<thead>
<tr>
<th>Number</th>
<th>Initials</th>
<th>Age at Death</th>
<th>Sex</th>
<th>Clinical diagnosis on admission</th>
<th>Occupation</th>
<th>Cause of death</th>
<th>External post-mortem findings</th>
<th>Internal post-mortem findings</th>
<th>Length of stay group*</th>
<th>Number and length of Attack</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>ET</td>
<td>17</td>
<td>F</td>
<td>Acute delirious mania</td>
<td>Domestic servent.</td>
<td>Acute delirious mania</td>
<td>Well nourished “tending to stout” Dark fluid on upper thirds of thighs as “though commencing to be gangrenous”</td>
<td>Pia shows passive congestion but no thickening. Grey Matter “thinner than average” Nil else Heart smaller than average. Pericardium normal. Mitral valve dilated and “obstructed by tough fibrinous clot” Nil else Slight hypostatic congestion in both lungs.</td>
<td>B</td>
<td>First</td>
</tr>
<tr>
<td>2.</td>
<td>JK</td>
<td>40</td>
<td>M</td>
<td>ME</td>
<td>-</td>
<td>Phthisis</td>
<td>Tall and very emaciated</td>
<td>Brain NAD Heart valves normal. Heart muscle “flabby and hypertrophied” Lungs – cavities and adhesions.</td>
<td>E</td>
<td>?</td>
</tr>
</tbody>
</table>
### Case 3.
**Name:** GL  
**Age:** 52  
**Gender:** M  
**Diagnosis:** Delusional insanity  
**Exam Findings:** Apoplexy, Well nourished—“very corpulent”, Bedsores  
**Pathological Findings:** Meninges NAD, L cerebral haemorrhage affecting internal capsule, Thickened cerebral arteries, Heart muscle is “pale and flabby” and that of L ventricle thinned, Slight hypostatic congestion in both lungs.

### Case 4.
**Name:** JC  
**Age:** 40  
**Gender:** M  
**Diagnosis:** Recurrent mania  
**Exam Findings:** Acute military TB, Well nourished  
**Pathological Findings:** Brain NAD, Pericardium studied with nodules of miliary tubercles, Valves normal, L ventricle hypertrophied, Old scars in L apex R lung shows cavity, Miliary tubercles++

### Case 5.
**Name:** JT  
**Age:** 32  
**Gender:** F  
**Diagnosis:** ME  
**Exam Findings:** Duodenal ulceration, Well nourished  
**Pathological Findings:** Brain NAD, Heart valves normal, Heart muscle normal apart from some
Thickening. Patches of atheroma in aorta. Lungs – apical scars otherwise NAD Perforations in duodenum AND “bowel” Exudation++

<table>
<thead>
<tr>
<th>6. JR 55 M</th>
<th>ME</th>
<th>Joiner</th>
<th>Asphyxia</th>
<th>Well nourished Ligature marks</th>
<th>Pia and cerebral tissue congested. Trachea is compressed. Lungs “gorged” with blood. Heart muscle is hypertrophied. Valves OK</th>
<th>B First 6 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>7. MM 43 F</td>
<td>ME</td>
<td>Hydatid cyst Pleurisy</td>
<td>Much emaciated</td>
<td>Pia etc milky and opaque Macroscopically normal brain Pericardium normal. Valves and muscle normal. Oedematous lungs with evidence of “pleurisy” Cyst in spinal cord into chest contained sago grain bodies microscopically typical of hydatid cysts.</td>
<td>E ?</td>
<td></td>
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<td><strong>8.</strong></td>
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</tr>
<tr>
<td><strong>JP</strong></td>
<td>Delusional mania</td>
<td>Housekeeper</td>
<td>Brain Atrophy</td>
<td>Much emaciated</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>50 F</strong></td>
<td></td>
<td></td>
<td></td>
<td>Subdural fluid++</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Bullae under pia.</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Generalised</td>
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<td></td>
<td></td>
<td></td>
<td>convolutional</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>thinning esp. at the</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>fissure of Rolando.</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Ventrices much</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>dilated. Vessels at</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>base of brain were</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>atheromatous.</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>arteries and aorta</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>were atheromatous.</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Heart valves normal.</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lungs – mild oedema</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td><strong>C</strong></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>First 14 weeks</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>9.</strong></td>
<td>Delusional insanity</td>
<td>-</td>
<td>Ca stomach</td>
<td>Body of aged woman emaciated</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>AL</strong></td>
<td></td>
<td></td>
<td></td>
<td>Pia etc normal</td>
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</tr>
<tr>
<td><strong>55 F</strong></td>
<td></td>
<td></td>
<td></td>
<td>Brain was firm and</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>large. Vessels at base</td>
<td></td>
<td></td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>of brain were</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>atheromatous</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Heart muscle is “somewhat flabby”</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lungs old TB and</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>were somewhat</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>oedematous.</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Stomach showed Ca</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>masses in and near</td>
<td></td>
<td></td>
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<td>stomach. Liver</td>
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<td>showed secondaries.</td>
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<tr>
<td>10.</td>
<td>WM</td>
<td>ME</td>
<td>With delusions</td>
<td>Phthisis Pneumonale</td>
<td>Much emaciated</td>
<td>Subdural fluid++ Bullae under pia. Convolutions atrophied “slightly”. Coronary arteries and aorta were “slightly” atheromatous. Lungs riddled with tubercular granulations with some cavitation</td>
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<td>11.</td>
<td>GJ</td>
<td>Acute mania with delirium</td>
<td>Domestic servant</td>
<td>Pneumonia</td>
<td>A little emaciated</td>
<td>Sub-arachnoid fluid++ Brain substance was “pale”. Heart valves normal. Lungs – pneumonia at base with consolidation</td>
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<tr>
<td>12.</td>
<td>DM</td>
<td>ME</td>
<td>-</td>
<td>Phthisis Pneumonale</td>
<td>Much emaciated</td>
<td>Pia etc normal Grey matter looked “pale and somewhat thin” Puncta vasculosa were well marked throughout the white matter. Vessels at base of brain were slightly atheromatous. Both ventricles contained atheroma.</td>
</tr>
</tbody>
</table>

297
<table>
<thead>
<tr>
<th>No.</th>
<th>Patient Info</th>
<th>Clinical Details</th>
<th>Diagnosis</th>
<th>Notes</th>
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</thead>
<tbody>
<tr>
<td>13.</td>
<td>MAF 24 F</td>
<td>ME - Phthisis Pneumonale</td>
<td>Abdomen much distended</td>
<td>Brain substance was “anaemic” and grey matter slightly thinned. Pericardial fluid++. Valves OK. Lungs riddled with tubercular granulations with some cavitation. Abdomen filled with fluid.</td>
</tr>
<tr>
<td>14.</td>
<td>RMcK 35 F</td>
<td>Acute mania with delirium - Phthisis Pneumonale</td>
<td>A little emaciated Sub-arachnoid fluid++. Brain substance was “pale”. Heart valves normal. Lungs – pneumonia at base with consolidation.</td>
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</tr>
<tr>
<td>15.</td>
<td>AA 25 F</td>
<td>MA - Septic Pneumonia</td>
<td>Much emaciated Grey matter was “pale” and slightly thinned. Atheroma of valves. Lung lobes consolidated and “gangrenous” with</td>
<td></td>
</tr>
</tbody>
</table>
16. JS 51 F
MA Servant and knitter Ca uterus Much emaciated
some tubercular nodules
Several large areas of cerebral softening
Heart valves normal.
NAD
Lungs –NAD Large cancer of uterus.
D Second ?

17. EM 27 F
MA Housewife Pneumonia Well nourished Pia etc. normal
Brain substance was very pale
Heart hypertrophied and dilated. Mitral valve was stenosed.
Atheromatous clot in R ventricle. L Lung showed consolidation.
C First 5 years

18. AS 25 M
ME with delusions. - Phthisis Pneumonale Much emaciated
Brain substance was “pale” but otherwise
NAD
Valves and muscle normal
Lungs had numerous tubercular foci.
D ?

19. WC 49 M
ME with delusions. - Phthisis Pneumonale Much emaciated
Brain substance was thinned all over brain and softened.
Heart valves normal.
NAD
Lungs - cavity formation with
D ?
<table>
<thead>
<tr>
<th>20.</th>
<th>MO</th>
<th>MA</th>
<th>Domestic</th>
<th>Exhaustion from mania</th>
<th>Well nourished</th>
<th>consolidation and pleural thickening and calcification.</th>
</tr>
</thead>
<tbody>
<tr>
<td>55 F</td>
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<td>D First 4 years</td>
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</table>

<table>
<thead>
<tr>
<th>21.</th>
<th>SC</th>
<th>MA</th>
<th>Knitter and washer</th>
<th>Exhaustion from mania</th>
<th>Much emaciated</th>
<th>Pia etc. normal Brain substance normal Valves and muscle normal. Some epicardial fat. Lungs had tubercular foci and a cavity.</th>
</tr>
</thead>
<tbody>
<tr>
<td>48 F</td>
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<td>C First 5 weeks</td>
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</table>

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<thead>
<tr>
<th>22.</th>
<th>JG</th>
<th>Delusional mania</th>
<th>Phthisis Pneumonale</th>
<th>Much emaciated</th>
<th>Vessels at base of brain were atheromatous otherwise NAD. Heart valves normal.</th>
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<tbody>
<tr>
<td>32 F</td>
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<tr>
<td>23. RB 52 F</td>
<td>Delusional mania</td>
<td>Farm servant’s wife</td>
<td>Valvular disease of heart</td>
<td>Well nourished</td>
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<tr>
<td>24. JMcA 44 M</td>
<td>ME</td>
<td>-</td>
<td>Phthisis Pneumonalis</td>
<td>Fairly well nourished</td>
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</tbody>
</table>

Pericardium thickened. Lungs: tubercular mass and cavity formation with consolidation and pleural thickening.

Pia opalescent Brain substance was congested and showed many vascular “markers”. Vessels at base of brain were atheromatous. Heart hypertrophied and dilated. Aortic and pulmonary valves were incompetent. Aorta and coronary vessels were diffusely atheromatous. Hypertrophy and fatty degeneration of ventricular muscles. Lung congestion. Epicardial fat.

Pia etc. normal Brain substance normal

D Second Few days
<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Age</th>
<th>Occupation</th>
<th>Symptoms</th>
<th>Pathological Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>25.</td>
<td>RB</td>
<td>39</td>
<td>MA w/ delusions</td>
<td>Phthisis and pericarditis. Miliary TB</td>
<td>Well nourished. Pia etc. thickened. Otherwise, NAD</td>
</tr>
<tr>
<td>26.</td>
<td>AN</td>
<td>32 M</td>
<td>MA</td>
<td>Intestinal obstruction</td>
<td>Abdomen distended</td>
</tr>
<tr>
<td>27.</td>
<td>HI</td>
<td>54 F</td>
<td>MA</td>
<td>Knitter and crofter</td>
<td>Pneumonia</td>
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<tr>
<td><strong>28. WW</strong></td>
<td><strong>ME</strong></td>
<td>None</td>
<td>Exhaustion from melancholia</td>
<td>Very emaciated Bedsores</td>
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<tr>
<td><strong>37 F</strong></td>
<td></td>
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<td></td>
<td>Brain - NAD other than some congestion Heart atrophied and dilated Lungs. Tubercular nuclei with some cavitation. One large cavity seen in which could be placed “a pigeon’s egg”</td>
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<tr>
<td><strong>29. SD</strong></td>
<td><strong>MA</strong></td>
<td></td>
<td>Advanced TB</td>
<td>Very much emaciated Minimal examination, “body needed for burial”. Caseation in lungs</td>
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<tr>
<td><strong>51 F</strong></td>
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<tr>
<td><strong>30. GC</strong></td>
<td><strong>MA</strong></td>
<td></td>
<td>Pneumonia</td>
<td>Well nourished Bruises over legs and arms “size of 5/ piece</td>
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<tr>
<td><strong>41 F</strong></td>
<td></td>
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<td></td>
<td>Brain showed “slight congestion”. Valves normal. Heart normal “to all appearances” No atheroma. Lungs consolidated. No sign of TB</td>
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<tr>
<td><strong>31. MM</strong></td>
<td><strong>MA</strong></td>
<td></td>
<td>Pneumonia</td>
<td>Rather emaciated Congestion of brain and pia etc. and some softening. Heart was flabby and L ventricle</td>
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<tr>
<td><strong>54 F</strong></td>
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<tr>
<td>No.</td>
<td>Age</td>
<td>Gender</td>
<td>Occupation</td>
<td>Disease</td>
<td>Condition</td>
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<tr>
<td>34.</td>
<td>AC</td>
<td>43</td>
<td>M</td>
<td>ME</td>
<td>Exhaustion from melancholia/gangrene of lungs</td>
</tr>
</tbody>
</table>
and conspicuous than normal. Basal arteries NAD.
Valves normal.
Myocardium pale.
Lungs congested and oedematous with “gangrene” at bases

