

# **Mortality in Victorian Asylums, ca. 1870-1910.**

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

**Thesis submitted in partial fulfilment of the requirements for a**

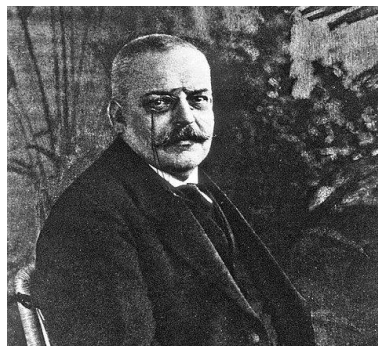
**Doctor of Philosophy**

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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 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!

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## Abbreviations

FH +ve	Family History of insanity
GPI	General Paralysis of the Insane
LCA	London County Asylum
LCC	London County Council
LC	Lunacy Commission
ME	Melancholia
MPA	Medico-Psychological Association
NAD	No Abnormality Detected
REA	Royal Edinburgh Asylum
SD	Standard Deviation
SMR	Standardised Mortality Rate

## Glossary<sup>1</sup>

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

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<sup>1</sup> 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.<sup>2</sup> 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.<sup>3</sup> 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.<sup>4</sup> 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.<sup>5</sup> 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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<sup>2</sup> Rab Houston. “A Latent Historiography? The Case of Psychiatry in Britain, 1500– 1820,” *The Historical Journal* 57 (2014): 291; Andrew Scull, “Madfolk and their Keepers: Roy Porter and the History of Psychiatry,” in *The Insanity of Place/the Place of Insanity*, ed. Andrew Scull (Abingdon: Routledge, 2006), 38-53; Allan Beveridge, “Reading about ... the History of Psychiatry,” *British Journal of Psychiatry* 200 (2012): 431.

<sup>3</sup> Jennifer Wallis, “Post-mortem Records,” in *Sources in the History of Psychiatry, from 1800 to the Present*, ed. Chris Millard and Jennifer Wallis (Abingdon: Routledge, 2022), 50.

<sup>4</sup> Roy Porter. “The Patient’s View: Doing Medical History from Below,” *Theory and Society* 14 (1985): 175.

<sup>5</sup> Tatjana Buklijas, “The Laboratory and the Asylum: Francis Walker Mott and the Pathological Laboratory at London County Council Lunatic Asylum, Claybury, Essex (1895–1916),” *History of Psychiatry* 28, no. 3 (2017): 311–25; Eric J. Engstrom, *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice* (Ithaca, NY: Cornell University Press, 2003); Stephen Jacyna and Stephen Casper, *The Neurological Patient in History* (Rochester, NY: University of Rochester Press, 2012); Tom Quick, “From Phrenology to the Laboratory: Physiological Psychology and the Institution of Science in Britain (c.1830–80),” *History of the Human Sciences* 27 (2014): 54–73; Jennifer Wallis, *Investigating the Body in the Victorian Asylum: Doctors, Patients, and Practices* (Cham, Switzerland: Palgrave Macmillan, 2017); Jonathan Andrews, “Introduction: Lunacy’s Last Rites,” *History of Psychiatry* 23, no. 1 (2012): 3-5.

The phenomenon of premature and frequent deaths 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.<sup>6</sup> 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.<sup>7</sup> 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.<sup>8</sup>

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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<sup>6</sup> Patricia Jalland, *Death in the Victorian Family* (New York: Oxford University Press, 1996); Peter Jupp and Glennys Howarth, *The Changing Face of Death: Historical Accounts of Death and Disposal* (Basingstoke: Macmillan, 1997); Andrews, "Introduction: Lunacy's Last Rites," 3-5; Wallis, *Investigating the Body*; Lynsey Cullen, "Post-mortem in the Victorian Asylum: Practice, Purpose and Findings at the Littlemore County Lunatic Asylum, 1886-7," *History of Psychiatry* 28, no. 3 (2017): 280-96; Elizabeth Hurren, "'Abnormalities and Deformities': The Dissection and Interment of the Insane Poor, 1832-1929," *History of Psychiatry* 23, no. 1 (2012): 65-77.

<sup>7</sup> David Wright, Laurie Jacklin and Tom Themeles, "Dying to Get Out of the Asylum: Mortality and Madness in Four Mental Hospitals in Victorian Canada, c.1841-1891," *Bulletin of the History of Medicine* 87 (2013): 591-621.

<sup>8</sup> Constance McGovern, "The Myths of Social Control and Custodial Oppression: Patterns of Psychiatric Medicine in Late Nineteenth-Century Institutions," *Journal of Social History* 20 (1986): 3-23.

considered and is largely unknown.<sup>9</sup> 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.<sup>10</sup> 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.<sup>11</sup> 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,

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<sup>9</sup> Wright, Jacklin and Themeles, “Dying to Get Out of the Asylum,” 591–621 ; Joseph Melling and Bill Forsythe, *The Politics of Madness: The State, Insanity and Society in England, 1845–1914* (London: Routledge, 2006); Leonard Smith, “Welcome Release; Perspectives on Death in the Early County Lunatic Asylums, 1810–50,” *History of Psychiatry* 23, no. 1 (2012): 117-28; Cathy Smith, “‘Visitation by God’: Rationalizing Death in the Victorian Asylum,” *History of Psychiatry* 23, no. 1 (2012): 104-16; Jonathan Andrews, “Death and the Dead-House in Victorian Asylums: Necroscopy versus Mourning at the Royal Edinburgh Asylum, c. 1832–1901,” *History of Psychiatry* 23, no. 1 (2012): 6-26.

<sup>10</sup> David Cutler, Angus Deaton and Adriana Lleras-Muney, “The Determinants of Mortality,” *Journal of Economic Perspectives* 20 (2006): 97-120.

<sup>11</sup> Smith, “Welcome Release,” 117-28; Melling and Forsythe, *The Politics of Madness*, 255.

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."<sup>12</sup> 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.<sup>13</sup> 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.<sup>14</sup>

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

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<sup>12</sup> Waltraud Ernst, "The Limits of Comparison: Institutional Mortality Rates, Long-term Confinement and Causes of Death during the Early Twentieth Century," *History of Psychiatry* 23, no. 4 (2012): 404-18; Simon Szreter, "Author Response: Debating Mortality Trends in 19th century Britain," *International Journal of Epidemiology* 33 (2004): 707.

<sup>13</sup> Anne Shepherd and David Wright, "Madness, Suicide and the Victorian Asylum: Attempted Self-murder in the Age of Non-restraint," *Medical History* 46 (2002): 175-196.

<sup>14</sup> David Wright, "The Dregs of Society? Occupational Patterns of Male Asylum Attendants in Victorian England," *International History of Nursing Journal* 1 (1996): 5-19; Catherine Smith, "Insanity and the 'Civilising Process': Violence, the Insane and Asylums in the Nineteenth Century," in *Assaulting the Past: Violence in Historical Context*, ed. Katherine D. Watson (Newcastle: Cambridge Scholars Press, 2007), 250-68; Smith, "Behind Closed Doors: Lunatic Asylum Keepers, 1800-60," *Social History of Medicine* 1 (1988): 301-27.

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.<sup>15</sup>

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

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<sup>15</sup> 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.”<sup>16</sup> 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

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<sup>16</sup> Margaret Harris et al., “The Morbidity and Mortality Linked to Melancholia: Two Cohorts Compared, 1875–1924 and 1995–2005,” *History of Psychiatry* 24, no. 1 (2013): 3-14; Marvin Adland, “Review, Case Studies, Therapy, and Interpretation of the Acute Exhaustive Psychoses,” *Psychiatric Quarterly* 21 (1947): 65.

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.<sup>17</sup> 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.<sup>18</sup>

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.<sup>19</sup> Post-mortems were mandated as a method to identify and, more crucially, prevent violent deaths and deaths from

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<sup>17</sup> Andrews, “Death and the Dead-House,” 6-26; Cullen, “Post-mortem in the Victorian Asylum,” 280-96; Hurren, “‘Abnormalities and Deformities’,” 65-77; Wallis, *Investigating the Body*.

<sup>18</sup> Wright, Jacklin and Themeles. “Dying to Get Out of the Asylum,” 591-621.

<sup>19</sup> Hurren, “‘Abnormalities and Deformities’,” 65-77; Chris Philo, “Troubled Proximities: Asylums and Cemeteries in Nineteenth-Century England,” *History of Psychiatry* 23, no. 1 (2012): 91-103.

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.<sup>20</sup> 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 1855 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.<sup>21</sup> 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.<sup>22</sup> 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

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<sup>20</sup> David Zuck, "Mr Troutbeck as the Surgeon's Friend: The Coroner and the Doctors—an Edwardian Comedy," *Medical History* 39 (1995): 260.

<sup>21</sup> Cullen, "Post-mortem in the Victorian Asylum," 280-96.

<sup>22</sup> Jonathan Andrews, "Case Notes, Case Histories, and the Patient's Experience of Insanity at Gartnavel Royal Asylum, Glasgow, in the Nineteenth Century," *Social History of Medicine* 11 (1998): 267.



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.<sup>23</sup> 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.<sup>24</sup> 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.<sup>25</sup> 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.<sup>26</sup> Arguably, congestion, a term with no clear limits,

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<sup>23</sup> Ian Burney, *Bodies of Evidence: Medicine and the Politics of the English Inquest, 1830–1926* (Baltimore, MD: Johns Hopkins University Press, 2000).

<sup>24</sup> Andrews, "Death and the Dead-House," 9.

<sup>25</sup> Hurren, "Abnormalities and Deformities," 65-77; Dorothy MacKinnon, "Bodies of Evidence: Dissecting Madness in Colonial Victoria (Australia)," in *The Body Divided: Human Beings and Medical "Material" in Modern Medical History*, ed. S Ferber and S Wilde (Farnham: Ashgate Publishing, 2012), 75–107.

<sup>26</sup> 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-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.<sup>27</sup> 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.<sup>28</sup> This situation essentially pertained for the many subsequent decades leading to psychiatry being dubbed “the graveyard of Pathology” by Plum in 1972.<sup>29</sup> 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.”<sup>30</sup> 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

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<sup>27</sup> Hurren, “‘Abnormalities and Deformities,’” 65-77.

<sup>28</sup> Dominic Beer, “Psychosis: A History of the Concept,” *Comprehensive Psychiatry* 37 (1996): 273-91.

<sup>29</sup> Fred Plum, “Prospects for Research on Schizophrenia. 3. *Neurophysiology. Neuropathological findings*,” *Neurosciences Research Program Bulletin* 10 (1972): 384-88.

<sup>30</sup> Michael Worboys, “Was there a Bacteriological Revolution in Late Nineteenth-century Medicine?” *Studies in History and Philosophy of Biological and Biomedical Sciences* 38 (2007): 27.

nevertheless lead to practical and theoretical developments that were important to contemporary observers.”<sup>31</sup> 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-quantitatively. 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.

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<sup>31</sup> 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

whatever with the subject in hand.”<sup>32</sup> 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 Moulsoford/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 Moulsoford, in the parish of Cholsey. The asylum was known at first as the Moulsoford 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.

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<sup>32</sup>James C. Howden, “An Analysis of the Post-Mortem Appearances in 235 Insane Persons,” *Journal of Mental Science*, 17 (1871): 83.



Figure 1-1 Engraving of Mouldsford 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.<sup>33</sup>

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<sup>33</sup> Kate Tyte, "Berkshire Old and New: Fair Mile Hospital, Cholsey, 1870-1948" *Journal of the Berkshire Local History Association* 24 (2007), 28-38; Ian Wheeler, *Fair Mile Hospital: A Victorian Asylum* (Stroud: The History Press, 2015) - an invaluable source which draws together many aspects of the founding, design, growth, operation, tribulations, and personages that the hospital witnessed over its 133-year lifespan.

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.<sup>34</sup> 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."<sup>35</sup> Edginton thus makes the case that the design of these asylums was in keeping with the tenets and delivery of moral management.<sup>36</sup> The layout of the Berkshire dormitories, and the asylum more generally, conforms to that principle.

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<sup>34</sup> Markus Reuber, "Moral Management and the 'Unseen Eye': Public Lunatic Asylums in Ireland, 1800-1845," in *Medicine, Disease and the State in Ireland, 1650-1940*, ed. Greta Jones and Elizabeth Malcolm (Cork: Cork University Press, 1999) 208-33.

<sup>35</sup> Barry Edginton, "Moral Architecture: The Influence of the York Retreat on Asylum Design." *Health & Place* 3 (1997): 98.

<sup>36</sup> *Ibid*, 91-9.





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.<sup>37</sup> Records relating to Montrose Royal Asylum exist from 1797, although patient records were not kept in any detail before 1817.

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*.<sup>38</sup> 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.<sup>39</sup>

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

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37 David Henderson, *The Evolution of Psychiatry in Scotland* (Edinburgh: E. and S. Livingstone, 1964); Sharlene Walbaum, "The Invisible Woman: Susan Carnegie and Montrose Lunatic Asylum," *History of Psychiatry* 30, no. 4 (2019): 409-23.

38 Kenneth Keddie, "From "What it Was" towards "What it Ought to Be"—The Montrose Bicentenary," *Bulletin of the Royal College of Psychiatrists* 6 (1982): 98-102.

39 Andrew Scull, Charlotte Mackenzie, and Nicholas Hervey, "The Alienist as Propagandist: W A F Browne (1805-1885)," in *Masters of Bedlam*, ed. Andrew Scull, Charlotte Mackenzie and Nicolas Hervey (New Jersey: Princeton University Press, 2014), 84-122.

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>).

### 1.2.3 Records examined<sup>40</sup>

#### 1.2.3.1 Case Notes

Case notes have been relatively neglected as a resource by British scholars.<sup>41</sup> 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.<sup>42</sup> 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.”<sup>43</sup> 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.<sup>44</sup>

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

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40 A complete list of sources used in this work is to be found in the Bibliography.

41 Andrews, “Case Notes, Case Histories,” 255-81.

42 Gayle Davis, “The Most Deadly Disease of Asylumdom: General Paralysis of the Insane and Scottish Psychiatry, c. 1840-1940,” *Journal of the Royal College of Physicians of Edinburgh* 42 (2012): 266-73.

43 Volker Hess and Andrew Mendelsohn, “Case and Series: Medical Knowledge and Paper Technology, 1600–1900,” *History of Science* 48 (2010): 287.

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.<sup>45</sup> 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."<sup>46</sup> 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.<sup>47</sup> 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

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45 James Gibson, "A Critical Review of the Concept of Set in Contemporary Experimental Psychology," *Psychological Bulletin* 38 (1941): 781.

46 Henry Maudsley, *The Pathology of Mind* (New York: D. Appleton and Company, 1880), 114.

47 Janet Saunders, "Quarantining the Weak-minded: Psychiatric Definitions of Degeneracy and the late-Victorian Asylum," in *The Anatomy of Madness. Essays in the History of Psychiatry*, ed. William F. Bynum, Roy Porter and Michael Shepherd, vol. 3 *The Asylum and its Psychiatry* (London, Tavistock Press, 1988), 273-96.

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.”<sup>48</sup>

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

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<sup>48</sup> Sally Swartz, "Asylum Case Records: Fact and Fiction," *Rethinking History* 22 (2018): 289.

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.<sup>49</sup> However, in these two asylums only bare demographic detail and a telegraphic 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

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<sup>49</sup> Andrews, "Case Notes, Case Histories," 255-81.

stained with blood or damaged by preservatives, making them what Wallis calls “unusually resonant objects.”<sup>50</sup>

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.<sup>51</sup> 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.<sup>52</sup> 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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<sup>50</sup> Wallis, “Post-mortem Records,” 57.

<sup>51</sup> Hess and Mendelsohn, “Case and Series,” 287.

<sup>52</sup> Lisa Gitelman, *Paper Knowledge: Toward a Media History of Documents* (Durham, NC: Duke University Press, 2014).