<table>
<thead>
<tr>
<th>Age</th>
<th>Name</th>
<th>Occupation</th>
<th>Cause of Death</th>
<th>Condition</th>
<th>Post Mortem Notes</th>
</tr>
</thead>
</table>
| 35. | JR   | Knitter    | Pneumonia      | Well nourished | Brain - NAD
     | 45 F |            |                | Heart was flabby and L ventricle hypertrophied.
     |      |            |                | Epicardial and pericardial fat
     |      |            |                | Mitral valve thickened and aortic valve and aorta showed “commencing atheroma”.
     |      |            |                | R lung shows congestion and pneumonia. |

| 36. | JY   | -          | TB and Cerebral softening | Emaciated. Several scars on the back | Generalised diffuse softening of brain particularly of white matter in | D |
|     | 50 M |            |                            |                                     |                                               |

<p>| 305 |</p>
<table>
<thead>
<tr>
<th>No.</th>
<th>Name</th>
<th>Age</th>
<th>Sex</th>
<th>Occupation</th>
<th>Disease</th>
<th>Findings</th>
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</thead>
<tbody>
<tr>
<td>37.</td>
<td>SW</td>
<td>53</td>
<td>F</td>
<td></td>
<td>Phthisis and cardiac disease</td>
<td>Emaciated.</td>
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<td>TB nodules in both lungs++ with cavities and nodules.</td>
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<td>Heart NAD</td>
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<tr>
<td>38.</td>
<td>JC</td>
<td>48</td>
<td>F</td>
<td>Housewife</td>
<td>Congestion of brain</td>
<td>Thin</td>
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<tr>
<td>39.</td>
<td>WD</td>
<td>24</td>
<td>F</td>
<td></td>
<td>Phthisis</td>
<td>Emaciated. The penis has been amputated.</td>
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<td>40.</td>
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<td>Acute TB</td>
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<tr>
<td>Patient</td>
<td>Gender</td>
<td>Age</td>
<td>Occupation</td>
<td>Pathological Findings</td>
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<tr>
<td>34</td>
<td>F</td>
<td>30</td>
<td></td>
<td>TB nodules in both lungs++ with cavities and nodules. Aortic and mitral valve incompetent. Milk spot right ventricle.</td>
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<tr>
<td>41</td>
<td>JM</td>
<td>52</td>
<td></td>
<td>Phthisis Emaciated with bedsores Brain substance pale. TB nodules in both lungs++ with cavities. Cardiac muscle pale and flabby with gelatinous degeneration. Aortic incompetence</td>
<td></td>
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</tr>
<tr>
<td>42</td>
<td>CS</td>
<td>37</td>
<td>Domestic servant</td>
<td>Congestion of brain and pneumonia Emaciated Brain and grey matter congested. White matter puncta vasculosa are numerous. Aorta shows slight atheroma. Mitral cusp thickened. Lung is a state of red hepatisation.</td>
<td></td>
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</tr>
<tr>
<td>43</td>
<td>JG</td>
<td>42</td>
<td></td>
<td>Cerebral haemorrhage Swelling of neck and thighs Marked softening of the brain. Seen particularly in post parietal region and corpus callosum. Basal ganglia are in</td>
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</tr>
</tbody>
</table>

**Notes:**
- TB: Tuberculosis
- Phthisis: Emaciation
- Aortic incompetence: Aortic valve incompetence
- Congestion of brain and pneumonia
- Cerebral haemorrhage
- Marked softening of the brain
<table>
<thead>
<tr>
<th>Case</th>
<th>Name</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Description</th>
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<tbody>
<tr>
<td>44.</td>
<td>EA</td>
<td>37</td>
<td>F</td>
<td>Phthisis</td>
<td>Emaciated</td>
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<td></td>
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<td>Brain - NAD</td>
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<td>TB nodules</td>
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<td>in both</td>
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<td>lungs++</td>
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<td>with cavities</td>
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<td>and nodules.</td>
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<td>Myocardium</td>
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<td>is pale</td>
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<td></td>
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<td>and flabby.</td>
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<tr>
<td>45.</td>
<td>MT</td>
<td>44</td>
<td>M</td>
<td>Exhaustion</td>
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<td></td>
<td></td>
<td>Labelled TB</td>
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<td>Emaciated.</td>
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<td>Sinuses leading to</td>
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<td>bare bone on</td>
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<td>tibia</td>
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<td>Femur and toe.</td>
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<td>Several</td>
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<td>abscesses.</td>
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<td>Brain substance pale.</td>
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<td>Nodular masses</td>
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<td>scattered throughout</td>
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<td>the lungs of caseous material.</td>
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<td>Aortic valve is</td>
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<td>incompetent and</td>
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<td>there is commencing</td>
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<td></td>
<td>atheroma of aorta.</td>
<td></td>
</tr>
<tr>
<td>46.</td>
<td>ME</td>
<td>25</td>
<td>M</td>
<td>TB peritonitis</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Phthisis</td>
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<td></td>
<td></td>
<td>Much Emaciated</td>
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<td></td>
<td></td>
<td>Brain and grey matter</td>
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<td></td>
<td></td>
<td>thin and pale.</td>
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<td></td>
<td></td>
<td></td>
<td>Heart is anaemic and</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>wasted.</td>
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<td>Valves OK.</td>
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<td></td>
<td></td>
<td></td>
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<td>TB nodules and</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>caseation.</td>
<td></td>
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<tr>
<td>No.</td>
<td>47.</td>
<td>48.</td>
<td>49.</td>
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</tr>
<tr>
<td>Name</td>
<td>MG 46 F</td>
<td>ME 55 F</td>
<td>MA 45 F</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Acute Mania (puerperal)</td>
<td>Congestion of lungs</td>
<td>Exhaustion from colostomy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Manner</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Condition</td>
<td>Pneumonia</td>
<td>Emaciated</td>
<td>Emaciated. Colostomy L groin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Admission Status</td>
<td>Well nourished</td>
<td>Brain - NAD</td>
<td>Brain substance pale.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Notes</td>
<td>Abdomen filled with purulent fluid. Intestines covered with TB nodules. Liver enlarged with waxy degeneration.</td>
<td>Fibrous deposit on epicardium and R ventricle. Aortic valve incompetent – rest NAD. Lung congested with hepatisation. Frothy exudate.</td>
<td>Large white patch on R ventricle, otherwise NAD. Large abscess in abdomen. Contiguous with pelvis and acetabulum, which has tubercular disease.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cause</td>
<td>C ? 2nd attack</td>
<td>D First 6 weeks</td>
<td>A ?</td>
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</tr>
<tr>
<td><strong>Liver enlarged with waxy and fatty disease.</strong></td>
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</tr>
<tr>
<td><strong>50. AG 54 F</strong></td>
<td><strong>ME</strong></td>
<td>-</td>
<td>Cerebral haemorrhage</td>
<td>Well nourished</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>“Bullae” of fluid in R cortex near Sylvian fissure and parietal lobe. I L cortex there is attenuation and much softening. All arteries are thickened and calcareous. Diagram of lesions</td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Lungs emphysematous but otherwise NAD. Aortic valve is competent but has patches of sclerosis at coronary arteries. Mitral valve is sclerosed and thickened and there are calcareous patches on it.</td>
<td></td>
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</tr>
<tr>
<td><strong>51. JH</strong></td>
<td><strong>MA</strong></td>
<td>-</td>
<td>Cerebral apoplexy</td>
<td>Well nourished</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Large extravasation from pons through corpus striatum and optic thalamus. Acute softening of around haemorrhage</td>
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</tr>
</tbody>
</table>
and in corpus callosum.
Arteries at base are atheromatous.
Heart hypertrophied.
Valves competent.
Numerous patches of atheroma on aorta above valve. Small patch of atheroma on mitral valve.
Lungs congested.

<p>| 52. DS 46 M | MA | - | Intestinal obstruction from volvulus | Somewhat emaciated | Brain “somewhat shrunken” with deep sulci. Valves are competent. There is atheroma of aorta. Some congestion of lungs |
| 53. AMcK 43 F | MA | - | Ca breast | Greatly emaciated. Cancerous nodules all over trunk and head. Large Ca ulcer over scar of amputated R breast | Brain - NAD Heart - NAD. Nodes in lung, which are collapsed. Purulent lymph. |
| 54. BC 29 M | ME | - | Phthisis | Much emaciated. | Brain anaemic but no atrophy |</p>
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</tr>
<tr>
<td>55.</td>
<td>ME</td>
<td>-</td>
<td>Phthisis</td>
<td>Lungs infiltrated with TB nodules. Heart normal if anaemic.</td>
</tr>
<tr>
<td>J B</td>
<td>54</td>
<td>M</td>
<td></td>
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</tr>
<tr>
<td>56.</td>
<td>ME</td>
<td>-</td>
<td>Pneumonia</td>
<td>Brain anaemic with some atrophy. R lung infiltrated with TB nodules and cavities with haemorrhage (said to be cause of death). Cardiac muscle pale and flabby. Thickening of mitral cusps.</td>
</tr>
<tr>
<td>Alexander Bateman</td>
<td>53</td>
<td>M</td>
<td></td>
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<tr>
<td>57.</td>
<td>MA</td>
<td></td>
<td>Phthisis</td>
<td>Grey matter anaemic and thin.</td>
</tr>
<tr>
<td>A R</td>
<td>37</td>
<td>M</td>
<td></td>
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</tr>
<tr>
<td>F</td>
<td></td>
<td></td>
<td></td>
<td>Signs of pericarditis. Valves are competent and myocardium normal. There is no atheroma of aorta. Lungs infiltrated with TB nodules and cavities.</td>
</tr>
<tr>
<td>58. DB 21 F</td>
<td>MA</td>
<td>-</td>
<td>Phthisis</td>
<td>Much emaciated</td>
</tr>
<tr>
<td>59. MM 26 M</td>
<td>ME</td>
<td>-</td>
<td>Phthisis</td>
<td>Some bedsores</td>
</tr>
<tr>
<td>60. AF 35 M</td>
<td>ME</td>
<td>-</td>
<td>Phthisis</td>
<td>Much emaciated</td>
</tr>
</tbody>
</table>

Brain shows atrophy with softening Lungs infiltrated with TB caseous nodules. Heart normal if anaemic. Cardiac muscle pale and flabby. Thickening of mitral cusps.

Brain shows atrophy with softening R lung infiltrated with TB nodules and cavities. Signs of pericarditis. Cardiac muscle NAD.

Brain pale and anaemic with softening. Cardiac muscle pale and flabby There is atheroma of aorta.
<p>| | | | | |</p>
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<thead>
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</thead>
<tbody>
<tr>
<td><strong>61. JS 53 F</strong></td>
<td>ME</td>
<td>-</td>
<td>Rupture of bowel</td>
<td>“III” constructed adult.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Considerable atrophy of L frontal lobe.</td>
</tr>
<tr>
<td></td>
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<td>Lateral ventricles enlarged++ No softening.</td>
</tr>
<tr>
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<td></td>
<td></td>
<td>Signs of pericardial fluid. L ventricle hypertrophied.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Thickening of mitral cusps. Slight congestion of lungs.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Liver congested.</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Massive distension of large bowel. Three inches of gangrenous bowel with a rupture.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Evidence of scybala.</td>
</tr>
<tr>
<td><strong>62. SS 50 F</strong></td>
<td>MA</td>
<td>-</td>
<td>Pneumonia</td>
<td>Much emaciated.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Brain- Not examined (“ examination conducted in haste”)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Heart NAD.</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Fracture of rib with evidence of pleurisy.</td>
</tr>
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<td></td>
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<td></td>
<td>Evidence of consolidation at base.</td>
</tr>
<tr>
<td><strong>63. JR 53 M</strong></td>
<td>ME</td>
<td>-</td>
<td>Exhaustion from melancholia and Phthisis Pneumonale</td>
<td>Very much emaciated.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Grey matter pale and anaemic, soft and friable.</td>
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</tr>
<tr>
<td><strong>Signs of pericardial fluid. Cardiac muscle pale and flabby with mucoid degeneration.</strong></td>
<td><strong>R lung infiltrated with TB nodules and cavities. Little functional lung.</strong></td>
<td></td>
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</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>64.</th>
<th>WG</th>
<th>48 M</th>
<th>Acute delirious mania</th>
<th>Pneumonia</th>
<th>Well developed</th>
<th>Grey matter is of normal thickness and good colour. Punctate vasculosa are very marked as are the white matter. Pericardium inflamed. Cardiac muscles rather flabby and probably affected with cloudy swelling. Vegetation on mitral valve with congestion affecting mitral cusp. Red hepatisation in lung with consolidation</th>
</tr>
</thead>
<tbody>
<tr>
<td>65.</td>
<td>CY</td>
<td>49 F</td>
<td>Domestic servant</td>
<td>Cardiac Disease</td>
<td>Very oedematous</td>
<td>Brain substance was very oedematous. Ventricles enlarged. Otherwise, NAD.</td>
</tr>
</tbody>
</table>

C Single. Second attack. Sudden onset... a
<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Disease</th>
<th>Condition</th>
<th>Additional Details</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Pneumonia</td>
<td>Well developed</td>
<td>Not examined - “Friends waiting to take him”. Left ventricle dilated and L ventricle myocardium thinned. Valves NAD. Pleura adherent. Both lungs congested.</td>
</tr>
<tr>
<td>66. PG</td>
<td>MA</td>
<td>-</td>
<td>Well developed</td>
<td>Brain - NAD. Heart - NAD. Lungs – old TB.</td>
</tr>
<tr>
<td>67. DMcL</td>
<td>?</td>
<td>Garden labourer</td>
<td>Cut throat</td>
<td>Well developed. Large wound from near mastoid to midline severing muscles, jugular vein and I carotid artery; also, a second wound through first</td>
</tr>
</tbody>
</table>

Aortic valve was competent but showed atheroma. Tricuspid valve dilated. Myocardium was slightly fatty. Lungs were very oedematous and showed old TB (clinical notes said phthisis at one point).
<table>
<thead>
<tr>
<th>No.</th>
<th>Age</th>
<th>Sex</th>
<th>Occupation</th>
<th>Disease</th>
<th>Symptoms</th>
<th>Initial Observation</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>68.</td>
<td>JN</td>
<td>50</td>
<td>F</td>
<td>Mania</td>
<td>Crofter</td>
<td>Congestion of lungs</td>
<td>Brain essentially normal. No atheroma of basal vessels L ventricular hypertrophy. Myocardium seems healthy. Atheroma and fatty degeneration of aorta commencing. Apices emphysematous. Marked oedema and congestion of both lungs.</td>
</tr>
<tr>
<td>69.</td>
<td>MI</td>
<td></td>
<td>MA</td>
<td>Agricultural labourer</td>
<td>Cardiac Disease</td>
<td>Very stout</td>
<td>Brain NAD Pleural effusions. Lungs congested. L and R ventricular hypertrophy. All valves incompetent. Liver-acutely congested. Ascites.</td>
</tr>
<tr>
<td>70.</td>
<td></td>
<td></td>
<td>MA</td>
<td>Tool finisher</td>
<td>Phthisis Pneumonale</td>
<td>Emaciated somewhat</td>
<td>Brain - NAD Heart NAD Lungs TB cavities ++</td>
</tr>
<tr>
<td>71.</td>
<td></td>
<td></td>
<td>JE</td>
<td>Ploughman</td>
<td>Phthisis Pneumonale</td>
<td>Very much emaciated</td>
<td>Brain - NAD Heart dilated and walls thin. L ventricle hypertrophied</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lungs – collapsed, caseation material. Abscesses and pus.</td>
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</tr>
<tr>
<td>72.</td>
<td>GB</td>
<td>ME</td>
<td>Labourer</td>
<td>Phthisis Pneumonale</td>
<td>Very much emaciated</td>
<td>Brain NAD Heart pale and anaemic. Nodules throughout both lungs.</td>
<td></td>
</tr>
<tr>
<td>43</td>
<td>M</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>D ?</td>
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</tr>
</tbody>
</table>

**GB**

**ME**

**Labourer**

**Phthisis Pneumonale**

**Very much emaciated**

**Brain NAD Heart pale and anaemic. Nodules throughout both lungs.**

**D ?**
Appendix E. Post-mortem reports in cases with melancholia or mania dying at or under the age of 55 in Fair Mile, 1896-1905.

MA = Mania ME = Melancholia

*Length of stay categories:-  A – less than a week, B – 1-4 weeks,  C – 4 weeks- 12 months, D – 1-5 years, E – more than 5 years.

<table>
<thead>
<tr>
<th>Number</th>
<th>Initials</th>
<th>Clinical diagnosis on admission</th>
<th>Occupation</th>
<th>Cause of death</th>
<th>External post-mortem findings</th>
<th>Internal post-mortem findings</th>
<th>Length of stay group Number and length of Attack</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>SP</td>
<td>MA</td>
<td>Housewife</td>
<td>Morbis Cordis</td>
<td>Well nourished</td>
<td>Brain, grey matter and white matter pale Mitral valves incompetent. Myocardium thin at apex and pale and soft. Lungs congested. No sign of TB</td>
<td>E First</td>
</tr>
<tr>
<td>2.</td>
<td>HB</td>
<td>ME</td>
<td>Labourer</td>
<td>Phthisis</td>
<td>Extremely emaciated</td>
<td>Brain, grey matter and white matter pale and shrunken with oedema Heart small and pale Pericardial fluid++ Valves normal Both lungs had TB infiltration and cavities.</td>
<td>B</td>
</tr>
<tr>
<td>3.</td>
<td>ML</td>
<td>MA</td>
<td>Dressmaker</td>
<td>PP</td>
<td>Very much emaciated</td>
<td>Brain - Congested and “hyperaemic” but NAD Heart small and pale Pericardial fluid++.</td>
<td>D</td>
</tr>
<tr>
<td>No.</td>
<td>Name</td>
<td>Age</td>
<td>Gender</td>
<td>Occupation</td>
<td>Phthisis</td>
<td>Emaciation</td>
<td>Clinical Details</td>
</tr>
<tr>
<td>-----</td>
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</tr>
<tr>
<td>4.</td>
<td>SB</td>
<td>42</td>
<td>F</td>
<td>Housewife</td>
<td>Phthisis</td>
<td>Very much</td>
<td>Suppurating TB foci in both lungs</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>emaciated</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Brain - NAD but vessels atheromatous.</td>
</tr>
<tr>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Lumbar spine had mucous -purulent mass</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Heart normal</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lungs show phthisis and cavities</td>
</tr>
<tr>
<td>5.</td>
<td>MP</td>
<td>34</td>
<td>F</td>
<td>Cook</td>
<td>Phthisis</td>
<td>Very much</td>
<td>Brain softened but otherwise NAD</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>emaciated</td>
<td>Valves normal.</td>
</tr>
<tr>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td>Myocardium flabby with some atheroma.</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lungs showed signs of TB.</td>
</tr>
<tr>
<td>6.</td>
<td>MW</td>
<td>52</td>
<td>F</td>
<td>None</td>
<td>Ca rectum</td>
<td>Emaciated</td>
<td>Brain anaemic, white matter oedematous</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Heart was small size and myocardium soft and friable.</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td>Valves normal. Lungs congested and secondary Ca in one lung.</td>
</tr>
<tr>
<td>7.</td>
<td>TT</td>
<td>52</td>
<td>M</td>
<td>Agricultural Labourer</td>
<td>Advanced TB</td>
<td>Very much</td>
<td>Brain shrunken and anaemic, white matter oedematous</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>emaciated</td>
<td>Pericardial thickening and fluid++. Heart pale and friable.</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Valves dilated. Patches of atheroma.</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>TB masses and cavities</td>
</tr>
<tr>
<td>8.</td>
<td>MD</td>
<td>53</td>
<td>F</td>
<td>None</td>
<td>Heart disease</td>
<td>Very much</td>
<td>Grey and white matter congested</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>emaciated</td>
<td>Myocardium soft and friable. Valves OK.</td>
</tr>
<tr>
<td>#</td>
<td>Sex</td>
<td>Age</td>
<td>Occupation</td>
<td>Diagnosis</td>
<td>Status</td>
<td>Cause of Death</td>
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<td>No.</td>
<td>Name</td>
<td>Age</td>
<td>Sex</td>
<td>Occupation</td>
<td>Disease</td>
<td>Condition</td>
<td>Cause of Emaciation</td>
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<tr>
<td>12.</td>
<td>MK</td>
<td>42</td>
<td>M</td>
<td>School Mistress</td>
<td>Heart Disease</td>
<td>Much emaciated</td>
<td>Brain soft. Both grey and white matter congested and oedematous. Some pericardial fluid. Mitral valve somewhat dilated. Myocardium soft. Atheroma of some vessels.</td>
</tr>
<tr>
<td>15.</td>
<td>HR</td>
<td>48</td>
<td>M</td>
<td>Labourer</td>
<td>Phthisis</td>
<td>Very much emaciated</td>
<td>Brain pale. White matter oedematous NAD. Basal vessels show atheroma. Heart small and flabby. Lungs show TB tubercle and cavities”.</td>
</tr>
<tr>
<td>No.</td>
<td>Code</td>
<td>Age</td>
<td>Sex</td>
<td>Occupation</td>
<td>Condition</td>
<td>Examination Details</td>
<td>Cause of Death</td>
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<tr>
<td>17.</td>
<td>TB</td>
<td>48</td>
<td>M</td>
<td>Labourer</td>
<td>Exhaustion</td>
<td>Extremely Emaciated</td>
<td>TB</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Meninges engorged.</td>
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<tr>
<td></td>
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<td></td>
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<td></td>
<td>White matter oedematous</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Valves slightly dilated.</td>
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</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>R ventricle thin. Muscle pale and friable.</td>
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<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>Lungs congested.</td>
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<td>First (1 week)</td>
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<tr>
<td>18.</td>
<td>BB</td>
<td>32</td>
<td>F</td>
<td>None</td>
<td>Phthisis</td>
<td>Rather emaciated</td>
<td></td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Grey matter pale. White matter oedematous</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Heart was flabby and degenerated.</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Both lungs much congested and consolidated with cavities and TB deposits.</td>
<td></td>
</tr>
<tr>
<td>19.</td>
<td>GB</td>
<td>36</td>
<td>M</td>
<td>Fitter</td>
<td>Phthisis</td>
<td>Very much emaciated</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Grey matter soft. White matter oedematous</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Heart small. Some pericardial fluid. Lungs show apical consolidation and gangrene in parts.</td>
<td></td>
</tr>
<tr>
<td>20.</td>
<td>MK</td>
<td>34</td>
<td>F</td>
<td>Domestic Servant</td>
<td>Cardiac failure in recovery from typhoid</td>
<td>Emaciated</td>
<td>Not examined Flaccid heart.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lungs not congested. Large bowel ulcers healing</td>
<td></td>
</tr>
<tr>
<td>21.</td>
<td>EW</td>
<td>51</td>
<td>F</td>
<td>Housewife</td>
<td>Phthisis</td>
<td>Emaciated</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Heart was flabby and degenerated.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Lungs congested with purulent cavities.</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>Name</td>
<td>Sex</td>
<td>Occupation</td>
<td>Cause</td>
<td>Loss of appetite</td>
<td>Condition of Brain</td>
<td>Heart</td>
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<tr>
<td>25.</td>
<td>JS</td>
<td>ME</td>
<td>Butcher</td>
<td>Ca bladder</td>
<td>Extremely Emaciated</td>
<td>Grey matter soft. White matter oedematous. Atheroma of basal arteries. Heart was average size but ventricular walls thin. Valves somewhat dilated. Atheroma of arteries. Some pericardial fluid. Some congestion lower lobes LARGErenal abscess. NAD other than Ca mass in bladder</td>
<td></td>
</tr>
</tbody>
</table>

A: First (some days)
<p>| | | | | |</p>
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</thead>
<tbody>
<tr>
<td>27. LS 47 F</td>
<td>MA</td>
<td>Laundress</td>
<td>Exhaustion</td>
<td>Poorly nourished</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>A First (2 weeks)</td>
</tr>
</tbody>
</table>

| 28. SG 53 F | MA | None (Widow) | Cardiac failure | Rather emaciated |
|   |   |   |   | D First (3 months) |

| 29. AF 38 F | MA | Tramp | Phthisis | Emaciated |
|   |   |   |   | Grey matter and white matter pale. Thick pericardium> Myocardium pale and flabby. Pleural fluid Adherent lungs with cavities. |
|   |   |   |   | E ? |