Name	No. 94 K***** K**** (PM 212)
Occupation	Governess
PM performed	Lawson
Admission Register	Single, Farringdon. Transferred from Bethlem Hospital
Cause	Heredity and climacteric
Bodily condition	2 <sup>nd</sup> Class, thin and feeble
Duration of attack	14 months
Attack	First
Date of death	18 <sup>th</sup> Aug 1905
Date of admission	5 <sup>th</sup> Jan 1898
Age at death	53
Date of PM, PM delay	19th Aug 1905, 24 hours
Cause of death	Heart disease
Mental condition	Acute then Chronic mania
External appearance	Somewhat emaciated with no injuries etc.
Head	Brain soft and gyri are wasted. Sulci gaping. Grey matter wasted and white matter oedematous. Atheroma of basal arteries.
Heart/Thorax	Pericardium thickened. Myocardium soft and flabby. Aortic valve and aorta calcareous. Lungs congested at bases. Healed TB foci.
Abdomen	NAD
	Weight (oz)
Heart	6
R lung	18
L lung	20
Liver	26
Cerebrum	47

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

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

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.”<sup>53</sup> 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.”<sup>54</sup>

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<sup>53</sup> Graham Mooney, “Diagnostic Spaces: Workhouse, Hospital, and Home in Mid-Victorian London,” *Social Science History*, 33 (2009): 369.

<sup>54</sup> 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.<sup>55</sup>



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.<sup>56</sup> 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

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<sup>55</sup> Wheeler, *Fair Mile Hospital*, 28.

<sup>56</sup> 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.<sup>57</sup> 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.<sup>58</sup>

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.<sup>59</sup> 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

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<sup>57</sup> John Crammer, "English Asylums and English Doctors: Where Scull is Wrong," *History of Psychiatry* 5, no. 17 (1994): 103-15.

<sup>58</sup> Wheeler, *Fair Mile Hospital*, 28.

<sup>59</sup> Tyte, "Berkshire Old and New: Fair Mile Hospital," 38.

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.<sup>60</sup>

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

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<sup>60</sup> 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.<sup>61</sup> 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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<sup>61</sup> In late 2021, the Wellcome funded St George's Hospital, London, Post Mortem archive project was launched (<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 focussing 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.<sup>62</sup> One of the objectives of post-mortem practice was to throw

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<sup>62</sup> Nicholas Hervey, “A Slavish Bowing Down: The Lunacy Commission and the Psychiatric Profession 1845–60,” in *The Anatomy of Madness: Essays in the History of Psychiatry*, 3 vols, 2 (1985): 98-131; Jonathan Andrews, “They’re in the

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 Nineteenth-century Scotland, Occasional Publications, No. 8 (London: Wellcome Trust, 1998).*

## 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.<sup>63</sup> 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?”<sup>64</sup>

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

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<sup>63</sup> John Eyler, *Victorian Social Medicine: The Ideas and Methods of William Farr* (Baltimore: John Hopkins University Press, 1979).

<sup>64</sup> Wright, Jacklin and Themeles, “Dying to Get Out of the Asylum,” 599-600.

complete data set for scholars to examine, albeit one containing only limited demographic details.<sup>65</sup> 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.<sup>66</sup> 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.<sup>67</sup>

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.<sup>68</sup> 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.

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<sup>65</sup> Smith, "Welcome Release," 117-28.

<sup>66</sup> Melling and Forsythe, *The Politics of Madness*, 178-203, 255; Smith, "Welcome Release," 121; Smith, "Visitation by God," 104-16; Andrews, "Death and the Dead-house," 6-26; Anna Shepherd, *Institutionalizing the Insane in Nineteenth-century England* (London: Routledge, 2015), 109.

<sup>67</sup> Bill Luckin, "Death and Survival in the City, Approaches to the History of Disease," *Urban History Yearbook* (1980): 53-61; Anne Hardy, "'Death Is the Cure of All Diseases': Using the General Register Office Cause of Death Statistics for 1837-1920," *Social History of Medicine* 7 (1994): 472-92.

<sup>68</sup> Wright, Jacklin and Themeles, "Dying to Get Out of the Asylum," 599-600; Smith, "Welcome Release," 117-28.

### 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.<sup>69</sup> 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.<sup>70</sup> 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.<sup>71</sup> 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.<sup>72</sup> 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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<sup>69</sup> Anne Digby, "The Changing Profile of a Nineteenth-century Asylum: The York Retreat," *Psychological Medicine* 14 (1984): 739-48; William Parry-Jones, *The Trade in Lunacy: A Study of Private Madhouses in England in the Eighteenth and Nineteenth Centuries* (London: Routledge, 2013), 227-8.

<sup>70</sup> Smith, "Welcome Release," 122; Andrew Scull, *The Most Solitary of Afflictions: Madness and Society in Britain, 1700-1900* (Yale: Yale University Press, 1993), 96-103, 340.

<sup>71</sup> Digby, "The Changing Profile of a Nineteenth-century Asylum," 739-48.

<sup>72</sup> Peter McCandless, "Liberty and Lunacy: The Victorians and Wrongful Confinement," *Journal of Social History* 11 (1978): 366-86; David Hirst, "'A Ticklish Sort of Affair': Charles Mott, Haydock Lodge and the Economics of Asyludom," *History of Psychiatry* 16, no. 3 (2005): 311-32.

was promoted to the post of Chief Statistician in 1838.<sup>73</sup> 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, *inter alia*, to the requirement for asylum Superintendents to publish annual reports which included figures on mortality.<sup>74</sup> 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.<sup>75</sup> 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.<sup>76</sup> 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.<sup>77</sup> 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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<sup>73</sup> Tessa Richards, "Farr Sighted," *British Medical Journal* 6379 (1983): 1736.

<sup>74</sup> David Mellett, "Bureaucracy and Mental Illness: The Commissioners in Lunacy 1845–90," *Medical History* 25 (1981): 221-50.

<sup>75</sup> William Farr, "Report upon the Mortality of Lunatics," *Journal of the Statistical Society of London* 4 (1841): 17-33.

<sup>76</sup> Ruth Hodgkinson, "Provision for Pauper Lunatics 1834–1871," *Medical History* 10 (1966): 138-54; Luckin, "Death and Survival," 53-61; Jonathan Reinartz and Alistair Ritch, "Exploring Medical Care in the Nineteenth Century Provincial Workshop; a View from Birmingham," in *Medicine and the Workhouse*, ed. Jonathan Reinartz and Leonard Schwarz (Rochester, New York: University of Rochester Press, 2013), 140-64.

<sup>77</sup> Shepherd, *Institutionalizing the Insane*, 140; David Wright, "The Discharge of Pauper Lunatics from County Asylums in Mid-Victorian England: The Case of Buckinghamshire," in *Insanity, Institutions and Society 1800-1914*, ed. Joseph Melling and William Forsythe (London: Routledge, 1999), 100-3.

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.<sup>78</sup> 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.<sup>79</sup> 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.<sup>80</sup>

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

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<sup>78</sup> Noel Humphrey, “Statistics of Insanity in England, with Special Reference to its Alleged Increasing Prevalence,” *Journal of the Royal Statistical Society* 53 (1890): 201-52.

<sup>79</sup> Noel Humphreys, “The Alleged Increase of Insanity,” *Journal of the Royal Statistical Society* 70 (1907): 203-41.

<sup>80</sup> 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.”<sup>81</sup> 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.<sup>82</sup> 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.”<sup>83</sup> In 1894, Chapman further showed that the mortality of males aged between 40 and 50 was six times greater than the general population.<sup>84</sup> 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.<sup>85</sup> 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

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<sup>81</sup> John Millar, *A Plea in Favour of the Insane Poor to the Right Honourable the Earl of Shaftesbury*. LSE Pamphlets (London: Henry Renshaw, 1859), 8.

<sup>82</sup> Harriet C. G. Sturdy, “Boarding-out the insane, 1857-1913: a study of the Scottish system” (PhD diss., University of Glasgow, 1996).

<sup>83</sup> Algernon Chapman, “Note on the Comparative Mortality of Different Classes of Patients in Asylums,” *Journal of Mental Science* 23 (1877): 57.

<sup>84</sup> Algernon Chapman, “Ages and Death-rates of Lunatics in the District where ‘Accumulation’ of Lunatics is most Advanced,” *Journal of Mental Science*, 40 (1894): 385-93.

<sup>85</sup> Arthur Mitchell, “Contribution to the Study of the Death-rate of Persons in Asylums,” *Journal of Mental Science* 25 (1879): 1-4.



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.<sup>86</sup> 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.<sup>87</sup> 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.<sup>88</sup> 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."<sup>89</sup> 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.<sup>90</sup> Eyler gives a detailed account of the arguments that raged across the nineteenth century on this topic, pointing out that the arguments

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<sup>86</sup> Robert Jones, "Prognosis in Mental Diseases," *British Medical Journal* 1578 (1905): 83.

<sup>87</sup> Edward Renvoize and Allan Beveridge, "Mental Illness and the Late Victorians: A Study of Patients Admitted to Three Asylums in York, 1880-1884," *Psychological Medicine* 19 (1989): 19-28; Asa Briggs, *A Social History of England*, (London: Weidenfeld and Nicolson, 1983); Richard Adair, Joseph Melling and Bill Forsythe, "Migration, Family Structure and Pauper Lunacy in Victorian England: Admissions to the Devon County Pauper Lunatic Asylum, 1845-1900," *Continuity and Change* 12 (1997): 373-401.

<sup>88</sup> Melling and Forsythe, *The Politics of Madness*, 181; Smith, "Welcome Release," 126; Smith "'Visitation by God'," 110-113.

<sup>89</sup> John Thurnam, *Observations and Essays on the Statistics of Insanity* (London: Simpkins, Marshall & Co, 1845), 137.

<sup>90</sup> Smith, "Welcome Release," 122.

of both proponents and detractors of this metric had both strengths and weaknesses.<sup>91</sup> As 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.”<sup>92</sup> 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.<sup>93</sup> 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.<sup>94</sup>

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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<sup>91</sup> John Eyler, “Mortality Statistics and Victorian Health Policy: Program and Criticism,” *Bulletin of the History of Medicine* 50 (1976): 335-55.

<sup>92</sup> Smith, “‘Visitation by God’,” 111.

<sup>93</sup> Shepherd and Wright, “Madness, Suicide and the Victorian Asylum,” 178.

<sup>94</sup> Sarah York, “Alienists, Attendants and the Containment of Suicide in Public Lunatic Asylums, 1845–1890,” *Social History of Medicine* 25 (2011): 324.

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.<sup>95</sup> 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."<sup>96</sup> 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."<sup>97</sup> 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."<sup>98</sup> 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.<sup>99</sup> The emergence

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<sup>95</sup> Peter Bartlett, "The Poor Law of Lunacy: The Administration of Pauper Lunatics in Mid-nineteenth Century England, with Special Emphasis on Leicestershire and Rutland" (PhD diss., University of London, 1993), 120; Kathleen Jones, *Mental Health and Social Policy, 1845-1959* (London: Routledge, 2013).

<sup>96</sup> Smith, "'Visitation by God'," 111.

<sup>97</sup> Laurence Ray, "Models of Madness in Victorian Asylum Practice," *European Journal of Sociology* 22 (1981): 251.

<sup>98</sup> Smith, "Welcome Release," 122.

<sup>99</sup> 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).<sup>100</sup> 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.<sup>101</sup> 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.<sup>102</sup> 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.<sup>103</sup> 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

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<sup>100</sup> Hervey, "A Slavish Bowing Down," 98-131; Andrews, " 'They're in the Trade'."

<sup>101</sup> Marlene A. Arieno, *Victorian Lunatics: A Social Epidemiology of Mental Illness in Mid-Nineteenth-Century England* (Selinsgrove, PA: Susquehanna University Press, 1989), 101.

<sup>102</sup> Smith, "Behind Closed Doors," 301-27.

<sup>103</sup> Joseph Melling and Bill Forsythe ed., *Insanity, Institutions, and Society, 1800-1914: A Social History of Madness in Comparative Perspective* (London: Routledge, 1999).

rose further to 29.3 by 1890. This mirrored, but much outstripped, the rise in the population.<sup>104</sup> 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.<sup>105</sup> 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.<sup>106</sup> 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.<sup>107</sup> Arieno found that about 60% of patients were from the "labouring group" and about 35% from the "trades" group.<sup>108</sup> 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.<sup>109</sup> 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

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<sup>104</sup> John Walton, "Lunacy in the Industrial Revolution: A Study of Asylum Admissions in Lancashire, 1848-50," *Journal of Social History* 13 (1979): 1-22.

<sup>105</sup> Peter Bartlett and David Wright, *Outside the Walls of the Asylum: The History of Care in the Community 1750-2000* (London: A. & C. Black, 1999).

<sup>106</sup> Walton, "Lunacy in the Industrial Revolution," 1.

<sup>107</sup> Paul Tobia, "The Patients of the Bristol Lunatic Asylum in the Nineteenth Century" (PhD diss., University of the West of England, 2017), 71-83.

<sup>108</sup> Arieno, *Victorian Lunatics*, 80.

<sup>109</sup> David Wright, "Getting out of the Asylum: Understanding the Confinement of the Insane in the Nineteenth Century," *Social History of Medicine* 10 (1997): 137-55.

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.<sup>110</sup> 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.<sup>111</sup> 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.<sup>112</sup> 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.<sup>113</sup> 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.<sup>114</sup> 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.<sup>115</sup> 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.

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<sup>110</sup> Edward Myers, "Workhouse or Asylum: The Nineteenth Century Battle for the Care of the Pauper Insane," *Psychiatric Bulletin* 22 (1998): 575-7.

<sup>111</sup> Hodgkinson, "Provision for Pauper Lunatics," 146.

<sup>112</sup> Peter Bartlett, "The Asylum, the Workhouse, and the Voice of the Insane Poor in Nineteenth Century England," *International Journal of Law and Psychiatry* 21 (1998): 421-32.

<sup>113</sup> Robert Ellis, "The Asylum, the Poor Law, and a Reassessment of the Four-shilling Grant: Admissions to the County Asylums of Yorkshire in the Nineteenth Century," *Social History of Medicine* 19, no. 1 (2006): 55-71.

<sup>114</sup> Myers, "Workhouse or Asylum," 576.

<sup>115</sup> Peter Carpenter, "The Pauper Insane of Leicester in 1844," *History of Psychiatry* 8, no. 32 (1997): 517-37.

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.<sup>116</sup> 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.<sup>117</sup> Scull initially argued that asylums were “convenient places for inconvenient people.”<sup>118</sup> 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.”<sup>119</sup> 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.”<sup>120</sup>

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.<sup>121</sup> Wright agrees that economic pressure and the shift towards the nuclear family with industrialization led to an eroding of wider

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<sup>116</sup> Andrew Scull, “Psychiatry and Social Control in the Nineteenth and Twentieth Centuries,” *History of Psychiatry* 2, no. 6 (1991): 149-69.

<sup>117</sup> Wright “Getting out of the Asylum,” 154; Bill Forsythe, Joseph Melling and Richard Adair, “The New Poor Law and the County Pauper Lunatic Asylum — The Devon Experience 1834–1884,” *Social History of Medicine* 9, no. 3 (1996): 335-55.

<sup>118</sup> Scull, *The Most Solitary of Afflictions*, 135, 246.

<sup>119</sup> Melling and Forsythe ed., *Insanity, Institutions and Society*; John K. Walton, “Casting Out and Bringing Back in Victorian England: Pauper Lunatics, 1840–70,” in *The Anatomy of Madness. Essays in the History of Psychiatry*, ed. William F. Bynum, Roy Porter and Michael Shepherd, Vol. 2 Institutions and Society (London, Tavistock Press, 1985), 140–41; Walton, “Lunacy in the Industrial Revolution,” 3-18.

<sup>120</sup> Allan Beveridge, “Madness in Victorian Edinburgh: A Study of Patients Admitted to the Royal Edinburgh Asylum under Thomas Clouston, 1873-1908 Part II,” *History of Psychiatry* 6, no. 21 (1995): 152.

<sup>121</sup> Andrew Scull, *Museums of Madness: The Social Organization of Insanity in Nineteenth-century England* (New York: St. Martin's Press, 1979), 240.

ties of kinship and community and to increased admissions as the family's "pragmatic response."<sup>122</sup> This chimes in with the work of Melling who stresses the impact of factors like urbanisation, industrialisation and migration in increasing asylum admissions.<sup>123</sup> 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.<sup>124</sup> 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."<sup>125</sup> 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.<sup>126</sup> 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."<sup>127</sup> 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.<sup>128</sup> 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

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<sup>122</sup> Wright, "Getting out of the Asylum," 153.

<sup>123</sup> Melling and Forsythe, *Insanity, Institutions and Society*, 10.

<sup>124</sup> Michael Anderson and John Anderson, *Family Structure in 19th Century Lancashire* (Cambridge: Cambridge University Press, 1971), 2-4.

<sup>125</sup> Tobia, "The Patients of the Bristol Lunatic Asylum," 108.

<sup>126</sup> Smith, "Family, Community and the Victorian Asylum: A Case Study of the Northampton General Lunatic Asylum and its Pauper Lunatics," *Family and Community History* 9 (2006): 109-24; Carpenter, "The Pauper Insane of Leicester," 537; Renvoize and Beveridge, "Mental Illness and the Late Victorians," 19-28.

<sup>127</sup> Smith, "Family, Community and the Victorian Asylum," 122.

<sup>128</sup> Catherine Cox and Hilary Marland, "'A Burden on the County': Madness, Institutions of Confinement and the Irish Patient in Victorian Lancashire," *Social History of Medicine* 28, no. 2 (2015): 263-87.



authorities, in some cases because of shame or guilt.<sup>129</sup> 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.<sup>130</sup> 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.<sup>131</sup> 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."<sup>132</sup> 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."<sup>133</sup> Shepherd emphasises that the therapeutic environment of work, pastimes and entertainment continued in pauper asylums even as numbers grew.<sup>134</sup> 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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<sup>129</sup> Walton, "Lunacy in the Industrial Revolution," 15.

<sup>130</sup> Adair, Melling and Forsythe, "Migration, Family Structure and Pauper Lunacy," 389.

<sup>131</sup> Edward Hare, "Was Insanity on the Increase?" *British Journal of Psychiatry* 142 (1983): 439-55.

<sup>132</sup> Beveridge, "Madness in Victorian Edinburgh," 150; Andrew Scull, "Was Insanity Increasing? A Response to Edward Hare," *British Journal of Psychiatry* 144 (1984): 433.

<sup>133</sup> Bartlett, "The Asylum, the Workhouse, and the Voice of the Insane Poor," 429.

<sup>134</sup> Shepherd, *Institutionalizing the Insane*, 127-32.

findings were reported in studies of Ticehurst, Denbigh, Fife and Edinburgh asylums.<sup>135</sup> However, the perils of retrospective diagnoses are many, based as they are on the assumption that the nature of insanity is unchanging.<sup>136</sup> 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.<sup>137</sup> 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.<sup>138</sup> 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.<sup>139</sup> 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.<sup>140</sup> Sturdy and Parry-Jones point to the grant's effects in reducing the numbers of those boarded with their relatives

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<sup>135</sup> Renvoize and Beveridge, "Mental illness and the Late Victorians," 19-28; Trevor Turner, "A Diagnostic Analysis of the Casebooks of Ticehurst House Asylum, 1845–1890," *Psychological Medicine Monograph*, Supplement 21 (1992): 1-70; David Healy et al., "The Incidence of Admissions for Schizophrenia and Related Psychoses in Two Cohorts: 1875–1924 and 1994–2010," *British Medical Journal Open* 2 (2012): 1-10; Gillian Doody, Allan Beveridge, and Eve Johnstone, "Poor and Mad: A Study of Patients Admitted to the Fife and Kinross District Asylum between 1874 and 1899," *Psychological Medicine* 26 (1996): 887-97; Beveridge, "Madness in Victorian Edinburgh," 152.

<sup>136</sup> Jon Arrizabalaga, "Problematizing Retrospective Diagnosis in the History of Disease," *Asclepio* 54 (2002): 51-70.

<sup>137</sup> Turner, "A Diagnostic Analysis," 1-10; William F. Bynum, Roy Porter and Michael Shepherd ed., *The Anatomy of Madness: Essays in the History of Psychiatry* (New York and London: Tavistock Publications, 1985), 3 vols.

<sup>138</sup> See especially, David Wright, "The Certification of Insanity in Nineteenth-century England and Wales," *History of Psychiatry* 9, no. 35 (1998), 267-90

<sup>139</sup> John Thomas Arlidge, *On the State of Lunacy and the Legal Provision for the Insane: With Observations on the Construction and Organization of Asylums* (London: John Churchill, 1859); Jonathan Andrews, "The Rise of the Asylum in Britain," in *Medicine Transformed: Health, Disease and Society in Europe 1800-1930* ed. Deborah Brunton (Manchester: The Open University Press, 2004), 293-330.

<sup>140</sup> Andrew Scull, *The Most Solitary of Afflictions*, 370.

and conclude that it had a negative impact on the curative nature of asylums.<sup>141</sup> 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.<sup>142</sup> 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.<sup>143</sup> 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.<sup>144</sup> 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.<sup>145</sup> 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."<sup>146</sup> 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

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<sup>141</sup> Harriet Sturdy and William Parry-Jones, "Boarding-out Insane Patients: The Significance of the Scottish System 1857–1913," in *Outside the Walls of the Asylum*, ed. Peter Bartlett and David Wright (London: Athlone, 1999), 86–114.

<sup>142</sup> Allan Beveridge, "Madness in Victorian Edinburgh," 152.

<sup>143</sup> Graeme Yorston and Camilla Haw, "Old and Mad in Victorian Oxford: A Study of Patients aged 60 and over Admitted to the Warneford and Littlemore Asylums in the Nineteenth Century," *History of Psychiatry* 16, no. 4 (2005): 395–421; Emily Andrews, "Institutionalising Senile Dementia in 19th-century Britain," *Sociology of Health and Illness* 39 (2017): 244–57; Melling and Forsythe, *The Politics of Madness*, 186.

<sup>144</sup> Scull, *Museums of Madness*, 269–76; Melling and Forsythe, *The Politics of Madness*, 178–183.

<sup>145</sup> Edward Hare, "The Two Manias: A Study of the Evolution of the Modern Concept of Mania," *The British Journal of Psychiatry* 138 (1981): 439–55; Tobia, "The Patients of the Bristol Lunatic Asylum," 249.

<sup>146</sup> Roy Porter, "Foucault's Great Confinement," *History of the Human Sciences* 3 (1990): 47–54; Scull, *Museums of Madness*; Hodgkinson, "Provision for Pauper Lunatics," 150.

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.<sup>147</sup> 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.<sup>148</sup> 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.<sup>149</sup> 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.”<sup>150</sup>

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

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<sup>147</sup> Doody, Beveridge and Johnstone, “Poor and Mad,” 887-97.

<sup>148</sup> Melling and Forsythe, *The Politics of Madness*, 198.

<sup>149</sup> Catherine Cox, Hilary Marland and Sarah York, “Emaciated, Exhausted, and Excited: The Bodies and Minds of the Irish in Late Nineteenth-century Lancashire Asylums,” *Journal of Social History* 46 (2012): 501, 509, 517.

<sup>150</sup> 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.<sup>151</sup>

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.<sup>152</sup> 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.<sup>153</sup> 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.<sup>154</sup> The phenomenon of physically frail, disturbed patients being admitted and dying soon after lessened as the century progressed, as

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<sup>151</sup> Smith, “Welcome Release,” 121-25.

<sup>152</sup> 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.

<sup>153</sup> Melling and Forsythe, *The Politics of Madness*, 198.

<sup>154</sup> Adair, Melling and Forsythe, “Migration, Family Structure and Pauper Lunacy in Victorian England,” 373-401.

strikingly shown by Tobia in his detailed study of the Bristol Asylum.<sup>155</sup> 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.<sup>156</sup> Jacyna argues that this separation was artificial and did not reflect the true picture; clearly this was the case in relation to death.<sup>157</sup> 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.<sup>158</sup> Mellett and others argue that the Commissioners’ medicalization of insanity became particularly prominent 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.<sup>159</sup> 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.<sup>160</sup> Cramner proposes a number of behavioural reasons why morbidity and mortality were high in those committed to

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<sup>155</sup> Tobia, “The Patients of the Bristol Lunatic Asylum,” 128.

<sup>156</sup> Ray, “Models of Madness,” 229-64; Smith, “‘Visitation by God’,” 107.

<sup>157</sup> Stephen Jacyna, “Somatic Theories of Mind and the Interests of Medicine in Britain, 1850–1879,” *Medical History* 26 (1982): 233-58.

<sup>158</sup> Melling and Forsythe, *The Politics of Madness*, 6.

<sup>159</sup> Roy Porter and David Wright ed., *The Confinement of the Insane: International Perspectives, 1800–1965* (Cambridge: Cambridge University Press, 2003); Melling and Forsythe, *The Politics of Madness*, 202; Mellett, “Bureaucracy and Mental Illness,” 221-50; Adair, Melling and Forsythe, “Migration, Family Structure and Pauper Lunacy in Victorian England,” 373.

<sup>160</sup> Shepherd, *Institutionalizing the Insane*, 11.

the asylum and puts emphasis on how the insane “can be self-neglectful, unconcerned about chills or food.”<sup>161</sup> 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.<sup>162</sup> 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.<sup>163</sup> 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.<sup>164</sup> 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.”<sup>165</sup> 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.<sup>166</sup> However, doubts about this relationship continue to surface and most commentators agree these latter figures are unlikely

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<sup>161</sup> John Crammer, “English Asylums and English Doctors: Where Scull is Wrong,” *History of Psychiatry* 5, no. 17 (1994): 111.

<sup>162</sup> Andrew Sims, “Why the Excess Mortality from Psychiatric Illness?” *British Medical Journal* 6578 (1987): 986.

<sup>163</sup> Margaret Sorbie Thompson, “The Mad, the Bad, and the Sad: Psychiatric Care in the Royal Edinburgh Asylum, Morningside, 1813-1894,” *The Society for the Social History of Medicine Bulletin* 38 (1986): 29-33.

<sup>164</sup> Roy Porter, “The Drinking Man's Disease: The ‘Pre-History’ of Alcoholism in Georgian Britain,” *British Journal of Addiction* 80 (1985): 385-96.

<sup>165</sup> David Yellowlees, “Insanity and Intemperance,” *British Medical Journal* 666 (1873): 394.

<sup>166</sup> Emma Catherine Halliday, “Themes in Scottish Asylum Culture: The Hospitalisation of the Scottish Asylum 1880-1914” (PhD diss., University of Stirling, 2003), 144-45.

rates and numbers.<sup>167</sup> 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.<sup>168</sup> 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.<sup>169</sup> In 1876, Farr stated that the deaths from alcohol were rapidly decreasing, and that they were very few in number - less than 1,500 persons in England.<sup>170</sup> 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.<sup>171</sup> 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.<sup>172</sup> 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.<sup>173</sup> 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.<sup>174</sup> 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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<sup>167</sup> Shepherd, *Institutionalizing the Insane*, 124-5; Edward Hare, "The Two Manias," 83.

<sup>168</sup> Halliday, "Themes in Scottish Asylum Culture," 321.

<sup>169</sup> Peter McCandless, "'Curses of Civilization': Insanity and Drunkenness in Victorian Britain," *British Journal of Addiction* 79 (1984): 49-58; Virginia Berridge, "Dependence: Historical Concepts and Constructs," in *The Nature of Drug Dependence*, ed. Malcolm Lader and Griffith Edwards (Oxford: Oxford University Press, 1990), 1-18.

<sup>170</sup> Macdowel Cosgrave, "Alcohol and Longevity," *Dublin Journal of Medical Science* 90 (1890): 61-77.

<sup>171</sup> Frederick Mott, "A Discussion on Alcohol and Insanity," *British Medical Journal* 2439 (1907): 797-803.

<sup>172</sup> Shepherd and Wright, "Madness, Suicide and the Victorian Asylum," 177.

<sup>173</sup> Beveridge, "Madness in Victorian Edinburgh," 143.

<sup>174</sup> Melling and Forsythe, *The Politics of Madness*, 189-90.



attempts on their lives, and [were] likely to repeat them.”<sup>175</sup> 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.<sup>176</sup> 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.<sup>177</sup> 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.<sup>178</sup> As Ernst underlines, these infections particularly, though not exclusively, affect those already suffering from a fragile state of

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<sup>175</sup> Shepherd and Wright, “Madness, Suicide and the Victorian Asylum,” 177.

<sup>176</sup> Sturdy, “Boarding-out the Insane, 1857-1913”; Akihito Suzuki, *Madness at Home: The Psychiatrist, the Patient, and the Family in England, 1820-1860* (Berkeley, CA: University of California Press, 2006).

<sup>177</sup> Smith, “Welcome Release,” 122.

<sup>178</sup> *Ibid*, 117-28.

health on account of other illnesses, including insanity.<sup>179</sup> 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.<sup>180</sup> Renvoize and Beveridge provide evidence to support the contention that high patient: staff ratios and poor environmental conditions contributed to the increased death rate.<sup>181</sup> 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.<sup>182</sup> Associations between overcrowding, high use of restraint and high mortality rate were found in records of the Govan Asylum in the 1880s.<sup>183</sup> 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.<sup>184</sup>

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.<sup>185</sup> 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.<sup>186</sup> 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.<sup>187</sup> 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.<sup>188</sup> Alienists of the period had come to a similar viewpoint. George Jonathan Harder, the Medical

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<sup>179</sup> Ernst, “The Limits of Comparison,” 404-18.

<sup>180</sup> Wright, Jacklin and Themeles. “Dying to Get Out of the Asylum,” 606-08.

<sup>181</sup> Renvoize and Beveridge, “Mental Illness and the Late Victorians,” 19-28.

<sup>182</sup> Forsythe, Melling and Adair, “The New Poor Law and the County Pauper Lunatic Asylum,” 335-55

<sup>183</sup> Jonathan Andrews, “A Failure to Flourish? David Yellowlees and the Glasgow School of Psychiatry: Part 1,” *History of Psychiatry* 8, no. 30 (1997): 177-212.

<sup>184</sup> Wright, Jacklin and Themeles, “Dying to Get Out of the Asylum,” 599–600.

<sup>185</sup> Smith, “Behind Closed Doors,” 301-327.

<sup>186</sup> Melling and Forsythe, *The Politics of Madness*, 183.

<sup>187</sup> Richard Russell, “Mental Physicians and their Patients” (PhD diss., University of Sheffield, 1983), 312-7.

<sup>188</sup> Jennifer Wallis, “The Bones of the Insane,” *History of Psychiatry* 24, no. 2 (2013): 196-211.

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. Harder 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.<sup>189</sup> 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.<sup>190</sup> 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.<sup>191</sup>

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."<sup>192</sup> 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.<sup>193</sup> The average age at death of asylum patients rose across the period mirroring the

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<sup>189</sup> 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.

<sup>190</sup> Lee-Ann Monk, "Working in the Asylum: Attendants to the Insane," *Health and History* 11 (2009): 83-101.

<sup>191</sup> David Wright, "The Dregs of Society?" 5-19.

<sup>192</sup> Jonathan Andrews, "'Of the Termination of Insanity in Death', by James Cowles Prichard (1835)," *History of Psychiatry* 23, no. 1 (2012): 131.

<sup>193</sup> 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.<sup>194</sup> 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.<sup>195</sup> 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.<sup>196</sup> Smith suggests this imprecision was deliberate to excuse the medical profession from direct culpability in the deaths of their patients.<sup>197</sup> 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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<sup>194</sup> Wright, Jacklin, and Themeles, “Dying to Get Out of the Asylum,” 591-621; Jones, “Prognosis in Mental Diseases,” 1578-83; Melling and Forsythe, *The Politics of Madness*, 178-203.

<sup>195</sup> Smith, “Visitation by God,” 109.

<sup>196</sup> Smith, “Welcome Release,” 117-28; Steven Cherry, *Mental Health Care in Modern England: The Norfolk Lunatic Asylum/St. Andrew’s Hospital, 1810-1988* (Woodbridge: Boydell Press, 2003), 72; Samuel Shortt, *Victorian Lunacy: Richard M. Bucke and the Practice of Late Nineteenth-Century Psychiatry* (Cambridge: Cambridge University Press, 2011), 61.