<p>| 30. HD 55 F | ME | Labourer | Peritonitis | Fairly nourished |
|   |   |   |   | Grey matter less firm than normal. White matter somewhat oedematous. |
|   |   |   |   | A ? |
| 32. JS 33 F | Delusional insanity | Cook | Phthisis | Much emaciated | Grey matter and white matter pale. Some pericardial fluid otherwise NAD. TB cavities filled with pus. | D |
| 33. AP 47 F | Recurrent Mania | None | Exhaustion | Emaciated and bruises and bedsores | Grey matter slightly hyperaemic. White matter hyperaemic with punctate vasculosa. Myocardium soft and degenerated. | B Second (five weeks) |</p>
<table>
<thead>
<tr>
<th>No.</th>
<th>Surname</th>
<th>Age</th>
<th>Sex</th>
<th>Occupation</th>
<th>Chest Disease</th>
<th>Heart Disease</th>
<th>Other Findings</th>
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<tbody>
<tr>
<td>34.</td>
<td>HL</td>
<td>52</td>
<td>F</td>
<td>Domestic Servant</td>
<td>Pneumonia</td>
<td>Well nourished</td>
<td>Gyri are wasted especially centrally. Myocardium is soft and rather degenerated. Valves normal. Some congestion at base with pus.</td>
</tr>
<tr>
<td>35.</td>
<td>MR</td>
<td>48</td>
<td>F</td>
<td>Delusional insanity</td>
<td>Phthisis</td>
<td>Emaciated</td>
<td>Grey and white matter anaemic Myocardium is soft and rather degenerated. Lungs: adherent and congested</td>
</tr>
<tr>
<td>36</td>
<td>MH</td>
<td>32</td>
<td>F</td>
<td>None</td>
<td>Phthisis</td>
<td>Emaciated</td>
<td>Grey matter and white matter pale. Some pericardial fluid otherwise NAD. TB cavities filled with pus.</td>
</tr>
<tr>
<td>37.</td>
<td>SAK</td>
<td>41</td>
<td>F</td>
<td>None</td>
<td>Heart Disease</td>
<td>Fairly nourished</td>
<td>Grey matter slightly hyperaemic. White matter normal Myocardium soft and degenerated. Atheroma of valvular arteries and first part of aorta. Lungs NAD</td>
</tr>
<tr>
<td>38.</td>
<td>EG</td>
<td>24</td>
<td>F</td>
<td>None</td>
<td>Enteric fever</td>
<td>Well nourished</td>
<td>Grey matter pale. White matter soft and slightly oedematous Heart and lungs NAD. R iliac fossa filled with pus and bowel dilated</td>
</tr>
<tr>
<td>No.</td>
<td>Sex</td>
<td>Age</td>
<td>Occupation</td>
<td>Disease</td>
<td>Physical Description</td>
<td>Cause of Death/Findings</td>
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<tr>
<td>41. EW</td>
<td>38</td>
<td>F</td>
<td>Nursery Governess</td>
<td>Enteric fever</td>
<td>Fairly nourished</td>
<td>Grey and white matter slightly hyperaemic. Myocardium soft and degenerated. Liver-nodules of fatty degeneration. R iliac fossa filled with pus and bowel dilated with ulcers around ileocecal valve. Mesenteric lymph nodes enlarge. Lungs NAD.</td>
<td></td>
</tr>
<tr>
<td>42. ES</td>
<td>51</td>
<td>F</td>
<td>Housewife</td>
<td>Cerebral softening</td>
<td>Fairly well nourished</td>
<td>Brain is shrunken. Gyri are wasted, sulci gaping. Grey matter pale. White matter is sodden and slightly oedematous.</td>
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<tr>
<td>43. MW 49 F</td>
<td>MA</td>
<td>Housewife</td>
<td>Pneumonia</td>
<td>Fairly nourished</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Atheroma of basal arteries. Myocardium fatty and degenerated. Atheroma of mitral and aortic arteries</td>
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<tr>
<td>44. HH 44 F</td>
<td>ME</td>
<td>None</td>
<td>Broncho-Pneumonia</td>
<td>Emaciated</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>45. HW</td>
<td>Recurrent Mania</td>
<td>Prostitute</td>
<td>Ca uterus</td>
<td>Rather Emaciated</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>No.</td>
<td>Ref.</td>
<td>Age</td>
<td>Sex</td>
<td>Occupation</td>
<td>Condition</td>
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<td>Description</td>
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<tr>
<td>46.</td>
<td>FC</td>
<td>25</td>
<td>F</td>
<td>Domestic Servant</td>
<td>Phthisis</td>
<td>Rather Emaciated</td>
<td>Small fibrous tumour the size of a walnut from the dura pressing on Broca’s lobe. Brain soft and gyri are wasted, sulci gaping. White matter is oedematous. Myocardium flabby and degenerated. Valves and cavities NAD. Some oedema of lungs.</td>
</tr>
<tr>
<td>47.</td>
<td>AM</td>
<td>47</td>
<td>F</td>
<td>Recurrent Mania</td>
<td>None</td>
<td>Emaciated</td>
<td>Pericardium, myocardium and valves all normal. R lung show red hepatisation.</td>
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<tr>
<td>AL</td>
<td>SW</td>
<td>CW</td>
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<td>47</td>
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<td>ME</td>
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<tr>
<td>Dressmaker</td>
<td>Domestic Servant</td>
<td>Domestic Servant</td>
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<tr>
<td>Cerebral softening</td>
<td>Emaciated</td>
<td>Phthisis</td>
<td>Well nourished</td>
<td>Emaciated</td>
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50. AL 47 F
51. AG 40 F
52. SW 38 F
53. CW 53 F
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<th>Name/Initials</th>
<th>Gender</th>
<th>Occupation</th>
<th>Syndrome</th>
<th>Condition</th>
<th>Age</th>
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<td>54.</td>
<td>MAI</td>
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<td>Exhaustion</td>
<td>44</td>
<td>B</td>
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<td>Emaciated</td>
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<tr>
<td></td>
<td>None (workhouse)</td>
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<tr>
<td>55.</td>
<td>TH</td>
<td>M</td>
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<td>Phthisis</td>
<td>40</td>
<td>E</td>
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<td></td>
<td></td>
<td>Emaciated</td>
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</tr>
<tr>
<td></td>
<td>None</td>
<td></td>
<td></td>
<td></td>
<td>Brain soft and gyri are wasted. Grey matter pale. White matter is soft and oedematous. Atheroma of basal arteries. Pericardial sac has fluid. Myocardium anaemic soft and friable. Atheromatous aortic valve. Tubercle numerous in apices and cavities in lower lobes</td>
<td></td>
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<tr>
<td>56.</td>
<td>JR</td>
<td>M</td>
<td>Bricklayer</td>
<td></td>
<td>Exhaustion</td>
<td>40</td>
<td>A</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td>Well nourished</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bricklayer</td>
<td></td>
<td></td>
<td></td>
<td>Convolutions well marked but show some wasting. Grey matter is congested. White matter is congested with punctate vasculosa well-marked. Atheroma of basal arteries.</td>
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<tr>
<td>57. JD</td>
<td>MA</td>
<td>Groom</td>
<td>Exhaustion</td>
<td>Emaciated</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>54 M</td>
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<tr>
<td></td>
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<td></td>
<td></td>
<td>Atheroma of mitral and aortic valves. First part of aorta atheromatous. Myocardium pale and friable. Lungs acutely congested.</td>
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<td></td>
<td>B Second (3 months)</td>
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</tr>
<tr>
<td>58. EE</td>
<td>MA</td>
<td>Domestic Servant</td>
<td>Phthisis</td>
<td>Emaciated</td>
<td></td>
<td></td>
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<tr>
<td>39 F</td>
<td></td>
<td></td>
<td></td>
<td>Grey and white matter pale. White matter is oedematous. Myocardium soft. Valves and cavities NAD. Adherent lungs. Tubercular foci and cavities++</td>
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<td>E ?</td>
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<tr>
<td>59. MM</td>
<td>MA</td>
<td>None</td>
<td>Acute pulmonary TB</td>
<td>Emaciated</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>26 F</td>
<td></td>
<td></td>
<td></td>
<td>Grey and white matter pale. Pericardial sac has fluid. Myocardium anaemic soft and friable. All ventricles dilated.</td>
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<td>D ?</td>
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<td>Case</td>
<td>Age</td>
<td>Sex</td>
<td>Occupation</td>
<td>Disease</td>
<td>Condition</td>
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</tr>
<tr>
<td>60.</td>
<td>40</td>
<td>F</td>
<td>Housekeeper</td>
<td>Morbis Cordis</td>
<td>Emaciated</td>
<td>Grey and white matter hyperaemic. Pericardium adherent. Myocardium soft flabby and degenerated. Atheroma of mitral and aortic valves. Lungs congested and &quot;engorged with blood&quot;.</td>
<td>D First (6 weeks)</td>
</tr>
<tr>
<td>61.</td>
<td>38</td>
<td>F</td>
<td>Housewife</td>
<td>Dysentery</td>
<td>Emaciated</td>
<td>Grey and white matter hyperaemic. Pericardium normal. Myocardium soft otherwise NAD Lungs NAD Whole of ileocecal area and large bowel covered with sloughing ulcers</td>
<td>B ?</td>
</tr>
<tr>
<td>62.</td>
<td>50</td>
<td>F</td>
<td>Seamstress</td>
<td>Exhaustion</td>
<td>Emaciated</td>
<td>Superficial haemorrhage of recent date occupying R side of cranium from posterior and middle fossae. Grey and white matter hyperaemic. Pericardial sac has fluid Myocardium soft and degenerated. Valves and cavities NAD Pleural adherence but hard to read.</td>
<td>C First (6 weeks)</td>
</tr>
<tr>
<td>63.</td>
<td>50</td>
<td>M</td>
<td>Clerk</td>
<td>Exhaustion from acute mania/ Morbis Cordis</td>
<td>Emaciated</td>
<td>Gyri are shrunken. Grey matter thin, soft and congested. White matter soft and</td>
<td>D First (3 months)</td>
</tr>
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<td>No.</td>
<td>Sex</td>
<td>Age</td>
<td>Occupation</td>
<td>Disease</td>
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<td>Cause of Death</td>
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<td>65.</td>
<td>MA</td>
<td>33</td>
<td>Dressmaker</td>
<td>Suffocation from impacted bread in larynx.</td>
<td>Well Nourished. Bread the size of a “cob-nut” found in trachea above vocal cords.</td>
<td>Normal.</td>
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<td>70.</td>
<td>EB</td>
<td>36</td>
<td>F</td>
<td>MA</td>
<td>Tailoress</td>
<td>Exhaustion</td>
<td>Emaciated.</td>
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<td>Pericardium, myocardium, valves and cavities NAD. R Lung shows acute congestion, less at base of L lung.</td>
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<td>71. MW 55 F</td>
<td>MA</td>
<td>Nurse</td>
<td>Colitis</td>
<td>Fairly Emaciated. Grey and white matter hyperaemic. There is an excess of punctate vasculosa. Myocardium soft and some signs of degeneration. Slight atheroma of aortic and mitral valves. Lungs emphysematous. Liver fatty change. Large bowel has petechia and ulcers. Rest of bowel NAD but mesenteric glands enlarged and soft.</td>
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<td>No.</td>
<td>Age</td>
<td>Sex</td>
<td>Occupation</td>
<td>Clinical Finding</td>
<td>Description</td>
<td>Cause of Death</td>
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<td><strong>76.</strong>&lt;br&gt;MH&lt;br&gt;47&lt;br&gt;F</td>
<td>MA</td>
<td>None</td>
<td>Phthisis</td>
<td>Emaciated</td>
<td>Grey and white matter pale. Myocardium soft flabby and degenerated. Tubercular consolidation and cavities++</td>
<td></td>
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<tr>
<td><strong>77.</strong>&lt;br&gt;FH&lt;br&gt;44&lt;br&gt;F</td>
<td>ME</td>
<td>Housewife</td>
<td>Pneumonia</td>
<td>Fairly Emaciated</td>
<td>Brain extremely soft. On section, grey matter congested. White matter oedematous. Myocardium flabby and degenerated. Both ventricles dilated. Valves normal. Both lungs show consolidation and congestion at bases.</td>
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<td><strong>78.</strong>&lt;br&gt;KL&lt;br&gt;49&lt;br&gt;F</td>
<td>MA</td>
<td>Housekeeper</td>
<td>Phthisis</td>
<td>Emaciated</td>
<td>Grey matter anaemic otherwise NAD Pericardium normal. Myocardium soft and flabby. Valves and chambers normal. Lungs show cavities and tubercular foci.</td>
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<td>No.</td>
<td>Age</td>
<td>Sex</td>
<td>Occupation</td>
<td>Diagnosis</td>
<td>Clinical Details</td>
<td>Case</td>
<td>Fate</td>
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<tr>
<td>80</td>
<td>RG 22 M</td>
<td>MA</td>
<td>Farmer's son</td>
<td>Phthisis</td>
<td>Fairly Emaciated</td>
<td>Grey matter anaemic and white matter pale. Pericardial fluid ++ otherwise NAD. Tubercular consolidation and cavities ++</td>
<td>C</td>
</tr>
<tr>
<td>83</td>
<td>MS $$ F</td>
<td>MA</td>
<td>Domestic Servant</td>
<td>Fibro-sarcoma of uterus. Septicaemia</td>
<td>Fairly nourished. Large abdominal wound (?surgical) with strangulation ??</td>
<td>Brain soft and gyri are wasted. Sulci gaping. Grey matter pale. White matter sodden and oedematous. Heart NAD Lungs emphysematous with pleural collapse.</td>
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<td>84.</td>
<td>ME</td>
<td>Charwoman</td>
<td>Cardiac failure Subdural haemorrhage</td>
<td>Rather Emaciated</td>
<td>Large recent subdural haematoma involving a number of gyri. Grey matter pale and white matter softer than normal. Atheroma of basal arteries. Pericardium normal. Myocardium soft and friable. Valves and chambers normal. Some basal congestion.</td>
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<tr>
<td>85.</td>
<td>MA</td>
<td>School teacher</td>
<td>Cerebral haemorrhage</td>
<td>Fairly nourished</td>
<td>Skullcap said to be thin. Small “bruises on parietal and occipital regions” - dark green/blackish in colour and contain clotted blood – said to have been there for 3-6 days. Extensive recent haemorrhage in arachnoid space and occupying occipital and parietal regions on both sides. Longitudinal sinus contains clotted blood. Grey and white matter hyperaemic otherwise NAD. Ribs intact. Lungs tinged with blood. Left ventricle shows</td>
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<td>GH</td>
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<td>27</td>
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<tr>
<td>88. MW 53 F</td>
<td>Delusional insanity</td>
<td>Housewife</td>
<td>Fatty heart and bronchitis.</td>
<td>Well nourished.</td>
<td>Grey matter pale. White matter “? With the knife”. Pericardium normal. Myocardium soft and flabby and shows fatty change. L ventricle shows hypertrophy. Aortic valve shows atheroma. Other valves and chambers normal.</td>
<td>E</td>
<td>First 12 days</td>
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<td>#</td>
<td>Sex</td>
<td>Age</td>
<td>Occupation</td>
<td>Cause of Death</td>
<td>Condition</td>
<td>Post-Mortem Findings</td>
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<td>89. AM 24 F</td>
<td>MA</td>
<td>None</td>
<td>Meningitis and pneumonia (labelled as such)</td>
<td>Emaciated. Ulcers on legs and jaw.</td>
<td>White clot in sinus. Pus on leptomeninges and turbid serous fluid elsewhere. Brain soft. Grey and white matter thin. Heart NAD. R lung shows red hepatisation passing into grey.</td>
<td>C ?</td>
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<tr>
<td>90. SS 44 M</td>
<td>MA</td>
<td>Farm labourer</td>
<td>Pneumonia</td>
<td>Fairly nourished with no bruises or signs of violence.</td>
<td>Brain soft and gyri are wasted. Sulci gaping. Brain congested with punctate haemorrhages. Heart especially L ventricle shows marked hypertrophy. Valves normal. Numerous patches of bronchopneumonia. Healed TB foci.</td>
<td>D ?</td>
<td></td>
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<tr>
<td>91. SAS 40 F</td>
<td>ME</td>
<td>Housewife</td>
<td>Pneumonia</td>
<td>Fairly nourished with no bruises or signs of violence</td>
<td>Grey and white matter congested with vessels markedly engorged. Basal vessels healthy. Heart NAD. Lungs show red hepatisation.</td>
<td>B ?</td>
<td></td>
</tr>
<tr>
<td>92. AB 37 F</td>
<td>ME with stupor</td>
<td>None</td>
<td>Pneumonia</td>
<td>Fairly nourished with no bruises or signs of violence</td>
<td>Brain soft. Gyri OK. Grey and white matter congested. Basal vessels healthy. Pericardium normal. Myocardium soft and flabby. All chambers</td>
<td>A ?</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>Age</td>
<td>Sex</td>
<td>Occupation</td>
<td>Cause of Death</td>
<td>Notes</td>
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<td>93.</td>
<td>ET</td>
<td>F</td>
<td>ME and</td>
<td>Ulcerative colitis</td>
<td>Fairly nourished with no bruises or signs of violence.</td>
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<td></td>
<td>44</td>
<td></td>
<td>Domestic</td>
<td></td>
<td>“The head was not examined at the request of friends”</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Servant</td>
<td></td>
<td>Myocardium soft and flabby. All valves and chambers normal.</td>
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<td></td>
<td>Congestion of ileocecal valve and widespread ulcers in large bowel with punctiform haemorrhage.</td>
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<td>94.</td>
<td>MA</td>
<td>F</td>
<td>Governess</td>
<td>Cerebral softening and heart disease (labelled as latter)</td>
<td>Somewhat emaciated</td>
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<td>Brain soft and gyri are wasted. Sulci gaping.</td>
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<td>Grey matter wasted and white matter oedematous.</td>
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<td></td>
<td>Atheroma of basal arteries.</td>
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<td></td>
<td>Pericardium thickened.</td>
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<td>Myocardium soft and flabby. Aortic valve and aorta are calcareous.</td>
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<td>Lungs congested at bases. Healed TB foci.</td>
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<tr>
<td>95.</td>
<td>MA</td>
<td>F</td>
<td>Governess</td>
<td>Phthisis</td>
<td>Somewhat emaciated.</td>
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<td></td>
<td>Heart NAD. Lungs show numerous tubercular foci and cavities.</td>
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<td>Brain soft. Both grey and white matters are pale and anaemic.</td>
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<td></td>
<td>No atheroma of basal arteries.</td>
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<tr>
<td>96.</td>
<td>MA</td>
<td>F</td>
<td>Laundress</td>
<td>Fatty heart and cerebral softening. (labelled as latter)</td>
<td>Fairly nourished with no bruises or signs of violence.</td>
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<td></td>
<td>Brain soft and gyri especially parietal are wasted. Sulci gaping.</td>
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<td>Grey matter wasted and white matter oedematous.</td>
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<td>98. CC 45 M</td>
<td>MA</td>
<td>Labourer</td>
<td>Cardiac failure (morbus cordis)</td>
<td>Fairly nourished with no bruises or signs of violence</td>
<td>Grey matter pale and white matter oedematous. Lateral ventricle enlarged with fluid. No atheroma of basal arteries. Myocardium healthy. L ventricle shows hypertrophy and dilation, as do other chambers to lesser extent. Aortic valve is incompetent and thickened with calcareous nodules. The</td>
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<td>SW</td>
<td>ME</td>
<td>Housewife</td>
<td>Phthisis</td>
<td>Somewhat emaciated</td>
<td>Brain of normal consistency. Both grey and white matters pale and anaemic. No atheroma of basal arteries. Heart NAD. Lungs show numerous tubercular foci and cavities.</td>
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Second
Appendix F. Clinical notes of the two cases of sudden death by suicide in Sunnyside.

D M was 30-year-old married garden labourer who was admitted to Sunnyside with his second attack of melancholia of “unknown” cause of three weeks duration. His previous attack, 5 years previously, was of melancholia with delusions. He had got back to work but not in his previous factory job. He had influenza and then did not sleep for six weeks. On admission, he was “dull and vacant”, spoke little and expressed suicidal and hypochondriacal ideas. A few days later, he was found dead with deep gash from his left ear to the right side of his lower neck cutting everything in its path. He had done this with a pocket knife whose blades had recently been sharpened. According to the report of Dr Howden, the Physician Superintendent, it could not be ascertained how the patient had procured this knife and concealed it. The case was reported to the Procurator Fiscal but there are no details of if or how this was pursued. Dr Howden, about to retire after forty distinguished years of service in the asylum, noted in the case notes that this event “illustrates how effectually a determined suicidal patient can conceal his intention and how he can attain his end by means of simple appliances”.

J R was a married joiner from Kirriemuir in his first attack of melancholia. On admission, he was said not to be suicidal but was described as being “dull, determined, violent and forcible”. He had been a quiet hard-working man. He had had trouble with a subordinate joiner but it was he who gave up work as he worried a lot. He imagined that his wife has been unfaithful with certain parties and “he is to kill them”. He had bought a revolver to shoot one of his “persecutors”. He had delusions that everyone was against him and thought there had been “statements about him in the paper”. He had damaged a knee in the past which had fused but the joint had broken through the skin requiring dressings. He settled and played draughts. He was described as having a contented appearance and was no trouble. Early one morning, he was found dead with his neck firmly tied with the bandage used for his knee. Strangulation was the cause of death and the cases further exemplifies Dr Howden’s maxim.

Post-mortems of both cases were unremarkable.
Appendix G. The nine cases whose cause of death was changed from exhaustion after post-mortem.

1. Cause of death now changed to “Exhaustion with cavities”. Post-mortem showed lung nodules and cavities and was therefore probable TB.
2. Cause of death now changed to “senile decay and diarrhoea”. This 77-year-old lady had a markedly inflamed bowel at post-mortem.
3. Cause of death now changed to “puerperal convulsions, cerebral apoplexy and Bright’s disease”. There was evidence of a cerebral haemorrhage at post-mortem.
4. Cause of death now changed to “paralysis with exhaustion”. There was evidence of a cerebral haemorrhage at post-mortem.
5. Cause of death now changed to “intestinal catarrh” with a markedly inflamed bowel at post-mortem.
6. Cause of death now changed to “exhaustion from cerebral atrophy”. Post mortem showed extensive caseation around calcified lung nodules and was therefore highly probable TB.
7. Cause of death now changed to “exhaustion from cerebral softening”. This 79-year-old man had an “extremely shrunken brain”.
8. Cause of death now changed to “exhaustion from morbis cordis”. This 75-year-old man had an “extremely” enlarged heart.
Appendix H. Two cases which did not fit the pattern of the other 12 cases of senile mania closely.

The first was a 67-year-old single woman who was admitted to Sunnyside in June 1901 in her second attack and who died 12 days later. She had been well up to 10 days before when she had an argument with her brother with whom she lived. This had upset her and she became restless and maniacal shortly afterwards. On admission she appeared exalted, thought that her attendant was God and wanted to kiss him. She had auditory and visual hallucinations and delusions of identity but unlike the rest of the senile manias described in the text there was no mention of memory problems as a context to these delusions. She remained excited, developed food refusal, went down rapidly and died. At the post mortem the brain was not examined but there was evidence of heart disease and atheroma.

The second case labelled senile mania that did not fit with the stereotype outlined in the thesis was a 69-year-old married woman who was admitted to Fair Mile in 1902. Here the problem seems to have been intemperance. This was like her father before her – he was said to be an alcoholic- and she was the only case with a reported history of intemperance in the whole series. Her intemperance was longstanding and was coupled with drinking to excess since going into the workhouse a year previously. She was said to be unclean in habits and often destructive. She was disorientated and had some paranoid ideas based on her lack of grasp. Initially she was very restless but sank quickly and died. At post-mortem there was shrinking of the brain and widespread atheroma in brain in heart. Uniquely amongst all these cases there was degeneration of the liver. It thus appears the label of senile mania for her was a reflection of her age and that her insanity was a consequence of the mental and physical effects of alcohol abuse.
Appendix I.  A case of delirious mania.