<sup>197</sup> 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).

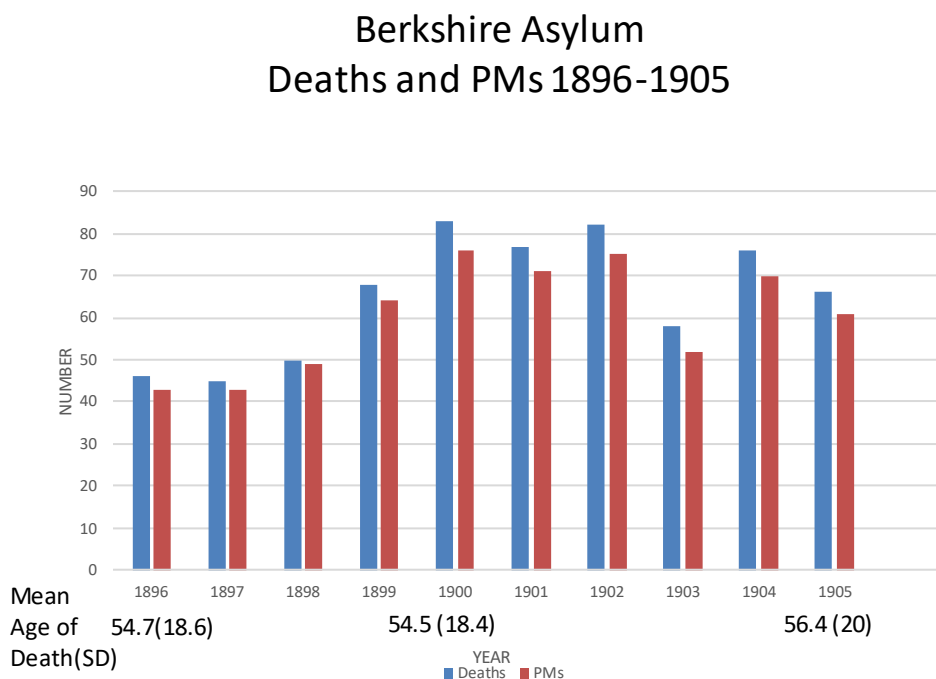


Figure 2-1 Deaths per annum across a ten-year period 1896-1905 at the Berkshire Asylum (blue columns) and numbers of post-mortems per annum (red columns). Figures are obtained from the Pathological Records.

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.<sup>198</sup> 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

<sup>198</sup> 45th Annual Report of the Commissioners in Lunacy (London: HMSO, 1891), 86-90; Hare, "The Two Manias," 83.

infection linked to water supply problems was seen at the Glenside Asylum Bristol during the same period.<sup>199</sup>

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.<sup>200</sup> 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.<sup>201</sup> 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

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<sup>199</sup> Donal Early, *The Lunatic Pauper Palace: Glenside Hospital Bristol, 1861-1994: Its Birth, Development and Demise* (Bristol: Friends of Glenside Hospital Museum, Bristol Archives, 2003), 12-13.

<sup>200</sup> Wheeler, *Fair Mile Hospital*, 21; Andrews, "Death and the Dead-house," 11.

<sup>201</sup> 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.<sup>202</sup> 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.<sup>203</sup> The influential alienist Clouston was one of the first and most powerful proponents of the clinical integrity of melancholia.<sup>204</sup>

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.<sup>205</sup> 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

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<sup>202</sup> German Berrios, "Melancholia and Depression during the 19th Century: A Conceptual History," *British Journal of Psychiatry* 153 (1988): 298-304; German Berrios, "Historical Aspects of Psychoses: 19th Century Issues," *British Medical Bulletin* 43 (1987): 484-98.

<sup>203</sup> Åsa Jansson, "Mood Disorders and the Brain: Depression, Melancholia, and the Historiography of Psychiatry," *Medical History* 55 (2011): 393-9.

<sup>204</sup> Thomas Clouston, *Clinical Lectures on Mental Diseases* (Philadelphia, PA: H. C. Lea's Son & Co, 1884).

<sup>205</sup> Melling and Forsythe, *The Politics of Madness*, 178-203.

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.<sup>206</sup> 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.<sup>207</sup> 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.<sup>208</sup> 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.<sup>209</sup> 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

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<sup>206</sup> Hugh Tunstall-Pedoe, "Crude Rates, Without Standardisation for Age, are Always Misleading," *British Medical Journal* 7156 (1998): 475.

<sup>207</sup> Farr, "Report upon the Mortality of Lunatics," 17-33; Richard Singer, "The First Mortality Follow-up Study: the 1841 Report of William Farr (Physician) on the Mortality of Lunatics," *Journal of Insurance Medicine* 33 (2001): 298-309.

<sup>208</sup> Charlotte Richardson, Alistair Robson, Lopindar Sood, Andrew Owen, "Standardised Mortality Rates among Residents at Hatton Asylum in the Pre-antipsychotic Era," personal communication.

<sup>209</sup> Davis, "The Most Deadly Disease of Asylums," 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.”<sup>210</sup> Davis observes that in some cases the diagnosis could not formally be given until “pathological proof” in the brain was uncovered at post-mortem.<sup>211</sup> However, by the time of the deaths in the current study, there was widespread knowledge of the clear and pathognomic 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.<sup>212</sup>

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.

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<sup>210</sup> Melling and Forsythe, *The Politics of Madness*, 131-2.

<sup>211</sup> Davis, “The Most Deadly Disease of Asylumdom,” 266-73.

<sup>212</sup> Charles Mercier, *A Text-book of Insanity* (London: S. Sonnenschein & Co. Ltd, 1902), 184.

### **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.

GROUP	Total number of deaths	CAUSE OF DEATH IN REGISTER OF DEATHS (BERKSHIRE ASYLUM)				
		Exhaustion n (%)	Brain Conditions (e.g., Senile decay/Cerebral softening/Cerebral atrophy) n (%)	Vascular disease (e.g., Morbis cordis/Heart disease/Heart failure, CVA) n (%)	Infection n (%)	Other n (%)
Mania	165	26 (16)	39 (24)	36 (22)	54 (33)	10 (6)
Melancholia	70	7 (10)	16 (21)	17 (26)	25 (36)	5 (7)
Dementia	169	1 (0)	88 (53)	21 (12)	52 (31)	7 (4)
Idiocy	72	3 (4)	12 (17)	7 (10)	34 (47)	16 (22)
Epilepsy	61	11 (18)	9 (15)	9 (15)	16 (26)	16 (26)
Total (% of all deaths)	537	48 (9)	164 (30)	90 (17)	181 (34)	54 (10)

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).<sup>213</sup> 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.<sup>214</sup> Smith gives details of

<sup>213</sup> This is even more marked when senile mania and melancholia cases are excluded (see Chapter 4).

<sup>214</sup> George Man Burrows, *Commentaries on the Causes, Forms, Symptoms, and Treatment, Moral and Medical, of Insanity* (London: T. and G. Underwood, 1828), 554; James Pritchard, *A Treatise on Insanity* (London: Gil Sherwood, 1835), 114; Luther Bell, “On a Form of Disease Resembling Some Advanced Stages of Mania and Fever, but so

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.<sup>215</sup> 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.<sup>216</sup> 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.”<sup>217</sup> The influential alienist W. A. F. Browne, for example, asserted in 1837 that the “prevailing opinion at

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Contradistinguished from any Ordinary Observed or Described Combination of Symptoms as to Render it Probable that it may be Overlooked and Hitherto Unrecorded Malady,” *American Journal of Insanity* 6 (1849): 97-127.

<sup>215</sup> William Farquharson, “On Melancholia: An Analysis of 730 Consecutive Cases,” *British Journal of Psychiatry* 40 (1894): 11-21.

<sup>216</sup> Harris *et al*, “The Morbidity and Mortality linked to Melancholia,” 3-14.

<sup>217</sup> Andrews, “Death and the Dead-house,” 9.

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.”<sup>218</sup> Barfoot, however, stresses that this view was not universally held.<sup>219</sup> 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.<sup>220</sup> The histographical trends in the labelling of deaths as vascular will be discussed in more detail in following chapters.

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<sup>218</sup> William Alexander Francis Browne, *What Asylums Were, Are, and Ought to be: Being the Substance of Five Lectures Delivered Before the Managers of the Montrose Royal Lunatic Asylum* (Edinburgh: Adam & Charles Black, 1837), 6.

<sup>219</sup> Michael Barfoot, “David Skae: Resident Asylum Physician; Scientific General Practitioner of Insanity,” *Medical History* 53 (2009): 469-88.

<sup>220</sup> Thomas Drapes, “Phthisis and Insanity: A Study Based Mainly on the Statistical Returns of Comparative Mortality in Ireland,” *Journal of Mental Science* 47, (1901): 667-78. For more on Drapes, see e.g. Pamela Cox, *Negotiating Insanity in the Southeast of Ireland, 1820–1900* (Manchester: Manchester University Press, 2012), 34, 45, 125, 200; Brendan D. Kelly, “Asylum Doctor Extraordinaire: Dr Thomas Drapes (1847–1919),” *Irish Journal of Medical Science* 184 (2015): 565-71.

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.<sup>221</sup> 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.<sup>222</sup> 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.”<sup>223</sup> 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.<sup>224</sup> 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.<sup>225</sup> 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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<sup>221</sup> Iain Hutchison and Dee Hoole, "Discharge, Disposal, and Death: Outcomes for Child Inmates of the Scottish National Institution, Larbert, and Stanley Hall, Wakefield, to 1913," *Journal of Family History* 45 (2020): 207-27; David Wright, "Family Strategies and the Institutional Confinement of 'Idiot' Children in Victorian England," *Journal of Family History* 23 (1998): 190-208.

<sup>222</sup> See e.g. Cherry, *Mental Health Care in Modern England*, 84-85.

<sup>223</sup> Wheeler, *Fair Mile Hospital*, 21.

<sup>224</sup> Smith, "Welcome Release," 120-21.

<sup>225</sup> Wright, Jacklin and Themeles, "Dying to Get Out of the Asylum," 599-600.

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.<sup>226</sup> 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.<sup>227</sup> 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

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<sup>226</sup> 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.

<sup>227</sup> Jones, “Prognosis in Mental Diseases,” 1578-83.

death being attributed to tuberculosis inappropriately.<sup>228</sup> 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.<sup>229</sup> However, data from the 75<sup>th</sup> 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.<sup>230</sup> 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."<sup>231</sup> 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.<sup>232</sup> 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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<sup>228</sup> See Chapter 3 for how this issue was dealt with in the current study.

<sup>229</sup> Drapes, "Phthisis and Insanity," 669.

<sup>230</sup> Hardy, "'Death Is the Cure of All Diseases'," 481; Crookshank, "The Frequency, Causation, Prevention, and Treatment of Phthisis Pulmonalis in Asylums," 657-83.

<sup>231</sup> Anon, "Tuberculosis in Lunatic Asylums," *British Medical Journal* 2182 (1902):1349.

<sup>232</sup> F. J. Stuart, "A Criticism on the Recent Report of the Tuberculosis Committee of the Medico-Psychological Association," *British Medical Journal* 2183 (1902): 1351.



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.<sup>233</sup> This recommendation became widely practiced, including at Fair Mile, and the norm by the early twentieth century.<sup>234</sup> 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.”<sup>235</sup> 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.<sup>236</sup> 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

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<sup>233</sup> Crookshank, “The Frequency, Causation, Prevention, and Treatment of Phthisis Pulmonalis in Asylums,” 657-683.

<sup>234</sup> Robert Jones, “Tuberculosis in the Insane,” *British Journal of Tuberculosis* 3 (1909): 8-19.

<sup>235</sup> Thomas Clouston, “Phthisical Insanity,” in *A Dictionary of Psychological Medicine* Vol. 2, ed Daniel Hack Tuke (London; Churchill, 1892), 940-1.

<sup>236</sup> German Berrios, “Memory and the Cognitive Paradigm during the Nineteenth Century: A Conceptual History,” in *Lectures on the History of Psychiatry: The Squibb Series*, ed. Robin Murray and Trevor H. Turner (London: Gaskell, 1990), 194–211.

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).<sup>237</sup> 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.<sup>238</sup> 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 emaciation brought on by starvation.

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<sup>237</sup> Yorston and Haw, "Old and Mad in Victorian Oxford," 416.

<sup>238</sup> 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.<sup>239</sup> Beveridge demonstrates that this low rate was despite the fact that many patients were described as suicidal in admission records.<sup>240</sup> 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.<sup>241</sup> 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.<sup>242</sup> 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

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<sup>239</sup> Shepherd and Wright, "Madness, Suicide and the Victorian Asylum," 175-96; Wright, Jacklin and Themeles, "Dying to Get Out of the Asylum," 591-621.

<sup>240</sup> Beveridge, "Madness in Victorian Edinburgh," 143.

<sup>241</sup> Shepherd and Wright, "Madness, Suicide and the Victorian Asylum," 194.

<sup>242</sup> 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.<sup>243</sup>

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.<sup>244</sup> 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.<sup>245</sup> 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.<sup>246</sup> 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

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<sup>243</sup> Smith, "Behind Closed Doors," 326.

<sup>244</sup> Richard Hunter and Ida McAlpine, *Psychiatry for the Poor: 1851 Colney Hatch Asylum--Friern Hospital 1973: A Medical and Social History* (London: Dawsons, 1974), 250-1.

<sup>245</sup> Renvoize and Beveridge, "Mental Illness and the Late Victorians," 19-28; R. Parker, A. Dutta, R. Barnes and T. Fleet, "County of Lancaster Asylum, Rainhill: 100 years Ago and Now," *History of Psychiatry* 4, no. 13 (1993): 95-105.

<sup>246</sup> Elizabeth Malcolm, "Swift's Hospital: A History of St Patrick's Hospital, Dublin, 1746-1889," *The Scriblerian and the Kit-Cats* 26 (1993): 77; Mark Finnane, *Insanity and the Insane in Post-Famine Ireland* (London: Croom Helm, 1981).

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.<sup>247</sup> 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.<sup>248</sup> For example, it was only then, as Luckin shows, that typhoid and typhus were reliably differentiated.<sup>249</sup> 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.”<sup>250</sup> 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

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<sup>247</sup> K. Codell Carter, “Causes of Disease and Causes of Death,” *Continuity and Change* 12 (1997): 189-98.

<sup>248</sup> John Woodward, “Medicine and the City: The Nineteenth-century Experience,” in *Urban Disease and Mortality in Nineteenth Century England*, ed. Robert Woods and John Woodward (London: Batsford Academic and Educational Ltd, 1984), 65-78; Hardy, “‘Death Is the Cure of All Diseases’,” 474-83.

<sup>249</sup> Luckin, “Death and Survival in the City,” 55.

<sup>250</sup> George Alter and Ann Carmichael, “Classifying the Dead: Toward a History of the Registration of Causes of Death,” *Journal of the History of Medicine and Allied Sciences* 54 (1999): 122.

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.<sup>251</sup>

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.<sup>252</sup> 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."<sup>253</sup> 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.<sup>254</sup> 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

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<sup>251</sup> Alice Reid, Eilidh Garrett, Chris Dibben and Lee Williamson, "'A Confession of Ignorance': Deaths from Old Age and Deciphering Cause-of-death Statistics in Scotland, 1855–1949," *The History of the Family* 20 (2015): 323.

<sup>252</sup> Melling and Forsythe, *The Politics of Madness*, 195.

<sup>253</sup> James Davey, "Lectures on Insanity, Delivered at the Bristol Medical School during the Summer Session of 1855," *Association Medical Journal* 3 (1855): 581.

<sup>254</sup> Bucknill, "Modes of Death Prevalent Among Insane," 338.

in asylum records particularly in the earlier parts of the century.<sup>255</sup> 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.<sup>256</sup> 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.<sup>257</sup> 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.<sup>258</sup> 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

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<sup>255</sup> Pamela Michael and David Hirst, "Recording the Many Faces of Death at the Denbigh Asylum, 1848–1938," *History of Psychiatry* 23, no. 1 (2012): 40-51; Smith, "Visitation by God," 104-16.

<sup>256</sup> Robert Woods and John Woodward, *Urban Disease and Mortality in Nineteenth-Century England* (London: BT Batsford Ltd, 1984).