E T (Case Book THB 23/5/2/12)

An 18-year-old single woman from Fettercairn, Angus was admitted in her first attack of “unknown cause”. She had been unwell for 8 days. Her admitting Certificates described a state of “great excitement, some at her mother” who she had threatened to kill. She “sings weeps and shouts alternately” and thinks her food is poisoned. She was extremely restless and noisy and moves “exhaustively in an aimless way”. She was particularly restless at night and refuses all food and was tube fed and also given an enema. She was described as having a “low stuttering delirium”. 2 days after admission she “collapsed” and the following day was noted to be “commencing cardiac failure” and the circulation in her left leg was almost absent below the knee. Two days later the limb was “as if” dead. She took “a fair quantity of stimulant” but her “cardiac failure became more obvious”. She sank into a coma and died 9 days after admission. Post-mortem revealed only congestion of organs and nothing specific.
Appendix J. Case summary of patient with potential catatonia

W H Admitted 1\textsuperscript{st} May 1875

A 19-year-old single agricultural labourer from Wantage was admitted in first attack of 4 months duration. The cause was said to be a hereditary predisposition as his uncle was insane. He was physically well built but his heart sounded feeble and pulse was weak. He had gradually become unwell and had to give up work a month before admission. He refused food, only eating “by stealth”. Called to the Lord to have mercy on him. Mental state was said to be melancholic stupor. He was oblivious of surroundings, silent and cataleptic and rigid with beads of sweat on his face. “Silent and stubborn, refusing food with utmost certainty” and “voluntary muscular action cataleptic... remaining in whichever posture he is placed in”. At night he cried out to the Lord to have mercy on him but stopped when attendants approached. Given a stimulant mixture and a cathartic enema. Rallied briefly and said he would like some beer which he was given but soon after became unconscious and died. The cause of death was listed as Exhaustion from melancholia and food refusal. The post-mortem was unremarkable, apart from some mild cerebral congestion.
Appendix K. 11. Collated information from pathological and clinical reports from vascular causes of death in Sunnyside, with condition of mania or melancholia and under 65 years of age at death in study period, 1892-1901.

Mental State Abbreviations:- ME=Melancholia   MA= Mania

*Length of stay categories:- A – less than a week, B – 1-4 weeks, C – 4 weeks - 12 months, D - 1- 5 years, E – more than 5 years.

<table>
<thead>
<tr>
<th>Number</th>
<th>Initials</th>
<th>Date of death</th>
<th>Date of admission</th>
<th>Mental state</th>
<th>Cause of death</th>
<th>Main findings at post-mortem</th>
<th>Main clinical findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>GL 52</td>
<td>13th Oct 1895</td>
<td>20th Nov 1888</td>
<td>MA</td>
<td>Apoplexy</td>
<td>Well nourished—”very corpulent” Bedsores Somewhat congested. Pia etc. L cerebral haemorrhage affecting internal capsule. Thickened cerebral arteries Heart muscle is “pale and flabby” and that of L ventricle thinned Slight hypostatic congestion in both lungs. Fatty infiltration in liver and fat++ in omentum</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>RB 52</td>
<td>10th Dec 1897</td>
<td>13th April 1895</td>
<td>MA</td>
<td>Valvular disease of heart</td>
<td>Well nourished Pia opalescent Brain substance was</td>
<td>Married. Farm servant’s wife. Three children 1st attack, of a few days’ duration</td>
</tr>
</tbody>
</table>
congested and showed many vascular “markers”. Vessels at base of brain were atheromatous. Heart was hypertrophied and dilated. Aortic and pulmonary valves were incompetent. Aorta and coronary vessels were diffusely atheromatous. Hypertrophy and fatty degeneration of ventricular muscles. Lung congestion. Epicardial fat Liver congested

Not epileptic, suicidal or dangerous. Certificates. “quite changed” Thinks “someone has seen her and is suffering from those effects” Says she has seen an angel. Had a similar slight attack a few years ago but made an excellent recovery. Suddenly became restless and excited and violent. Deluged child with water under the impression it was a baptism. Sister committed suicide and cousin was an inmate of Sunnyside. Bodily condition:- Indifferent. Pulse regular but 1st mitral sound abnormal. Described as being in state of profound melancholia with occasional impulsive violence. Restless delusional condition. July 20th. Maniacal condition “shouts, preaches and cries”. Continues in similar way. Develops a discharging sinus on her neck. Continues delusional and misidentifies people. Suddenly had fit and died.

<p>| 3. DS 53 | 15th Feb 1892 | 18th Feb 1881 C | MA | Cardiac valvular disease | Well nourished Very dropsied Softening in internal capsule Pleural fluid Both lungs Oedematous. L ventricle and other cavities hypertrophied. Mitral valve incompetent. Aorta NAD Liver enlarged...chronic venous congestion. | - |</p>
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</table>
| **4. JG 42** | 4th March 1893 | 27th Sept 1882 C | MA | Cerebral haemorrhage | Swelling of neck and thighs  
Marked softening of the brain. Seen partic. in post parietal region and corpus callosum. Basal ganglia are in state of "acute yellow softening"  
Heart dilated and hypertrophied.  
Aortic valve incompetent.  
Calcification round coronary arteries Mitral valve stenosed and thickened.  
Liver is of nutmeg variety. |
| **5. MM 55** | 1st July 1893 | 30th May 1891 D | ME | Congestion of lungs | Emaciated.  
Brain - NAD  
Valves are incompetent.  
There is atheroma of aorta.  
Congestion of lungs  
Liver congested |

Aged 53 Married housewife. Suicidal but not epileptic or dangerous  
1st attack of 6 weeks duration.  
History of episodes of depression but never admitted before.  
Lives a quiet life and is of a "saving disposition".  
Episode started suddenly with great depression and delusions. Thinks she has no food or money and refuses to eat.  
Thinks her son is "lost" and she became suicidal. Aunt on mother’s side committed suicide while in similar state.  
Certificates:- Answers questions reluctantly  
"Fixed" delusion she has done wrong. Refuses food but eats "uncleanly articles"
<table>
<thead>
<tr>
<th>Date</th>
<th>Age</th>
<th>Gender</th>
<th>Diagnosis</th>
<th>Physical:</th>
<th>Mental State:</th>
</tr>
</thead>
<tbody>
<tr>
<td>6. AG 54</td>
<td>9th Aug 1893</td>
<td>5th Feb 1874</td>
<td>ME</td>
<td>Cerebral haemorrhage</td>
<td>Bodily condition. Indifferent. CVS and other systems NAD</td>
</tr>
</tbody>
</table>

| Married | 2nd attack. Stated to be 4 days insane. |

Physical Examination. NAD apart from numerous petechial spots over body. Mentally:- Many delusions. Thinks house is full of men and wild animals. Thinks she is the subject of enchantment and that she is to be covered by molten lead to make her into the devil. Hears voices constantly, Violent at times and said by mother to be unmanageable. Previous admission had been for melancholia when she had delusions of suspicion. Now much worse. Remained the same for month of February. Bursts in to hysterical fits of laughter now and then and also demands her release. Over next few years remains general OK but subject to outbursts of excitement with the same delusions as above. Often attacks other harmless...
patients who she thinks are “souffing” her.


8. CY 49  30th May 1899  8th August 1898  C  MA  Cardiac Disease  Very oedematous Brain substance was very oedematous. Ventricles enlarged. Otherwise, NAD Aortic valve was competent but showed atheroma. Tricuspid valve was dilated. Myocardium was slightly fatty. Lungs were very oedematous and showed old TB (clinical notes said phthisis at one point) Single Domestic servant 2nd Attack Few days. ?Cause Talking constantly in incoherent way. Very excitable. Acting in an insane way On examination Pulse 90/min irregular Otherwise, NAD Discharged Perth Asylum April 1897 Mental condition Acute mania Acutely restless. Rambling incoherent speech Delusions of identity. Orientated Course Very overactive and restless. Talkative but incoherent. Sleep poor Oct...began to settle, less talkative. Given “Trimal tid” Improved mentally
<table>
<thead>
<tr>
<th>No.</th>
<th>Name</th>
<th>Date of Birth</th>
<th>Date of Admission</th>
<th>Sex</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Clinical Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>10. MI 52</td>
<td>20th Aug 1892</td>
<td>8th April 1891 D</td>
<td>MA</td>
<td>Cardiac Disease</td>
<td>Very stout brain NAD. Pleural effusions. Lungs congested. L and R ventricular hypertrophy. All valves incompetent. Liver-acutely congested. Ascites.</td>
<td>Restless and excited. Thinks she is surrounded by spirits. Hears noises and is convinced something is going to happen to her e.g. house falling on her. Sometimes shouts out loud. Similar attack 17 years previously which was treated at home. Since then, she has been weak minded but manages to work as an agricultural labourer. Long standing belief that spirits are following her.</td>
<td></td>
</tr>
</tbody>
</table>
More recently sleep less good.
Mental state on admission
Many delusions of suspicion. People around her have designs on her.
Thought attendants meant to drown her. Continues restless and hypochondriacal.
Developed two poisoned wounds on finger and legs.
Health became feeble. Very breathless.
Dropsy rapidly increasing. Southeys tubes in abdomen to draw fluid.
Growing weaker
Appendix L. Collated information from pathological and clinical reports of vascular causes of death in Fair Mile, with condition of MA or ME and under 65 years of age at death in study period, 1896-1905.

Mental State Abbreviations:- ME= Melancholia     MA= Mania

*Length of stay categories:- A – less than a week, B – 1-4 weeks, C – 4 weeks - 12 months, D - 1- 5 years, E – more than 5 years.

<table>
<thead>
<tr>
<th>No.</th>
<th>Initials</th>
<th>Age</th>
<th>Date of death</th>
<th>Date of admission</th>
<th>Length of stay category</th>
<th>Mental state</th>
<th>Cause of death</th>
<th>Main findings at post-mortem.</th>
<th>Main clinical findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>SP</td>
<td>40</td>
<td>8th Nov 1895</td>
<td>29/7/1882 D</td>
<td>D</td>
<td>MA</td>
<td>Morbis. cordis</td>
<td>Well nourished Brain, grey matter and white matter pale. Mitral valves incompetent. Myocardium thin at apex and pale and soft. Lungs congested. No sign of TB. Liver congested and soft.</td>
<td>Case notes missing Admission Register Cause : Heredity Health indifferent First attack of 4 weeks duration</td>
</tr>
</tbody>
</table>
| 3. MW | 22nd Feb 1905 | 1/12/1897. | MA | Bronchitis and fatty heart | Well nourished. Grey matter and white matter pale. Pericardium normal. Myocardium soft and flabby and shows fatty change. L ventricle shows hypertrophy Aortic valve shows atheroma. Other valves and chambers normal. Some basal pleural adhesions otherwise NAD. | Married, Reading Heredity and drink 2nd Class, feeble 12 days First Housewife. First attack of 12 days duration. Normally intelligent Second marriage 7 years ago, never lived happily with him. One miscarriage by first husband. Living comfortably. Intemperate and not taking food well. One brother was insane. |}

Feeble woman of anaemic complexion, many teeth missing. Body poorly nourished musculature poor. Pulse 100/min, poor. Mitral systolic murmur. Mental state. Restless and agitated. Much talk to herself, incoherent. Talks about the Queen a lot saying, “It’s a rum thing”. Thinks she has been wicked and was wicked even before she was born. Memory poor. Sleeps poorly but better with chloral. Food intake poor as she says she cannot afford it. Intermittently agitated requiring hyoscine. Multiple bruises and haematomas. Episode of diarrhoea with fluctuant temp. Remains resistive. Temperature begins to fluctuate. Sounds at bases Dies 25th Aug 1896
Liver fatty change. Changes of old typhoid?

12 days ago, suddenly accused husband of using electricity on her. Left house and found wandering. From certificates. Is deluded that husband wants to kill her with electricity or poison her with black powder. Refused food since. Excitable at times.

Physical; well nourished. Pulse 76 small. 1st heart sound faint. Two very small bruises on forearms. No ataxia.

Mental. Marked delusions. Memory and orientation poor. Thinks her husband wants to poison her so he can marry another woman. Continues to express delusions. Sleeps and eats quite well. Begins to take more unintelligibly. 1908.. much the same. Bodily health good. Feb Develops fluctuating fever. Sounds at R apex. Given medication but sinks into coma and dies.