<sup>257</sup> Thomas McKeown, *The Modern Rise of Population* (London: Edward Arnold, 1976).

<sup>258</sup> John Woodward, "Medicine and the City: The Nineteenth-Century Experience," in *Urban Disease and Mortality in Nineteenth Century England*, ed. Robert Woods and John Woodward (London: BT Batsford Ltd, 1984), 65-78.

period, when the British economy experienced sustained economic growth, mortality rates failed to improve at all.<sup>259</sup>

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).<sup>260</sup> 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.<sup>261</sup> 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.<sup>262</sup> 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

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<sup>259</sup> Simon Szreter, “Industrialization and Health,” *British Medical Bulletin* 69 (2004): 75-86.

<sup>260</sup> Paul Clayton and Judith Rowbotham, “How the Mid-Victorians Worked, Ate and Died,” *International Journal of Environmental Research and Public Health* 6 (2009): 1235-53.

<sup>261</sup> Clayton and Rowbotham, “How the Mid-Victorians Worked, Ate and Died,” 1246.

<sup>262</sup> Simon Szreter, “Economic Growth, Disruption, Deprivation, Disease, and Death: On the Importance of the Politics of Public Health for Development,” *Population and Development Review* 23 (1997): 693-728.



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.<sup>263</sup> 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.<sup>264</sup>

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 sewerage 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.<sup>265</sup> Another factor impinging on the reduced mortality over Victoria's reign is the reduction in what Anderson terms "crisis mortality."<sup>266</sup> 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

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<sup>263</sup> Melling and Forsythe, *The Politics of Madness*, 198-201.

<sup>264</sup> Arieno, *Victorian Lunatics*, 80; Tobia, "The Patients of the Bristol Lunatic Asylum," 71-83.

<sup>265</sup> Simon Szreter and Graham Mooney, "Urbanization, Mortality, and the Standard of Living Debate: New Estimates of the Expectation of Life at Birth in Nineteenth-century British Cities," *Economic History Review* (1998): 84-112.

<sup>266</sup> Michael Anderson, *Population Change in North-western Europe, 1750-1850* (London: Macmillan International Higher Education, 1988).

London asylums.<sup>267</sup> 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.<sup>268</sup> 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.<sup>269</sup> 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.”<sup>270</sup>

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

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<sup>267</sup> Wright, Jacklin and Themeles, “Dying to Get Out of the Asylum,” 602-3; Jones, “Prognosis in Mental Diseases,” 1578-83.

<sup>268</sup> Cutler, Deaton, Lleras-Muney, “The Determinants of Mortality,” 97-120.

<sup>269</sup> Edward Higgs, “Disease, Febrile Poisons, and Statistics: The Census as a Medical Survey, 1841–1911,” *Social History of Medicine* 4 (1991): 465-78.

<sup>270</sup> Beveridge, “Madness in Victorian Edinburgh,” 147.

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.<sup>271</sup> 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).<sup>272</sup> 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.<sup>273</sup> 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

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<sup>271</sup> Hurren, “‘Abnormalities and Deformities,’” 65-77

<sup>272</sup> Andrews, “Case Notes, Case Histories,” 255-81.

<sup>273</sup> Angela McCarthy, Catharine Coleborne, Maree O’Connor, and Elspeth Knewstubb, “Lives in the Asylum Record, 1864 to 1910: Utilising Large Data Collection for Histories of Psychiatry and Mental Health,” *Medical History* 61 (2017): 358-79; Bill Luckin, “Death and Survival in the City,” 53-61.

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.<sup>274</sup> 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.<sup>275</sup> 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

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<sup>274</sup> Hodgkinson, "Provision for Pauper Lunatics," 138-54.

<sup>275</sup> Susan Lanzoni, "The Asylum in Context: An Essay Review," *Journal of the History of Medicine and Allied Sciences* 60 (2005): 499-505.

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.<sup>276</sup>

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<sup>276</sup> McGovern, "The Myths of Social Control and Custodial Oppression," 3-23.

## 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.<sup>277</sup> 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.<sup>278</sup> 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.<sup>279</sup> 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.

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<sup>277</sup> See e.g., Andrews, "Introduction: Lunacy's Last Rites," 3-5; Andrews, "Death and the Dead-house," 6-26; Cullen, "Post-mortem in the Victorian Asylum," 280-96; Hurren, "'Abnormalities and Deformities,'" 65-77; idem, *Hidden Histories of the Dead: Disputed Bodies in Modern British Medical Research* (Cambridge: Cambridge University Press, 2021); Wallis, *Investigating the Body*; Jacyna and Casper, *The Neurological Patient in History*.

<sup>278</sup> See e.g., Engstrom, *Clinical Psychiatry in Imperial Germany*; Leslie Topp, "Complexity and Coherence: The Challenge of the Asylum Mortuary in Central Europe, 1898–1908," *Journal of the Society of Architectural Historians* 71 (2012): 8-41.

<sup>279</sup> Wallis, *Investigating the Body*, 16; Roger Cooter, "After Death/After-'Life': The Social History of Medicine in Post-postmodernity," *Social History of Medicine* 20 (2007): 447-8.

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.<sup>280</sup> 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.<sup>281</sup> 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.<sup>282</sup> 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.<sup>283</sup> 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.<sup>284</sup> 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

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<sup>280</sup> Guenter Risse, “Cause of Death as a Historical Problem,” *Continuity and Change* 12 (1997): 175-88.

<sup>281</sup> Anthony Gal, “In Search of the Origins of Modern Surgical Pathology,” *Advances in Anatomic Pathology* 8 (2001): 1-13.

<sup>282</sup> Stephen Jacyna, “The Localization of Disease” in *Medicine Transformed: Health, Disease and Society in Europe*, ed. Deborah Brunton (Manchester: Manchester University Press and The Open University, 2004), 1-27; Thomas Schlich, “The Emergence of Modern Surgery” in *ibid*, 61-95.

<sup>283</sup> Alvin Rodin, “Canada’s Foremost Pathologist of the Nineteenth Century--William Osler,” *Canadian Medical Association Journal* 107 (1972): 891.

<sup>284</sup> Steve Sturdy, “Scientific Method for Medical Practitioners: The Case Method of Teaching Pathology in early Twentieth-century Edinburgh,” *Bulletin of the History of Medicine* 81 (2007): 760-92.

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.<sup>285</sup> 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.<sup>286</sup>

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.”<sup>287</sup>

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

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<sup>285</sup> Mooney, Graham, Bill Luckin and Andrea Tanner. “Patient Pathways: Solving the Problem of Institutional Mortality in London during the Later Nineteenth Century,” *Social History of Medicine* 12 (1999): 227-69.

<sup>286</sup> Reid, Garrett, Dibben and Williamson, “‘A Confession of Ignorance’,” 320-44.

<sup>287</sup> Sturdy, “Scientific Method for Medical Practitioners,” 787.



nineteenth century brought objects and bodies—dead and alive—into increasingly public spaces for display.<sup>288</sup> 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.<sup>289</sup> 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.”<sup>290</sup> 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.”<sup>291</sup> 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.<sup>292</sup> 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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<sup>288</sup> Alberti, Samuel, *Morbid Curiosities: Medical Museums in Nineteenth-century Britain* (Oxford; Oxford University Press, 2011).

<sup>289</sup> Helen M. Dingwall, *A Famous and Flourishing Society. The History of the Royal College of Surgeons of Edinburgh, 1505–2005* (Edinburgh: Edinburgh University Press, 2005), 201–2.

<sup>290</sup> Stephen Jacyna, “The Laboratory and the Clinic: The Impact of Pathology on Surgical Diagnosis in the Glasgow Western Infirmary, 1875-1910,” *Bulletin of the History of Medicine* 62 (1988): 404.

<sup>291</sup> Andrews, “Death and the Dead-house,” 9.

<sup>292</sup> Jennifer Wallis, “The Bones of the Insane,” *History of Psychiatry* 24, no. 2 (2013): 196-211.

causes of death, but also to advance knowledge of the pathology and treatment of various forms of insanity.<sup>293</sup> 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.<sup>294</sup> 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.”<sup>295</sup> 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.<sup>296</sup> Bucknill, for example, was clear that the carrying out of a post-mortem did not improve accuracy on cause of death.<sup>297</sup> 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.”<sup>298</sup> 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.<sup>299</sup> 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

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<sup>293</sup> Hunter and McAlpine, *Psychiatry for the Poor*, 243-6.

<sup>294</sup> Hurren, “‘Abnormalities and Deformities’,” 65-77.

<sup>295</sup> R. H. B Wickham, “Post Mortem Examinations in Lunatic asylums,” *British Medical Journal* 884 (1877): 831.

<sup>296</sup> James Adam, “On Pathological Research in Asylums for the Insane,” *Journal of Mental Science* 30 (1884): 359.

<sup>297</sup> Bucknill, “Modes of Death Prevalent among Insane,” 336-42.

<sup>298</sup> Wickham, “Post Mortem Examinations,” 381.

<sup>299</sup> Andrews, “Death and the Dead-house,” 17.

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."<sup>300</sup>

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.<sup>301</sup> Strange shows that many relatives claimed bodies from institutions so as to avoid dissection, carry out mourning rituals and arrange decent burials.<sup>302</sup> 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.<sup>303</sup> 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

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<sup>300</sup> Hunter and McAlpine, *Psychiatry for the Poor*, 243-6.

<sup>301</sup> Hurren, "Abnormalities and Deformities," 65-77.

<sup>302</sup> Julie-Marie Strange, "Only a Pauper whom Nobody Owns: Reassessing the Pauper Grave c. 1880-1914," *Past & Present* 178 (2003): 148-75.

<sup>303</sup> Andrews, "Death and the Dead-house," 21.

staff and bereaved relatives.<sup>304</sup> Many asylums followed the blueprint for ideal mortuary facilities published by Burdett in 1891.<sup>305</sup> Mortuary laboratories and the appointment of pathologists became more common.<sup>306</sup> There was, in spatial, resource and personnel terms, in Andrews's words, an "increasing primacy of anatomico-pathological research."<sup>307</sup> 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.<sup>308</sup> 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."<sup>309</sup> 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."<sup>310</sup> 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

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<sup>304</sup> Philo, "Troubled Proximities," 91-103; Andrew Scull, "Moral Architecture: The Victorian Lunatic Asylum," in *Social Order/Mental Disorder: Anglo-American Psychiatry in Historical Perspective*, ed. Andrew Scull (Berkeley: University of California Press, 1989), 213-38.

<sup>305</sup> 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.

<sup>306</sup> Alfred Newth, "The Necessity for a Museum and Laboratory of Cerebral Pathology and Physiology," *Journal of Mental Science* 45 (1899): 321-5.

<sup>307</sup> Andrews, "Death and the Dead-house," 21.

<sup>308</sup> Wallis, *Investigating the Body*, 225-6.

<sup>309</sup> Andrew Scull, *The Most Solitary of Afflictions*, 263.

<sup>310</sup> 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.<sup>311</sup>

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.<sup>312</sup> To those who believed that “insanity had no pathology”, the counter-argument was that this was merely a consequence of “imperfect means of investigation.”<sup>313</sup> 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).<sup>314</sup> 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.<sup>315</sup> 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 to be a way of searching for “morphological deviation” to verify the

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<sup>311</sup> Ibid.

<sup>312</sup> Shortt, *Victorian Lunacy*, 140.

<sup>313</sup> Duncan Greenlees, “Observations with the Sphygmograph on Asylum Patients,” *Journal of Mental Science* 32 (1887): 472.

<sup>314</sup> Andreas Steck, “Milestones in the Development of Neurology and Psychiatry in Europe,” *Schweizer Archiv für Neurologie und Psychiatrie*, 161 (2010): 85-9; John Pearce, “Brain Disease Leading to Mental Illness: A Concept Initiated by the Discovery of General Paralysis of the Insane,” *European Neurology* 67 (2012): 272-78.

<sup>315</sup> Wallis, *Investigating the Body*, 206.

stigmata of degeneracy.<sup>316</sup> 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).<sup>317</sup> 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.<sup>318</sup> 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.<sup>319</sup> 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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<sup>316</sup> Edward Shorter, *A History of Psychiatry: From the Era of the Asylum to the Age of Prozac* (New York: Wiley, (1997), 69; Jacyna, “Somatic Theories of Mind,” 233-58; Shortt, *Victorian Lunacy*, 103.

<sup>317</sup> Andrews, “Death and the Dead-house,” 18.

<sup>318</sup> *Ibid*, 9; Elizabeth Hurren, “Abnormalities and Deformities,” 65-77; MacKinnon, *Bodies of Evidence*, 99; Smith “‘Visitation by God’,” 113.

<sup>319</sup> Personal communication from Professor A K Foulis, Professor of Pathology, University of Glasgow.

large and varied experiences,” observations which, in the main, chime in with the findings of this study.<sup>320</sup>

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.<sup>321</sup> 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.<sup>322</sup> 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.<sup>323</sup> 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.<sup>324</sup> 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.<sup>325</sup>

The widely held contemporary view that better regulation and monitoring were the ways to counteract high asylum mortality is described by several commentators.<sup>326</sup> 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

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<sup>320</sup> MacKinnon, *Bodies of Evidence*, 85.

<sup>321</sup> Burney, *Bodies of Evidence*.

<sup>322</sup> MacKinnon, *Bodies of Evidence*, 85.

<sup>323</sup> Cullen, “Post-mortem in the Victorian asylum,” 284.

<sup>324</sup> Clouston, “Tuberculosis and Insanity,” 38.

<sup>325</sup> Andrews, “Case Notes, Case Histories,” 267.

<sup>326</sup> Smith, “Welcome Release,” 117-28; Scull, *The Most Solitary of Afflictions*, 96-103, 340; Anne Digby, “The Changing Profile of a Nineteenth-century Asylum,” 739-48.

encouraging the practice of keeping detailed pathological records in asylums.<sup>327</sup> 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.<sup>328</sup> 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.<sup>329</sup> 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.<sup>330</sup>

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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<sup>327</sup> Cullen, "Post-mortem in the Victorian Asylum," 280-96.

<sup>328</sup> Monk, "Working in the Asylum," 83-101; Smith, "Behind Closed Doors," 301-27; Wright, "The Dregs of Society?" 5-19.

<sup>329</sup> A similar point is made in Cullen, "Post-mortem in the Victorian Asylum," 287.

<sup>330</sup> 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.”<sup>331</sup> This mortuary building is shown in Figure 3.2.

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<sup>331</sup> Ian Wheeler, personal communication.

BERKS, READING & NEWBURY LUNATIC ASYLUM.