<table>
<thead>
<tr>
<th>4. KK</th>
<th>18(^{th}) Aug 1905</th>
<th>E</th>
<th>MA</th>
<th>Cerebral softening and heart disease (labelled as latter)</th>
<th>Somewhat emaciated with no etc. Brain soft and gyri are wasted. Sulci gaping. Grey matter wasted and white matter oedematous. Atheroma of basal arteries.</th>
</tr>
</thead>
</table>

Single, Farringdon. Transferred from Bethlem Hospital. Heredity and climacteric. 2\(^{nd}\) Class, thin and feeble. 14 months. First. Single governess. Causes are said to be heredity, shock and the climacteric. mother’s death one year.
Pericardium is thickened. Myocardium soft and flabby. Aortic valve and aorta are calcareous. Lungs congested at bases. Healed TB foci. Abdo - NAD before that. Well nourished. Pulse and heart and lungs normal. Mental state. Manic state, very talkative. Hears voices especially at night of men who have come to her room. Content unclear. Thinks this relates to shape of Admission order hard to read. Evidence of delusions and complains of voices when no one in room. Probable neologisms. Physical:- Skin looks emaciated, looks anaemic. Senile arcus Duration is at least 6/12. Thinks doctor has been feigning voices while they have been talking. Incoherent at times. Course. Very talkative. Talks to herself. Says she will have justice. Can be abusive to staff. Suspicious and restless Remains deluded and acting out hallucinations. In 1905 noted to be in heart failure with marked oedema of legs. Sinks rapidly. Becomes weak and dies.

5. HH 26
16th Feb 1897
13th April 1894
MA
Cardiac failure
Well nourished

Single, Newbury (admitted from Knowle Asylum)
Heredity
First class
24-year-old single lance corporal. First attack, duration 2 years and 4 months. Mother is an inmate of the asylum and an uncle on his father’s side was also insane. Present attack commenced suddenly. Tried to
| 6. MK 41 | 29th March 1897 | 30th July 1881 E | MA | Heart disease and Addison’s disease | Much emaciated Brain soft. Both grey and white matter congested and oedematous. Some pericardial fluid. Mitral valve somewhat dilated. Myocardium soft. Atheroma of some vessels. Liver passive congestion and nodular. . | Single, Reading Unknown Health indifferent 3 days First Single school mistress aged 25. Transferred from Shrewsbury Asylum where she has been resident since 1880. No FH From admission records: - Deluded about receiving a telegram from “papa” who said he was coming to see her. Feels that worms have come away from her brain. Barricaded herself in her bedroom as she thought enemies were breaking into the house. Physically: - Indifferent health and reduced condition. Heart and lungs healthy. Mentally: - Peculiar manner and deportment. Subject to delusions and hallucinations of sight and sound. Voices like telephones appear. | strangle himself. Admitted to another Asylum initially. Physical. Palate rather vaulted. Body well nourished, musculature good. Pulse 72/min. Heart and lungs NAD. Intermittently abusive and aggressive. At other times dull and vacant though laughs loudly and raucously for no apparent reason. Works well. Sleep and health good. Begins to collect and hoard rubbish Said to be full of delusions. Tied a ligature round his penis “to benefit his eyesight”. |
to her at night and that the spirits of her mother and father appear to her at night and converse with her. Intermittently excitable and abusive but most of the time industrious. Pattern as above continues. Intermittently accuses others of stealing possessions and threatens to kill them. Health varies between good and indifferent. Eats badly.

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<tr>
<td>7. MK 34</td>
<td>10th Sept 1898</td>
<td>?</td>
<td>MA</td>
<td>Cardiac failure in recovery from typhoid</td>
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<tr>
<td>8. SG 51</td>
<td>16th April 1899</td>
<td>26th Jan 1897 D</td>
<td>MA</td>
<td>Cardiac failure</td>
</tr>
</tbody>
</table>

51-year-old widow, of no occupation. First attack, duration 3 months. Husband died 18 years ago. Two grown up children, one died in infancy. Not been in workhouse, living comfortably. Refuses food on occasions. Is intemperate. No FH Gradually getting worse over past 3 months. On certificates. Said she had found the philosopher's stone. Electric wires are in chimneys of houses and Hospitals. Thinks electricity is tearing her eyes out. Her meat has been poisoned by vitriol. Quite robust and well nourished, superficial capillaries injected.
<table>
<thead>
<tr>
<th>9. SAK 41</th>
<th>11th Feb 1901</th>
<th>9th Dec 1891 E</th>
<th>MA</th>
<th>Heart disease</th>
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<td>Fairly nourished</td>
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<td>Grey matter slightly hyperaemic. White matter normal.</td>
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<td>Myocardium soft and degenerated.</td>
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<td>Atheroma of valvular arteries and first part of aorta.</td>
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<td></td>
<td>Lungs NAD</td>
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<td>Liver- nodules of fatty degeneration.</td>
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<td>Married, Wokingham</td>
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<td></td>
<td></td>
<td>Lactation</td>
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<td>Exhausted, 2nd class 6 days First</td>
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<td>Married to farm labourer. Third attack of 5 days duration.</td>
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<td>Cause said to be exhaustion from lactation from too frequent pregnancies.</td>
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<td>Married 10 years . has four children, the last 7 months ago. One died of croup. Also had 6 miscarriages, the last said to be 3 months ago.</td>
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<td>Normally intelligent and temperate.</td>
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<td>No FH. Said to be dangerous to</td>
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</table>
others and suicidal – attempted to jump out of window. 
Mental state. Rambling and incoherent Attention poor. Talking to herself all the time. 
Given belladonna and strychnine plus others unreadable. 
Sleeps well sometimes but at others awake all night. Remain incoherent and talkative. Making no progress. Occasionally more tractable. 
Described as a very noisy chronic. 
Notes continued Vol 1 p 126.

<table>
<thead>
<tr>
<th>Date</th>
<th>Date</th>
<th>Mental</th>
<th>Physical</th>
<th>Mental</th>
<th>Physical</th>
</tr>
</thead>
<tbody>
<tr>
<td>10. OL</td>
<td>23rd Aug</td>
<td>12th July</td>
<td>MA</td>
<td>Morbis Cordis</td>
<td>Well nourished</td>
</tr>
<tr>
<td>43</td>
<td>1901</td>
<td>1899 D</td>
<td></td>
<td></td>
<td>Brain soft. Gyri are wasted. Grey matter pale.</td>
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<td>White matter is slightly oedematous</td>
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<td>Atheroma of basal arteries.</td>
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<td>Pericardium contains fluid.</td>
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<td>Liver-coarse cirrhosis</td>
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<td>Married, Previous episode 2nd class</td>
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<td></td>
<td>Morbis cordis 1 month. Second</td>
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<td>Married dressmaker. Second attack, one month’s duration.</td>
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<td>She has been married twice, has 3 live children and 2 dead, cause unknown.</td>
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<td>Two months since her last confinement. Child died. In poor general health. Attack began suddenly.</td>
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<td>Admission order. Talks incoherently, cannot keep to subject for more than a second or two. Talks about the Lord and conversations with him.</td>
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<td>Says her eye is to be taken out. Can become very excited.</td>
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<td>Physical. Feeble. Pulse too small, mitral systolic murmur.</td>
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<td></td>
<td>Mental. Mildly excited. Voluble and talk incoherent with shifting themes.</td>
</tr>
</tbody>
</table>
Hears voices from the Lord and from her children who tell her what to do. Says there is electricity in the looking glass which makes her chatter. Says the cat talks to her. Remains very talkative. Continues to hear voices especially from the cat. Eats well but health only moderate. Smiles to herself but also grimaces. Can be troublesome, suddenly attacked a nurse. March 8th, 1900. Sudden onset of pre-cordial chest pain. Heart racing. Given digitalis. Settles but in June gets marked "cardiac dyspnoea". Becomes markedly oedematous. Increasing "failure of cardiac compensation". Dies. Said to have morbis cordis (valvular).


Housekeeper
Widowed, Reading
Drink and morbis cordis. 2nd class, heart disease. 6 weeks. First Husband died 8 years ago of TB. First attack of 6 weeks duration. One child alive, one died in infancy and one died of TB 3 months ago. No FH. Has weak heart and says she has a FH of that. Drinking to excess but temperate for last month. Admission order. Says that people are disappearing and being murdered. They are trying to get at her. House is full of dead people. Says the policeman is a murderer in stolen clothes. Daughter confirms
increasing restlessness and use of bottle.

Bruises on thighs.
Mental. Restless. Thinks that everyone is disappearing and that burglars are in the house. Thinks she should protect everyone. Restless and refuses food.
Continues very restless. Tears at clothes. Said to be very depressed several times
Clinical cause of death given as pericarditis and morbis cordis.

| 12. AY | 8th June 1903 | 29th Oct 1901 D | Syncope (labelled as choking) | Emaciated. Signs of tracheostomy. Trachea filled with hard pulp. Brain soft and gyri are wasted. Sulci gaping. Grey matter pale. White matter sodden and oedematous. Left ventricle hypertrophied. Mitral and tricuspid valve slightly incompetent. Occasional TB foci. | Married housewife Second attack Duration 18 months, 2nd class (morbis cordis) Not suicidal Married 4 years. 3 living children, one died, 6 months since the last one Living comfortably, clean, good habits No FH History hard to read. Says someone is stealing her food. |
People watch her. Something about stomach contents and her breast.
Probable delusions.
Feeble, physically. Feeble musculature.
Grey complexion
Pulse 80/min.
Aortic systolic bruit.
Mental state: delusions and hallucinations. Auditory and olfactory
Someone has bewitched her. Hears voices. Thinks her neighbour is involved.
Continues much the same. Has brief fever following attack of facial erysipelas
Sleeps well but has delusions and hallucinations. Thinks there is poison in her bed and people coming to kill her.
Passed bright blood per rectum which did not recur.
Uterus is much enlarged.
Weak and anaemic
Liver is possibly enlarged but no growth.
Sudden syncope when having breakfast. Doctor called and artificial respiration tried. Lot of food in mouth.
Cause of death Syncope while having breakfast (verdict of Coroner jury)

<table>
<thead>
<tr>
<th>Date</th>
<th>Death Date</th>
<th>Initials</th>
<th>Diagnosis</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>54</td>
<td>1903</td>
<td>1902</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>ME</td>
<td></td>
<td>Married, Windsor Heart disease 3rd class. 2 weeks First 53-year-old married housewife. First attack, duration 14 days. Wife of a policeman. No children</td>
</tr>
</tbody>
</table>


Indifferent health. Rheumatic fever 5 years ago. Suffers from fainting fits. Normally cheerful, temperate. FH. Mother committed suicide. Became depressed, stopped eating food. Lost interest in habits or surroundings. Depressed with a dreadful feeling. Sees people around her when there is no one. Says she can see a rabbit with one eye and electricity coming from fingers. Left alone she attempts to climb out window saying, “I’ll join the dead” “I see the dead”. Tells people to escape from her and that she will pray for them. Feeble old woman. Poorly nourished. Poor musculature. Pulse 64/min of poor volume. Vessels “atheromatous”. Heart action feeble, apex beat imperceptible. Systolic bruit. Mental state. Very depressed. Memory poor. Feels frightened. Remains very depressed, constantly weeping and saying, “don’t send me away”. Heart action weaker and dies. Cardiac disease.

14. GH 27
10th Dec 1904 9th Dec 1904 A MA Cerebral haemorrhage Fairly nourished Bruises R arm and scalp. No skull fracture or “signs of violence” School teacher Single, Farrington Over study
Skull cap said to be thin. Small “bruises on parietal and occipital regions” - dark green/blackish in colour and contain clotted blood – said to have been there for 3-6 days. Extensive recent haemorrhage in arachnoid space and occupying occipital and parietal regions on both sides. Longitudinal sinus contains clotted blood. Grey and white matter hyperaemic otherwise NAD.
Ribs intact.
Lungs tinged with blood L ventricle shows marked hypertrophy otherwise NAD.
NAD

“Heredity neurotic”. Single school teacher in her first attack of 14 days duration.
 Normally in good general health.
Intelligent and temperate.
No FH
Said to be over anxious in preparing for an examination.
Admission order. Visited four times by doctor. Getting increasingly restless. Has delusion that doctor is someone else.
History from father, also a school master. Changed completely since a trivial boating accident in the summer.
Heart and lungs normal.
Mental. Rambles incoherently. Misidentifies place. Says she has only one foot and that she came here by motor from India.
That night became very restless and excited Talking as if she was in a school exam. Became resistive.
Struggle with nurse ensued.
Snatched nurses cap off and pulled her hair. Taken to the lavatory but 15 mins later apparent she had had a “fit”. Doctor summoned who also called Dr Murdoch. Heart was “acting” but respiration had ceased.
Artificial respiration was attempted and strychnine given without success. Died.
Cause of death cerebral haemorrhage (“Verdict of Coroners Jury”)

NAD
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<tbody>
<tr>
<td>16. JC</td>
<td>6th Jan 1906</td>
<td>24th June 1898 E</td>
<td>MA</td>
<td>Fatty heart</td>
<td>Brain soft and gyri are wasted. Sulci gaping. Grey matter wasted and white matter oedematous. No atheroma of basal arteries Myocardium soft and flabby. L ventricle shows hypertrophy. Aortic valve shows atheroma. Other valves and chambers normal. Lungs emphysematous and congested at bases. Liver fatty change. Otherwise, NAD</td>
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</tbody>
</table>
| 17. CC 45 | 9th Jan 1906 | 28th June 1890 E | MA | Cardiac failure (morbus cordis) | Fairly nourished with no bruises or signs of violence. Grey matter pale and white matter oedematous. Lateral ventricle enlarged with fluid. No atheroma of basal arteries. Myocardium healthy. L ventricle shows hypertrophy and dilation as do other chambers to lesser extent. Aortic valve is incompetent and thickened with calcareous nodules. The aorta is atheromatous. Other valves healthy. Lungs NAD.