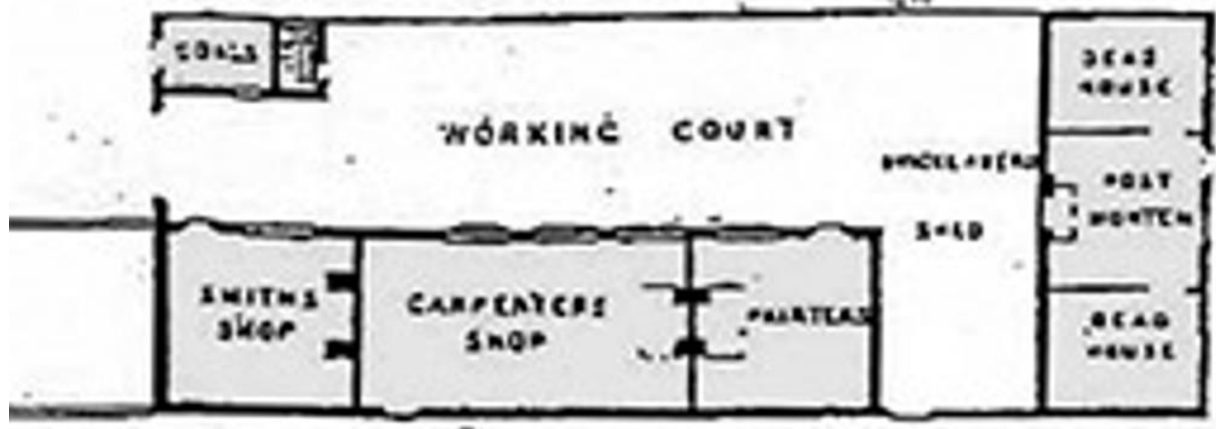
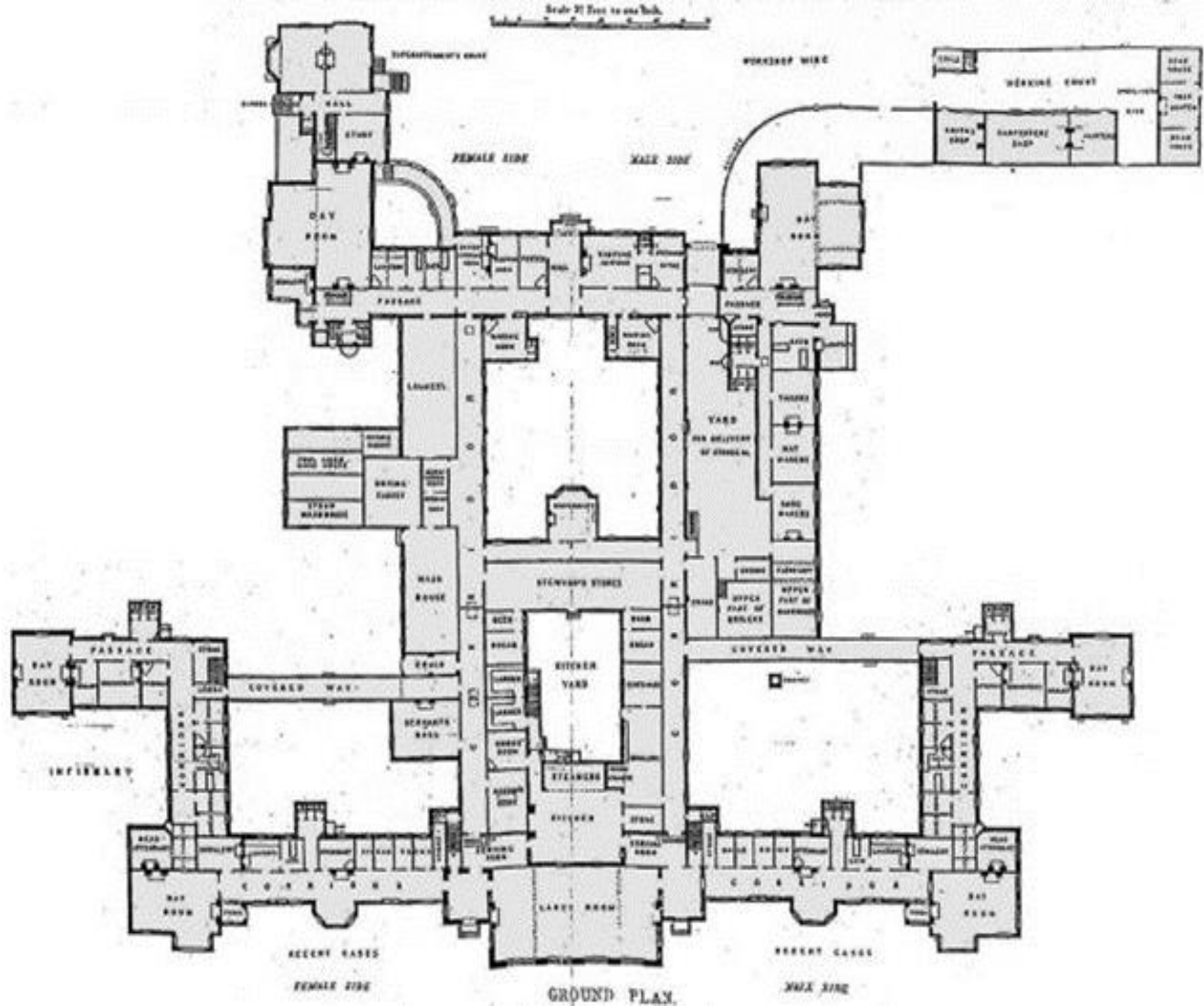


Figure 3-1 Above:-The 1870 plan of Berkshire Asylum (Berkshire Records Office, C/Cl/G1/25/2). The dead-house is the very top right building. Below:- Enlarged version of top right corner of plan showing the detail of the dead-house. (Photograph courtesy of Ian Wheeler).



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.<sup>332</sup> 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."<sup>333</sup>

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.<sup>334</sup> 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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<sup>332</sup> Andrews, "Death and the Dead-house," 6-26.

<sup>333</sup> 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.

<sup>334</sup> Andrews, "Death and the Dead-house," 21.



Figure 3-4 Photographs of the mortuary at Montrose Royal Asylum taken by the author in 2017. (Above - rear view, Below - front view).

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.<sup>335</sup> 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

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<sup>335</sup> Cullen, “Post-mortem in the Victorian Asylum,” 280-96.

was central and was what made doctors special.<sup>336</sup> 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.<sup>337</sup>

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.<sup>338</sup> According to Wheeler, he was highly thought of and was “highly intelligent, well informed and keen on research.”<sup>339</sup> 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.<sup>340</sup>

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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<sup>336</sup> Kenneth Nystrom, “Post-mortem Examinations and the Embodiment of Inequality in 19th century United States,” *International Journal of Paleopathology* 1 (2011): 164-72.

<sup>337</sup> Sturdy, “Scientific Method for Medical Practitioners,” 760-92.

<sup>338</sup> Buklijas, “The Laboratory and the Asylum,” 311-25.

<sup>338</sup> Cullen, “Post-mortem in the Victorian Asylum,” 281.

<sup>339</sup> Wheeler, *Fair Mile Hospital*, 21.

<sup>340</sup> Dr (Lennon-) Brown was a graduate of Edinburgh where pathology teaching was particularly prominent and highly regarded. Dr Brown had been much sought after to come to the Berkshire Asylum (so much so that he was promised a rise (from £130pa to £150pa) and a refurbished bedroom).

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.<sup>341</sup> 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.”)<sup>342</sup> 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.<sup>343</sup> 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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<sup>341</sup> Sylvia Barnard, *To Prove I'm not Forgotten: Living and Dying in a Victorian City* (Manchester; Manchester University Press, 1990).

<sup>342</sup> Wheeler, *Fair Mile Hospital*, 159.

<sup>343</sup> 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.

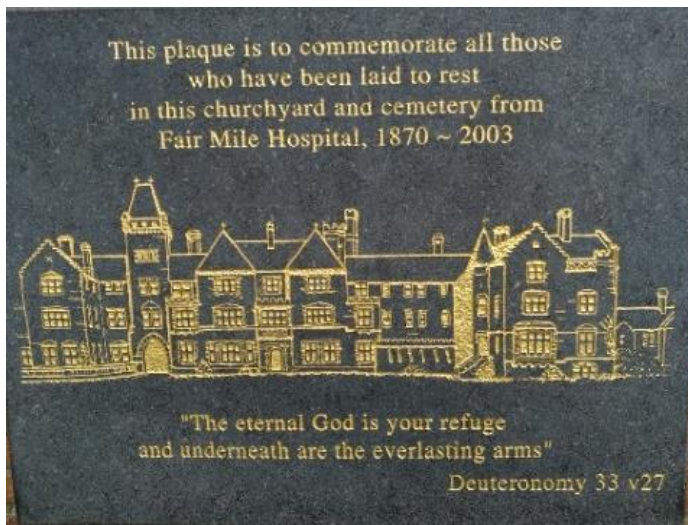


Figure 3-6 Plaque on wall bordering pauper graves at Cholsey Parish Church, photographed by author in 2017.

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 Sleepyhillock 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.<sup>344</sup> There was a hospital chaplain at Sunnyside but he was paid £70 per annum and was therefore likely to be part-time.<sup>345</sup> 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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<sup>344</sup> Personal communication from Linda Fraser, former custodian of Montrose Museum.

<sup>345</sup> 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.<sup>346</sup> 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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<sup>346</sup> Reid et al, “‘A Confession of Ignorance’,” 320-44.

POST-MORTEM NOTES	Number ...
Name and Number in Register of Patients .....	
Sex .... Age ..... Previous occupation .....	
Date of admission .....	
Date of death .....	
[ On Admission .....	
Form of Mental Disorder	
[ At death .....	
Certified cause of death .....	
Case book ..... Vol ..... Page .....	
Date and hour of Post-mortem Examination .....	
Condition of Body, and External Appearances, ] .....	
And whether or not Bedsores present ] .....	
Head .....	
Thorax (describe condition of Ribs) .....	
Abdomen .....	
Weight of Organs:- Brain ... Lungs (R) ... (L) ... Liver ... Kidneys (R) ... (L) ...	
Spleen ...	
Microscopic appearances, and any Special Notes .....	
Signed .....	

Figure 3-7 Schematic of a post-mortem record proforma from Fair Mile Asylum. An example of an original post-mortem report can be seen in Appendix A.

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.”<sup>347</sup> Andrews indicates that this concern might be particularly evident in families where religious beliefs emphasised resurrection.<sup>348</sup> Wallis observes that across many cultures the feeling that the brain is intimately linked with the self is commonplace.<sup>349</sup>

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

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<sup>347</sup> Cullen, “Post-mortem in the Victorian Asylum,” 285.

<sup>348</sup> Andrews, “Death and the Dead-house,” 16.

<sup>349</sup> 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.<sup>350</sup> 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.<sup>351</sup> 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.<sup>352</sup>

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.<sup>353</sup> 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.<sup>354</sup> 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.<sup>355</sup> 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

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<sup>350</sup> Cullen, “Post-mortem in the Victorian Asylum,” 284-86; Andrews, “Death and the Dead-house, 15; Wickham, “Post mortem Examinations,” 831.

<sup>351</sup> Andrews, “Death and the Dead-house,” 16.

<sup>352</sup> Phillip Fennell, *Treatment without Consent: Law, Psychiatry and the Treatment of Mentally Disordered People since 1845* (Abingdon: Routledge, 2002); Wallis, *Investigating the Body*, 81.

<sup>353</sup> Cullen, “Post-mortem in the Victorian Asylum,” 284-286; Elizabeth Hurren, “Remaking the Medico-legal Scene: A Social History of the late-Victorian Coroner in Oxford,” *Journal of the History of Medicine and Allied Sciences* 65 (2009): 207-52; Pam Fisher, “Getting away with Murder? The Suppression of Coroners’ Inquests in early Victorian England and Wales,” *Local Population Studies* 78 (2007): 47.

<sup>354</sup> Zuck, “Mr Troutbeck as the Surgeon’s Friend,” 260.

<sup>355</sup> 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.<sup>356</sup> 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.<sup>357</sup> 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.<sup>358</sup> 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.<sup>359</sup> An associate, John Batty Tuke, of Fife and Kinross Asylum, also began publishing tables of observed brain lesions as an

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<sup>356</sup> Wheeler, *Fair Mile Hospital*, 21.

<sup>357</sup> Nystrom, “Post-mortem Examinations,” 164-72.

<sup>358</sup> Andrews, “Death and the Dead-house,” *Asylums*,” 16.

<sup>359</sup> Howden, “An analysis of the post-mortem appearances,” 83-93.

appendix to his asylum's annual reports.<sup>360</sup> 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.<sup>361</sup> Howden published his "Pathological Index" in 1891.<sup>362</sup> 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.<sup>363</sup> However, several detailed manuals for post-mortems were available in the UK at this period.<sup>364</sup> 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.<sup>365</sup> 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).<sup>366</sup> 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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<sup>360</sup> John Batty Tuke, "A Pathological Classification of Mental Disease," *Journal of Mental Science* 16 1870: 195–210.

<sup>361</sup> W. G. Balfour, "Pathological Appearances observed in the Brains of the Insane," *Journal of Mental Science* 20 (1874): 49-60.

<sup>362</sup> James Howden, *Scheme for Pathological Index* (Glasgow: MacDougall: 1891).

<sup>363</sup> Medico-Psychological Association, *Report of a Committee appointed by the Medico-Psychological Association of Great Britain and Ireland, 1891*. Accessed May 12<sup>th</sup>, 2022, <https://wellcomecollection.org/works/frrvpk2e>

<sup>364</sup> Thomas Amos, *A Practical Guide for Making Post-mortem Examinations, and for the Study of Morbid Anatomy* (Philadelphia: Boericke & Tafel, 1873); Alfred Newth, *A Manual of Necroscopy, or, A Guide to the Performance of Post Mortem Examinations, with Notes on the Morbid Appearances, and Suggestions for Medico Legal Examinations: for the Use of Practitioners and Students* (London: Smith, Elmer, 1878).

<sup>365</sup> Alfred Newth, "The Importance of Uniformity in Microscopical Observations of Brain Structure," *Journal of Mental Science* 22 (1877): 554-9; William Lewis, *The Human Brain: Histological and Coarse Methods of Research: A Manual for Students and Asylum Medical Officers* (London: J. and A. Churchill, 1882).

<sup>366</sup> Buklijas, "The Laboratory and the Asylum," 311-25; Andrews, "Death and the Dead-house," 16.

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.



Asylum & number of cases (n)		Length of admission before death				
		Less than 1 week	1 week to less than 4 weeks	4 weeks to 1 year	1 to 5 years	More than 5 years
Sunnyside n=72	n	6	5	18	26	17
	%	8	7	25	36	24
Fair Mile n=100	n	9	10	19	36	24
	%	9	10	19	36	24
Combined n=172	n	15	15	37	62	41
	%	9	9	22	36	24

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.<sup>367</sup> 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).

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<sup>367</sup> Wallis, “The Bones of the Insane,” 196-211.

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."<sup>368</sup>

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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<sup>368</sup> Sturdy, "Scientific Method for Medical Practitioners," 776.

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

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."<sup>369</sup> 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.

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<sup>369</sup> MacKinnon, *Bodies of Evidence*, 80.

Asylum Group		CAUSE OF DEATH AFTER POST-MORTEM PERFORMED				
		Exhaustion	Cerebral (Senile decay/Cerebral softening/Cerebral atrophy)	Vascular disease (Morbus cordis/ heart disease/ heart failure/ apoplexy)	Infection	Other
Sunnyside Mania n= 45 Age= 43	n(%)	3 (7)	2 (4)	8 (18)	27 (60)	5 (11)
	Age	40	49	50	40 TB= 39	45
Sunnyside ME n= 27 Age= 40	n(%)	2 (7)	0 (0)	2 (7)	19 (71)	4 (15)
	Age	40	-	55	39 TB= 37	43
Fair Mile Mania n=71 Age= 42	n(%)	10 (14)	4 (6)	15 (21)	37 (52)	5 (7)
	Age	47	47	44	39 TB= 38	44
Fair Mile ME N= 29 Age= 45	n(%)	3 (10)	3 (10)	2 (7)	18 (62)	3 (10)
	Age	49	48	47	43 TB= 39	51
TOTAL Mania n= 116 Age= 42	n(%)	13 (11)	6 (5)	23 (20)	64 (55)	10 (9)
	Age	45	47	46	39	44
TOTAL ME n= 56 Age= 43	n(%)	5 (9)	3 (5)	<b>4 (7)</b>	37 (66)	7 (13)
	Age	45	48	51	41	46

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.<sup>370</sup> 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.”<sup>371</sup> The views of these authorities are supported by subsequent commentators.<sup>372</sup> 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.<sup>373</sup> 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.

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<sup>370</sup> Clouston, “Tuberculosis and Insanity,” 36-65.

<sup>371</sup> Frederick Mott, “Tuberculosis in London County Asylums,” *Archives of Neurology and Psychiatry* 4 (1909): 78.

<sup>372</sup> Drapes, “Phthisis and Insanity,” 667-78; Crookshank, “The Frequency, Causation, Prevention, and Treatment of Phthisis Pulmonalis in Asylums,” 657-83.

<sup>373</sup> Harris et al, “The Morbidity and Mortality Linked to Melancholia,” 3-14.

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 “lypemia” (as he dubbed what Berrios shows is essentially melancholia) in 1820 and found that about a third died, “often of tuberculosis.”<sup>374</sup> 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.<sup>375</sup>

The association of melancholia, tuberculosis and death was well recognised in the eighteenth and nineteenth centuries and was often romanticized in literature and music.<sup>376</sup> 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.”<sup>377</sup> 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.”<sup>378</sup> The romantic allure of tuberculosis has been much examined

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<sup>374</sup> Berrios, “Melancholia and Depression,” 300.