Liver congested. Otherwise, NAD


Single, Bradfield.

Unknown Heart disease

2nd Class

Not known 30-year-old single labourer.

Admitted as a criminal lunatic and then transferred to Pauper register on 28/6/1890.

Said to be manic as he was obstinate and defiant. Says that we are taking the breath away from him and by forcing him to work he has gone mad. Said to have peculiar ideas of persecution. Refused food as it was drugged. Saying people are trying to take his blood.

Has episodes when he becomes grandiose. He sees this place as belonging to him. That he has “millions of money” and was going to bring in the military to sweep the place. Expansive ideas of his own importance and sometimes believes he is a Russian Count.

At other times he is sullen and morose. Said to be spiteful and insulting but “fortunately it is seldom he deigns to speak”.

Put a shirt button up his penis to stop it being “injured” and also toenails in his ears which became... |
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<tr>
<th>No.</th>
<th>Name</th>
<th>Date of Birth</th>
<th>Date of Admission</th>
<th>Age</th>
<th>Gender</th>
<th>Diagnosis</th>
<th>Physical Appearance</th>
<th>Medical History</th>
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</thead>
<tbody>
<tr>
<td>19. PL 59</td>
<td>23rd July 1897</td>
<td>26th June 1897</td>
<td>MA</td>
<td>Heart Disease</td>
<td>Fairly well nourished</td>
<td>Brain soft. White matter sodden and oedematous.</td>
<td>Single, of no occupation. First attack of several weeks.</td>
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<tr>
<td>20. EY 57</td>
<td>27 Oct 1900</td>
<td>27th April 1900 C</td>
<td>ME</td>
<td>Pulmonary haemorrhage and gangrene.</td>
<td>Fairly well nourished Gyri shrunken to slight degree. Grey matter hyperaemic. Basal vessels not atheromatous. Myocardium flabby and soft. With fatty degeneration. Atheroma of aortic and mitral valves. Blood clots in lungs. Liver fatty degeneration. Widow of no occupation. In first attack of 7 weeks duration. Suicidal. Son is a patient in asylum, no other FH. Fairly intelligent. Temperate, good habits, not in Union. Actively suicidal for 7 weeks since she became very depressed. Nil specific physically. Mentally very melancholic and vacant. Thinks that people are coming to kill her and draw all her teeth out. Said to remain very depressed and that she is very wicked. Food refusal and fed with the pump. Develops phlebitis.</td>
<td>Living comfortably but stated to be unintelligent. Threatened suicide by drowning. Not refused food. Not in workhouse. Swollen and varicose legs for some time and present attack is said to relate to worry about this. History of tearing off her clothes and attempting to jump out of a window. Feeble, but well nourished. Diastolic murmur in aortic area. Pulse 100/min, small. Varicose ulcers on feet. Said to be both demented and deluded but no details given. No evidence of hallucinations. Restless with poor sleep. Given medication (? name) to no effect. Develops diarrhoea and becomes weaker. Passes a bloody stool and dies.</td>
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</table>
| 21. LP | 16th Feb 1902 | 11th Jan 1902 C | MA | Cardiac failure | Emaciated | Drink and heredity 2nd Class Blind  
First 2 weeks  
Married wife of labourer. In first attack of 14 days duration.  
Normally an intelligent woman but has been in the habit of drinking spirits heavily.  
No FH  
Became dirty and destructive 14 days ago.  
Briefly in Reading Union where she swore a lot.  
Blind – one eye said to be disorganized and cataract in the other.  
Nil specific physically. Bruises on arms  
Raving incoherently. Says this is a police state.  
Impossible to test attention. Uses obscene expressions.  
Overactive and noisy.  
Develops cellulitis of foot, raised temperature and dies.  

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| 22. ES | 23 Dec 1902 | 13th Nov 1902 C | MA | Fatty Heart | Well nourished  
Myocardium extreme fatty change. L ventricle dilated. Atheroma of 1st part of aorta. Tricuspid valve dilated.  
First. Single mat maker, in first attack of one year. Dangerous to others.  
Normally industrious and intelligent but hasn’t worked for one year. Lost his sight in infancy (as did his sister).  
No FH.  
Became “queer” a year ago. In last two months been violent at night.  
Became dull and apathetic, constantly repeating senseless phrases with a religious theme. Odd |   |   |   |   |   |   |
<table>
<thead>
<tr>
<th>Date</th>
<th>Age</th>
<th>Gender</th>
<th>Diagnosis</th>
<th>Physical</th>
<th>Mental</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>23. BA 59</td>
<td>20th April 1903</td>
<td>10th Sept 1901</td>
<td>ME</td>
<td>Cardiac failure</td>
<td>Emaciated</td>
<td>Carpet planner, married with 7 children. First attack.</td>
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<td>Basal vessels very atheromatous.</td>
<td>Not worked for two years.</td>
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<td>Tricuspid v dilated. Lungs oedematous. Liver congested</td>
<td>Also threatened suicide.</td>
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<td>From admission record. Great change, once intelligent and quiet, now assertive and combative.</td>
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<td>Strange beliefs:- thought his mistress was Holland’s gin and that he was knocked over by Puck.</td>
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<td>Physically, poorly nourished and feeble. Heart sounds weak, pulse poor tone.</td>
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<td>Mentally, anxious and emotional. Thinks he has been abducted and this has been caused by his wife’s sister. Says he understands quotations but his family don’t so they think he is insane. Worried about the business as he is not there.</td>
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<td>Remains deluded re sister-in-law.</td>
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</table>
Becomes more rational but still deluded re plot and he sees many were in it.
Intermittently very depressed and was briefly in a cataleptic state.
Had another bout of severe depression with catalepsy and in the middle of this a burst of excitement.
Lost interest in surroundings. Sudden syncope and died.

24. GJ
59
21st May 1901
20th July 1903
ME
Morbis cordis

Emaciated
Basal vessels atheromatous
Myocardium soft pale and fatty.
Valves dilated especially the tricuspid.
1st part of aorta atheroma. Aortic valves thickened. LV dilated and hypertrophied. Lungs show evidence of bronchitis and congestion.
Liver congested

Married with 10 children.
Living comfortably, never in workhouse, but has not worked for past 6 months.
Supposed cause is worry.
Temperate, good behaviour.
Despondent for some time, fearful he would do his family harm.
Refusing food.
Tried to kill himself by hanging.
Says his jaw has “gone” and his face is split into halves.
Actively suicidal.
Emaciated on admission.
Marked arcus senilis. Heart weak and irregular. Pulse 65/min, poor tone, vessels thick.
Looks depressed. Says he has no stomach. Back passage blocked by a body that comes down from his chest.
Course. Sleep poor, food intake poor, hypochondriacal, “very deluded about his viscera”.
Continues this way for many months. Recurrent attacks of bronchitis with “great cardiac embarrassment”.

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<tr>
<td>25. ET</td>
<td>15th Feb 1903</td>
<td>26th Feb 1904</td>
<td>ME</td>
<td>Not stated</td>
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<td>65</td>
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<td>Not nourished</td>
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<td>Brain soft, gyri wasted.</td>
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<td>Grey and white matter oedematous. Basal vessels atheromatous</td>
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<td>Myocardium soft and friable.</td>
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<td>Atheroma of aortic and mitral valves.</td>
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<td>Lungs congested.</td>
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<td>Commencing cirrhosis of liver. Congestion of gut</td>
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<td>Loses strength, dies.</td>
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Housewife. 
Normally an intelligent woman. 
6 months in Union. 
Attempted suicide by drowning. 
First attack followed her husband’s death. Became depressed then disinhibited and troublesome. 
Refuses food. 
Fair health. Pulse 84/min, normal. 
Thyroid enlarged. 
Depressed and agitated. Keeps saying “oh dear”. 
Memory and orientation very poor. 
Developed a tachycardia, raised temp and loose stools and died. 
Annie Rowles 
Married dressmaker in second attack of 3 months duration. 
One child. Intelligent and temperate. 
Threatened to drown herself. 
Food refusal. 
Strong FH of insanity. 
In Littlemore 16 years ago 
Out of work for some time, said to be cause of attack. 
Has become depressed and says she cannot stand this any longer. 
Repeatedly talks of drowning herself. 
Sees her face as damaged. Sleep very poor. 
Mentally, she reports that everything she did went wrong. 
Hears voices talking to her which distresses her. Did attempt to drown herself. Expression is melancholic. 
No findings on physical examination. 
Remains agitated and very depressed.
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<tr>
<th>No.</th>
<th>Name</th>
<th>Date of Birth</th>
<th>Date of Admission</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Symptoms</th>
<th>Course</th>
<th>Outcome</th>
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<tr>
<td>No.</td>
<td>Name</td>
<td>Date of Birth</td>
<td>Date of Entry</td>
<td>Age</td>
<td>Condition</td>
<td>Details</td>
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<td>28. WK 59</td>
<td>20th Dec 1904</td>
<td>9th Dec 1901</td>
<td>MA</td>
<td>Morbis cordis</td>
<td>Fairly well nourished</td>
<td>Liver nodular change.</td>
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<td>Brain soft- otherwise NAD</td>
<td>Mentally, she reports that everything she did went wrong.</td>
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<td>Myocardium soft. Aorta dilated and clotted.</td>
<td>Hears voices talking to her which distresses her. Did attempt to drown herself. Expression is melancholic.</td>
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<td>Valves all incompetent. Marked hypertrophy</td>
<td>No findings on physical examination.</td>
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<td>Lung shows evidence of pneumonia.</td>
<td>Remains agitated and very depressed.</td>
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<td>Abdo NAD</td>
<td>Developed a high temperature and died.</td>
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<td>Unknown</td>
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<td>2nd class</td>
<td>Case notes missing</td>
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<td>First 57 months.</td>
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<td>29. SC 63</td>
<td>4th March 1905</td>
<td>29th May 1883</td>
<td>ME</td>
<td>Morbis cordis</td>
<td>Fairly well nourished</td>
<td>Case notes missing</td>
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<td>Basal vessels atheromatous.</td>
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<td>Myocardium hypertrophied.</td>
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<td>Anterior cusp of mitral valve atheroma. Lungs congested.</td>
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<td>Abdo NAD</td>
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<td>30. LL 60</td>
<td>26th Oct 1905</td>
<td>26th April 1898</td>
<td>ME</td>
<td>Cerebral haemorrhage</td>
<td>Well nourished.</td>
<td>53, widow, stewardess. Transferred from Portsmouth.</td>
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<td>Brain soft, gyri wasted. Probable second attack.</td>
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<td>Large yellow softening on R side affecting large parts of brain. Basal vessels atheromatous.</td>
<td>Rambling and incoherent.</td>
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<td>Continually says “I can’t” or “I couldn’t”. Blames everything on a Mr. Coleman. Says he robbed her of</td>
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<tr>
<td>Myocardium hypertrophied.</td>
<td>all her money. Poor intake of food, constant preoccupation. Continually says “can I sleep in this bed tonight?”. Appears very depressed. Continues like this for long period. Has epileptic fit. Develops fever and changes at lung base. Said to have had a cerebral haemorrhage as cause of death later but no neurological changes described.</td>
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<td>Atheroma of aortic and mitral valves.</td>
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<td>Lungs congested.</td>
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<td>Congestion of liver.</td>
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