<sup>375</sup> Benjamin Malzberg, “Mortality among Patients with Involution (sic) Melancholia,” *American Journal of Psychiatry* 93 (1937): 1231–38; Vera Norris, *Mental Illness in London* (Oxford: Oxford University Press, 1959).

<sup>376</sup> Ashley Wilsey, “Half in Love with Easeful Death: Tuberculosis in Literature,” *Humanities Capstone Projects* 11 (2012): 1–39.

<sup>377</sup> Susan Sontag, *Illness as Metaphor and AIDS and its Metaphors* (New York: Macmillan, 2001). NB this is a conjoint later edition, Sontag’s two short texts originally being separately published in 1978 and 1989.

<sup>378</sup> David A. Shephard, “Tuberculosis in Time,” review of *The White Death*, by Thomas Dormandy, *Canadian Medical Association Journal* 163, (2000): 1024; Rene Dubois and J. Dubois, *The White Plague: TB, Man and Society* (New Brunswick, NJ: Rutgers University Press, 1952), 58.



by medical and literary historians.<sup>379</sup> 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."<sup>380</sup> 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."<sup>381</sup> 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.<sup>382</sup> 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.<sup>383</sup> 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.<sup>384</sup> According to the historian Leonard

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<sup>379</sup> Carolyn Day, *Consumptive Chic: A History of Beauty, Fashion, and Disease* (London; Bloomsbury Publishing, 2017).

<sup>380</sup> Clark Lawlor and Akihito Suzuki, "The Disease of the Self: Representing Consumption, 1700-1830," *Bulletin of the History of Medicine* 74 (2000): 458, 481, 494.

<sup>381</sup> Gotthard Booth, "Three Psychobiological Paths toward Death: Cardiovascular Disease, Tuberculosis, and Cancer," *Bulletin of the New York Academy of Medicine* 51 (1975): 415.

<sup>382</sup> Helen Bynum, *Spitting Blood: The History of Tuberculosis* (Oxford: Oxford University Press, 2012).

<sup>383</sup> Linda Bryder, *Below the Magic Mountain* (New York: Oxford University Press, 1988).

<sup>384</sup> Hugh Beevor, "On the Declension of Phthisis (Pulmonary Tuberculosis)," *Lancet* 3946 1899: 1005-20.

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.<sup>385</sup> 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.<sup>386</sup> 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.<sup>387</sup> 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.<sup>388</sup> 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.

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<sup>385</sup> Leonard Wilson, "The Historical Decline of Tuberculosis in Europe and America: its Causes and Significance," *Journal of the History of Medicine and Allied Sciences* 45 (1990): 366-96.

<sup>386</sup> McKeown, *The Modern Rise of Population*.

<sup>387</sup> Michael Worboys, "Before McKeown: Explaining the Decline of Tuberculosis in Britain, 1880-1930," in *Tuberculosis Then and Now: Perspectives on the History of an Infectious Disease*, ed. Flurin Condrau and Michael Worboys (Montreal: McGill-Queen's University Press, 2010), 148-70.

<sup>388</sup> Arthur Newsholme, "The Relative Importance of the Constituent Factors Involved in the Control of Pulmonary Tuberculosis," *Transactions, Epidemiological Society of London* 25 (1906): 31-140.

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 of 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.<sup>389</sup> 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.<sup>390</sup> 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?<sup>391</sup> 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,

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<sup>389</sup> Mott, "Tuberculosis," 78; Clouston, "Phthisical Insanity," 939-44; Crookshank, "The Frequency, Causation, Prevention, and Treatment of Phthisis Pulmonalis in Asylums," 673.

<sup>390</sup> Cullen, "Post-mortem in the Victorian Asylum," 289.

<sup>391</sup> 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.<sup>392</sup> 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.<sup>393</sup> 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.

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<sup>392</sup> Smith, "Welcome Release," 122; Ernst, "The Limits of Comparison," 411.

<sup>393</sup> Smith, "Welcome Release," 117-28; Ernst, "The Limits of Comparison," 411-3.

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

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.<sup>394</sup> 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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<sup>394</sup> Smith, "Welcome Release," 117-28; Clayton and Rowbotham, "How the Mid-Victorians Worked, Ate and Died," 1235-53; Szepter, "Industrialization and Health," 75-86.

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.”<sup>395</sup> 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.”<sup>396</sup> 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.<sup>397</sup> 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.<sup>398</sup> 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.<sup>399</sup> 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

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<sup>395</sup> A sulcus in neuroanatomy is a depression or groove in the cerebral cortex.

<sup>396</sup> An excess of small active blood vessels throughout the tissue.

<sup>397</sup> Atheroma is a deposit of fatty material on the inside surface of an artery.

<sup>398</sup> Personal communication from Professor A K Foulis, Professor of Pathology, University of Glasgow.

<sup>399</sup> 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.

Pathological Finding	Number	Cause of death sub-category				
	Total n=103	Exhaustion n=18	Cerebral n=9	Vascular n=26	Infective n=34	Other n=16
Brain 'softening'	38 (37%)	5/18 (28%)	6/9 (66%)	11/26 (42%)	11/34 (32%)	6/16 (38%)
'Shrunken' grey matter or cortex	32 (31%)	6/18 (33%)	6/9 (66%)	6/26 (23%)	12/34 (35%)	2/16 (13%)
'Oedema' of white matter	32 (31%)	5/18 (28%)	6/9 (66%)	9/26 (35%)	7/34 (21%)	5/16 (31%)
'Congestion/Hyperaemia'	39 (38%)	14/18 (78%)	0/9 (0%)	8/26 (31%)	15/34 (44%)	2/16 (12%)
'Atheroma' of basal vessels	28 (27%)	7/18 (39%)	6/9 (66%)	8/26 (31%)	4/34 (12%)	3/16 (19%)

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.<sup>400</sup> 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."<sup>401</sup>

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.<sup>402</sup> 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

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<sup>400</sup> Plum, "Prospects for Research on Schizophrenia," 384-8; G. B. David, "The Pathological Anatomy of the Schizophrenias," in *Schizophrenia: Somatic Aspects*, ed. Derek Richter (Oxford: Pergamon Press, 1957), 93-130.

<sup>401</sup> James Cowles Prichard, "Observations on the Connexion of Insanity with Diseases in the Organs of Physical Life: Illustrated by a Remarkable Case," *Provincial Medical Journal and Retrospect of the Medical Sciences* 7 (1844): 324.

<sup>402</sup> See e.g. Christina Mayerl et al, "Atherosclerosis Research from Past to Present—on the Track of Two Pathologists with Opposing Views, Carl von Rokitansky and Rudolf Virchow," *Virchows Archive* 449 (2006): 96-103.

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.

Pathological Finding	Total group (n=103)	Cause of death sub-category				
		Exhaustion (n=18) n (% of group)	Cerebral (n=9) n (% of group)	Vascular (n=26) n (% of group)	Infective (n=37) n (% of group)	Other (n=18) n (% of group)
Hypertrophy n (%)	30 (28%)	3 (17%)	2 (22%)	11 (42%)	10 (27%)	4 (22%)
Myocardium soft/friable/fatty n (%)	46 (43%)	10 (56%)	4 (44%)	12 (46%)	15 (40%)	5 (27%)
Atheroma of cardiac vessels n (%)	41 (38%)	6 (33%)	4 (44%)	17 (65%)	10 (27%)	5 (27%)

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.

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

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.<sup>403</sup> 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.”<sup>404</sup>

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<sup>403</sup> Karl Freiherr von Rokitansky, *A Manual of Pathological Anatomy*. Vol. 3 (Philadelphia, PA: Blanchard & Lea, 1855); John Hughlings Jackson, "A Lecture on Softening of the Brain," *Lancet* 2714 (1875): 335-9.

<sup>404</sup> John P Gray, "General Paresis or Complete Progressive Paralysis," *Transactions of the Medical Society of the State of New York* 133 (1866): 69; cited in Andrews, "Death and the Dead-house," 9.

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.<sup>405</sup> 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.<sup>406</sup>

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 *Recherches sur le Ramollissement du Cerveau* that softening (“ramollissement”) in the brain was “the most frequent cerebral lesion.”<sup>407</sup> 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.<sup>408</sup> In 1843, Maxine Durand-Fardel, a Parisian neurologist and pathologist, published his *Treatise on Cerebral Softening*. This volume largely focused on the lacunae (small cavitory lesions)

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<sup>405</sup> Barfoot, “David Skae: Resident Asylum Physician,” 469-88.

<sup>406</sup> Hunter and McAlpine, *Psychiatry for the Poor*, 196.

<sup>407</sup> Louis Léon Rostan, *Recherches sur le Ramollissement du Cerveau* (Paris: Bechet, 1820), 4.

<sup>408</sup> John Abercrombie, *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.<sup>409</sup>

Rokitansky described cerebral softening in detail in his 1855 book *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<sup>410</sup>. In 1856, Virchow opined that “yellow softening” of the brain was secondary to arterial obliteration and that inflammation was secondary.<sup>411</sup> 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.<sup>412</sup> 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.<sup>413</sup> 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 *Lancet* article in 1875

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<sup>409</sup> Maxine Durand-Fardel *Traité du Ramollissement du Cerveau* (Paris: JB. Baillière, 1843).

<sup>410</sup> Rokitansky, *A Manual of Pathological Anatomy*.

<sup>411</sup> Mayerl et al, "Atherosclerosis Research," 101.

<sup>412</sup> Embolization of material means spread via the arteries. Amy Gunning et al, "Mural Thrombosis of the Internal Carotid Artery and Subsequent Embolism," *Quarterly Journal of Medicine* 33 (1964): 155-95.

<sup>413</sup> Bernard Tomlinson, Garry Blessed and Martin Roth, "Observations on the Brains of Non-demented Old People," *Journal of the Neurological Sciences* 7 (1968): 331-56.

“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.<sup>414</sup> 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.<sup>415</sup> 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, *A Manual of Diseases of the Nervous System*, to the topic of cerebral congestion and hyperaemia.<sup>416</sup> 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.<sup>417</sup> He published what some have considered to be the first modern text book of neurology (*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

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<sup>414</sup> Jackson, "A Lecture on Softening of the Brain," 335, 336-39.

<sup>415</sup> Gustavo Román, "Cerebral Congestion: A Vanished Disease," *Archives of Neurology* 44 (1987): 444.

<sup>416</sup> William Gowers, *A Manual of Diseases of the Nervous System* (London: Churchill, 1886).

<sup>417</sup> Frank Freeman, "Lincoln finds a Surgeon General: William A. Hammond and the Transformation of the Union Army Medical Bureau," *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.

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.”<sup>418</sup> 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 Hyperaemia* 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.”<sup>419</sup> 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.”<sup>420</sup>

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<sup>418</sup> William Alexander Hammond, *A Treatise on the Diseases of the Nervous System* (New York: Appleton, 1876), 34-42.

<sup>419</sup> Hammond, *Cerebral Hyperæmia*, 6.

<sup>420</sup> Bonnie Blustein, "The Brief Career of 'Cerebral Hyperaemia': William A. Hammond and His Insomniac Patients, 1854–90," *Journal of the History of Medicine and Allied Sciences* 41 (1986): 41.



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.<sup>421</sup> 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 cribriform, as if moth eaten ... vessels changed.”<sup>422</sup> Etat criblé was a term first coined by Durand-Fardel in 1842 to describe the “cribriform 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.<sup>423</sup> 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

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<sup>421</sup> Worboys, "Was there a Bacteriological Revolution in Late Nineteenth-century Medicine?" 20.

<sup>422</sup> Hunter and McAlpine, *Psychiatry for the Poor*, 196.

<sup>423</sup> Maxine Durand-Fardel, *Traité du Ramollissement du Cerveau*.

both of brain and/or heart. With the advent of blood pressure measurements, *état criblé* became known to be associated with hypertension.<sup>424</sup> 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.<sup>425</sup> 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

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<sup>424</sup> Gustavo Román, "On the History of Lacunes, *Etat Crible*, and the White Matter Lesions of Vascular Dementia," *Cerebrovascular Diseases* 13 (2002): 1-6.

<sup>425</sup> Guha Krishnaswamy, "The Inflammation Paradigm and Coronary Artery Disease: What Celsius, Virchow and Gene Knock-outs have taught us," *Cardiovascular and Haematological Disorders-Drug Targets* 10 (2010): 246-56; Mayerl et al, "Atherosclerosis Research," 97-8.

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 sulphonal 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.<sup>426</sup> “Gentlemanly agreements” between asylum staff and coroners means that, the impact of this layer of public protection may

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<sup>426</sup> 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.”<sup>427</sup> 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.<sup>428</sup> 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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<sup>427</sup> Adam, “On Pathological Research in Asylums,” 359.

<sup>428</sup> Andrews, “Case Notes, Case Histories,” 267.

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.<sup>429</sup> 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.<sup>430</sup> More recently using complex refined techniques and a great

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<sup>429</sup> Sturdy, "Scientific Method for Medical Practitioners," 776.

<sup>430</sup> Plum, "Prospects for Research on Schizophrenia," 384-8.

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.”<sup>431</sup> 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.<sup>432</sup>

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

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<sup>431</sup> Wallis, *Investigating the Body*, 182.

<sup>432</sup> Hervey, “A Slavish Bowing Down,” 98-131; Andrews, “They’re in the Trade... of Lunacy.”



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.<sup>433</sup> 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.

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<sup>433</sup> Alan Blum, "The Enigma of the Brain and its Place as Cause, Character and Pretext in the Imagery of Dementia," *History of Human Science* 235 (2012): 108-24.

## 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.<sup>434</sup> John Haslam gave examples of death from exhaustion following the excited behaviour of mania and the self-starvation of melancholia in 1809.<sup>435</sup> 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”

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<sup>434</sup> Louis-Florentin Calmeil, *Dictionnaire de Médecine: Our Répertoire Général des Sciences Médicales Considérées Sous le Rapport Théorique et Pratique* (Paris: Bechet, 1832) 2nd ed; Andrews, ““Of the Termination of Insanity in Death’,” 129-36.

<sup>435</sup> John Haslam, *Observations on Madness and Melancholy: Including Practical Remarks on Those Diseases, Together with Cases, and an Account of the Morbid Appearances on Dissection* (London: J. Callow, 1809).

conjoined with “raving and excitement.”<sup>436</sup> 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.<sup>437</sup> 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.”<sup>438</sup> 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.”<sup>439</sup>

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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<sup>436</sup> Burrows, *Commentaries*, 554.

<sup>437</sup> Pritchard, *A Treatise on Insanity*, 114.

<sup>438</sup> Bell, “On a Form of Disease Resembling Some Advanced Stages of Mania and Fever,” 97; Samuel H. Kraines, “Bell’s Mania,” *American Journal of Psychiatry* 91 (1934): 29-40.

<sup>439</sup> Cecil Beadles, “The Hour of Death,” *British Medical Journal* 1871 (1896): 1409.

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.<sup>440</sup> 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.<sup>441</sup> 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.<sup>442</sup> 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.<sup>443</sup> 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.<sup>444</sup> 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

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<sup>440</sup> Smith, "Welcome Release," 122.

<sup>441</sup> Smith, "'Visitation by God'," 104-16.

<sup>442</sup> Andrews, "'Of the Termination of Insanity in Death'," 131.

<sup>443</sup> Cox, Marland and York, "Emaciated, Exhausted, and Excited," 500-24.

<sup>444</sup> Wright, Jacklin and Themeles, "Dying to Get Out of the Asylum," 591-621.

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.<sup>445</sup> 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.<sup>446</sup> 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."<sup>447</sup> Similarly, Steven Cherry observes that "deaths were *often casually* [my italics] attributed to old age, general decay, general paralysis or "exhaustion after mania."<sup>448</sup> 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.<sup>449</sup> 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.<sup>450</sup> A year later, Thomas Clouston similarly reproached asylums for using imprecise terminology such as exhaustion and decay.<sup>451</sup> 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

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<sup>445</sup> Renvoize and Beveridge, "Mental Illness and the Late Victorians," 19-28.

<sup>446</sup> Onome Atigari et al, "Bipolar Disorder and Its Outcomes: Two Cohorts, 1875–1924 and 1994–2007, Compared," *History of Psychiatry* 27, no. 1 (2016): 75-84.

<sup>447</sup> David Wright, Laurie Jacklin and Tom Themeles, "Dying to Get out of the Asylum," 600.

<sup>448</sup> Cherry, *Mental Health Care in Modern England*, 72.

<sup>449</sup> Shortt, *Victorian Lunacy*, 61, 104.

<sup>450</sup> Bucknill, "Modes of Death Prevalent among Insane," 354-62.

<sup>451</sup> Clouston, "Tuberculosis and Insanity," 36-65.

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.”<sup>452</sup> 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.”<sup>453</sup> Germán Berrios also usefully charts the historical ebbs and flows of the labels fatigue and exhaustion.<sup>454</sup> 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.<sup>455</sup> 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.<sup>456</sup> 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.”<sup>457</sup>

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

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<sup>452</sup> Anna Katharina Schaffner, *Exhaustion: A History* (New York: Columbia University Press, 2016), 5.

<sup>453</sup> *Ibid*, 7, 12.

<sup>454</sup> Germán Berrios, “Feelings of Fatigue and Psychopathology: A Conceptual History,” *Comprehensive Psychiatry* 31 (1990): 140-51.

<sup>455</sup> Angelo Mosso, *La Fatica* (Treves: Fratelli, 1891).

<sup>456</sup> Edward Shorter, *Chronic Fatigue Syndrome* (New Jersey: John Wiley and Sons, 1993).

<sup>457</sup> Pierre Bugard, *La Fatigue* (Paris: Masson, 1960).

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.”<sup>458</sup> 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.<sup>459</sup> 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.”<sup>460</sup> 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.”<sup>461</sup> 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.”<sup>462</sup>

Historians observe that toxæmia 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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<sup>458</sup> Charles Rosenberg, “The Place of George M. Beard in Nineteenth-Century Psychiatry,” *Bulletin of the History of Medicine* 6 (1962): 251.

<sup>459</sup> Charles Féré, *The Pathology of Emotions* (London: University Press, 1899).

<sup>460</sup> Schaffner, *Exhaustion*, 7.

<sup>461</sup> G. W. Foster, “Common Features in Neurasthenia and Insanity: Their Common Basis and Common Treatment,” *American Journal of Psychiatry* 56 (1900): 413.

<sup>462</sup> Clifford Albutt et al, “A Discussion on the Relation of Neurasthenia to Insanity,” *British Medical Journal* 2181 (1902): 1210.

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.<sup>463</sup> 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.<sup>464</sup> There has been little attention to the issue of whether the concepts of neurasthenia, degeneracy and toxæmia 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

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<sup>463</sup> Clouston, *Clinical Lectures on Mental Diseases*, 1883, 596 and 1904, 111.

<sup>464</sup> Richard Noll, “The Blood of the Insane,” *History of Psychiatry* 17, no. 4 (2006): 395-418. These theories became even more developed (and acted upon) in the 1920s and 1930s in the both the UK (Rebecca Wynter, “Ambition, ‘Failure’ and the Laboratory: Birmingham as a Centre of Twentieth-century British Scientific Psychiatry,” *The British Journal for the History of Science* 54 (2021): 19-40) and the USA (Andrew Scull, *Madhouse: A Tragic Tale of Megalomania and Modern Medicine* (New Haven, CT: Yale University Press, 2005).



the latter group.<sup>465</sup> 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.<sup>466</sup> 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.”<sup>467</sup> 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.<sup>468</sup> Furthermore, Melling’s study of Exminster Asylum in the 1880s shows that only 2% of male admissions were ascribed to “overwork/over

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<sup>465</sup> Smith, “Welcome Release,” 118-22.

<sup>466</sup> Ray, “Models of Madness,” 229-64; Anon, Review of John C. Bucknill and Daniel H. Tuke, “A Manual of Psychological Medicine: Containing the History, Nosology, Description, Statistics, Diagnosis, Pathology, and Treatment of Insanity. With an Appendix of Cases,” *American Journal of Psychiatry* 15 (1858): 227-33.

<sup>467</sup> Franklin Klaf and John Hamilton, “Schizophrenia—a Hundred Years Ago and Today,” *British Journal of Psychiatry* 107 (1961): 825.

<sup>468</sup> Ray, “Models of Madness,” 250.

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.<sup>469</sup>

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.<sup>470</sup> 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.”<sup>471</sup> 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.<sup>472</sup> 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.”<sup>473</sup> 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-

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<sup>469</sup> Melling and Forsythe, *The Politics of Madness*, 181-96.

<sup>470</sup> George Engel, “Sudden and Rapid Death during Psychological Stress: Folklore or Folk Wisdom?” *Annals of Internal Medicine* 74 (1971): 771-83; George Engel, “Sudden Death and the ‘Medical Model’ in Psychiatry,” *Canadian Psychiatric Association Journal* 15 (1970): 527-38.

<sup>471</sup> W. G. Atchison Robertson, “Sudden Deaths from Trivial Causes,” *Practitioner* 111 (1923): 111.

<sup>472</sup> Edward Stern, “Acute Confusional Insanity and Delirium,” *British Journal of Psychiatry* 90 (1944): 761-66; Norman Shulack, “Exhaustion Syndrome in Excited Psychotic Patients,” *American Journal of Psychiatry* 102 (1946): 466-75.

<sup>473</sup> Donald Gregg, “Lethal Power of Emotions,” *Mental Hygiene* 20, 1936: 30.

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.<sup>474</sup> 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.<sup>475</sup> 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.<sup>476</sup> 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).<sup>477</sup>

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

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<sup>474</sup> Renvoize and Beveridge. "Mental Illness and the Late Victorians," 19-28.

<sup>475</sup> Klaf and Hamilton, "Schizophrenia—a Hundred Years Ago and Today," 822.

<sup>476</sup> Cox and Marland, "'A Burden on the County'," 263-87.

<sup>477</sup> Cox, Marland and York, "Emaciated, Exhausted, and Excited," 500-24.

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.<sup>478</sup> 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.<sup>479</sup> 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.<sup>480</sup> 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.<sup>481</sup> 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

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<sup>478</sup> Smith, "Visitation by God'," 110.

<sup>479</sup> Melling and Forsythe, *The Politics of Madness*, 198.

<sup>480</sup> David Ranson, "Excited Delirium Syndrome: A Political Diagnosis?" *Journal of Law and Medicine* 19 (2012): 667-72.

<sup>481</sup> Sarathchandra Kodikara, Kristopher Cunningham and Michael Pollanen, "Excited Delirium Syndrome': Is It a Cause of Death?" *Legal Medicine* 14 (2012): 252-4.

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.<sup>482</sup> 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.<sup>483</sup> 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."<sup>484</sup> 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.<sup>485</sup> 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).<sup>486</sup> 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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<sup>482</sup> C. Bamford and H. Bean, "A Histological Study of a Series of Acute Dementia Praecox," *Journal of Mental Science* 78 (1932): 353-61.

<sup>483</sup> Zachariah Laurence, "A Case of Sudden Death, without Any Adequate Post Mortem Appearances," *British Medical Journal* 177 (1860): 376; Kraepelin cited in Karl-Jürgen Bär, "Cardiac Autonomic Dysfunction in Patients with Schizophrenia and Their Healthy Relatives – a Small Review," *Frontiers in Neurology* 6 (2015): 139.

<sup>484</sup> H. Stefan, "Natural Death as a Result of Great Excitement in Acute Psychoses without Actual Anatomically Demonstrable Cause," *Zeitschrift für die gesamte Neurologie und Psychiatrie* 152 (1935): 480.

<sup>485</sup> Bamford and Bean, "A Histological Study," 358.

<sup>486</sup> Charles Larson, "Fatal Cases of Acute Manic-Depressive Psychosis," *American Journal of Psychiatry* 95 (1939): 971-82.

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.<sup>487</sup> 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.<sup>488</sup> 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.<sup>489</sup> 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."<sup>490</sup> 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 sulphonal. 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

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<sup>487</sup> Smith, "Welcome Release," 122-5.

<sup>488</sup> Leslie Topp, "Single Rooms, Seclusion and the Non-restraint Movement in British Asylums, 1838–1844," *Social History of Medicine* 31 (2018): 754-73; Akihito Suzuki, "The Politics and Ideology of Non-restraint: The Case of the Hanwell Asylum," *Medical History* 39 (1995): 1-17; Andrews, "A Failure to Flourish?" 187.

<sup>489</sup> Klaf and Hamilton, "Schizophrenia—a Hundred Years Ago and Today," 825-6.

<sup>490</sup> Shepherd, *Institutionalizing the Insane*, 117.

of eminent Scottish colleagues such as Clouston.<sup>491</sup> [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 way of mitigating fatigue and exhaustion.<sup>492</sup> 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.<sup>493</sup> 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.<sup>494</sup> 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.”

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<sup>491</sup> John Havelock, “Presidential Address on Modern Advances in the Treatment of the Insane,” *Scottish Medical and Surgical Journal* 9 1901: 98-107; Thomas Clouston, “The Medical Treatment of Insanity,” *Journal of Mental Science* 16 (1870): 24-30. Havelock found paraldehyde particularly useful for “intractable nocturnal restlessness,” 104.

<sup>492</sup> [Sir] Robert [Armstrong] Jones, “The Medical Treatment of Insanity,” *British Medical Journal* 2312 (1905): 875-8. Jones found hyoscine, administered subcutaneously to be a “powerful and valuable remedy for the control of acute motor excitement in violent maniacal cases,” 877.

<sup>493</sup> *Ibid*, 878.

<sup>494</sup> John Ferguson, “The Insanity Following Exhaustion, Acute Diseases, Injuries etc.,” *Alienist and Neurologist* 13 (1892): 407-38.

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.”<sup>495</sup> 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.<sup>496</sup> 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.<sup>497</sup> 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

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<sup>495</sup> Jones, "The Medical Treatment of Insanity," 875-6.

<sup>496</sup> Smith, "Welcome Release," 117-28.

<sup>497</sup> 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.<sup>498</sup> 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.<sup>499</sup> 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.<sup>500</sup>

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<sup>498</sup> Hugh Jarvie and Martin Hood, "Acute Delirious Mania," *American Journal of Psychiatry* 108 (1952): 758-63.

<sup>499</sup> Harris et al., "The Morbidity and Mortality linked to Melancholia," 3-14.

<sup>500</sup> David Healy, *The Creation of Psychopharmacology* (Boston: Harvard University Press, 2009).

## 4.2 Methods, Results and Commentary

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.<sup>501</sup> 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.<sup>502</sup> 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.<sup>503</sup> 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

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<sup>501</sup> 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.

<sup>502</sup> Naomi Williams, "The Reporting and Classification of Causes of Death in Mid-Nineteenth-Century England: The Example of Sheffield," *Historical Methods: A Journal of Quantitative and Interdisciplinary History* 29 (1996): 58-71.

<sup>503</sup> Mooney, "Diagnostic Spaces," 357-90.

experienced during the often-prolonged attacks and in these circumstances, exhaustion was often recorded as a cause of death.<sup>504</sup>

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.<sup>505</sup> 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

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<sup>504</sup> Mackenzie Bacon, "On the Modes of Death in Epilepsy," *Lancet* 2331 (1868): 555-6; James Crichton-Browne, "Notes on Epilepsy, and its Pathological Consequences," *Journal of Mental Science* 19 (1873): 19-46.

<sup>505</sup> 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.

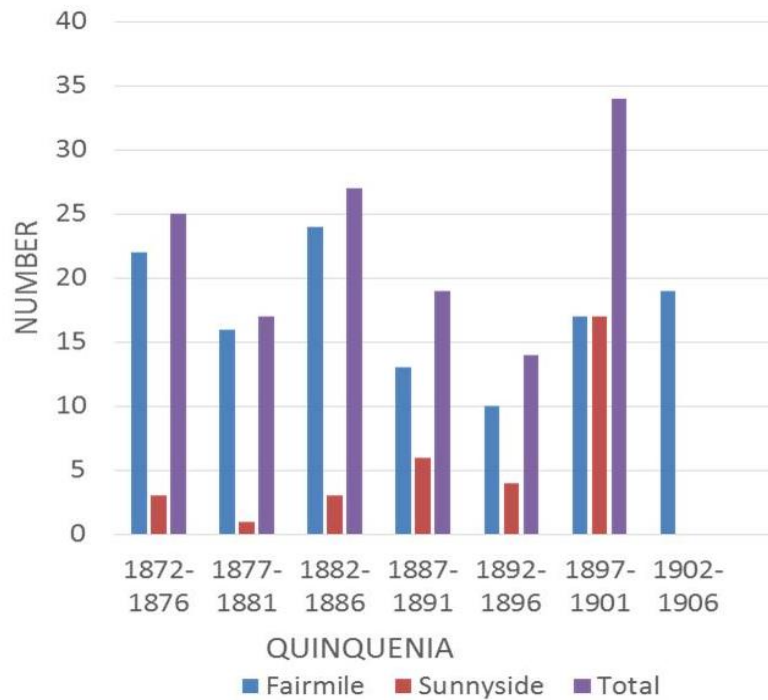


Figure 4-1 Deaths from exhaustion in the two Asylums in five-year blocks from 1872-1900.

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.<sup>506</sup> 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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<sup>506</sup> 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.<sup>507</sup> 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.<sup>508</sup> These observations should be borne in mind when considering the

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<sup>507</sup> David Levy, “Centenary of William Osler’s 1885 Gulstonian Lectures and their Place in the History of Bacterial Endocarditis,” *Journal of the Royal Society of Medicine* 78 (1985): 1039-46; William Osler, “Malignant Endocarditis,” *British Medical Journal* 1263 (1885): 467-70, 522-6, 577-9.

<sup>508</sup> Irving Derby, “Manic-Depressive “Exhaustion” Deaths,” *Psychiatric Quarterly* 7 (1933): 436-49.

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.<sup>509</sup> 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.

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

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%.<sup>510</sup> 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

<sup>509</sup> David Healy et al , "Mortality in Schizophrenia and Related Psychoses: Data from Two Cohorts, 1875–1924 and 1994–2010," *British Medical Journal Open* 2 (2012): 1-10.

<sup>510</sup> 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.<sup>511</sup> 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.<sup>512</sup> 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.<sup>513</sup> 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.<sup>514</sup> 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.<sup>515</sup> 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

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<sup>511</sup> *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.

<sup>512</sup> Renvoize and Beveridge, “Mental Illness and the Late Victorians,” 19-28; Wright, Jacklin and Themeles, “Dying to Get Out of the Asylum,” 600.

<sup>513</sup> Ernst, “The Limits of Comparison,” 404-18.

<sup>514</sup> Risse, “Cause of Death as a Historical Problem,” 175-88.

<sup>515</sup> Douglas Anderton and Susan Leonard, “Grammars of Death: An Analysis of Nineteenth-Century Literal Causes of Death from the Age of Miasmas to Germ Theory,” *Social Science History* 28 (2004): 111-43.



conceptual categories used and shifting diagnostic practices.<sup>516</sup> 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

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<sup>516</sup> Lindsay Prior, "Making Sense of Mortality," *Sociology of Health & Illness* 7 (1985): 167-90; Ernst, "The Limits of Comparison," 404-18.

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.<sup>517</sup> 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.<sup>518</sup> 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.<sup>519</sup> 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.<sup>520</sup> 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.<sup>521</sup>

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<sup>517</sup> These terms were explained and discussed in Chapter 3.

<sup>518</sup> 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.

<sup>519</sup> Berrios, “Memory and the Cognitive Paradigm During the Nineteenth Century,” 194–211.

<sup>520</sup> Yorston and Haw, “Old and Mad in Victorian Oxford,” 416.

<sup>521</sup> Andrews, “Institutionalising Senile Dementia,” 244–57.

### 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.<sup>522</sup> 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.<sup>523</sup>

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.<sup>524</sup> 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

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<sup>522</sup> Harris et al., "Morbidity and Mortality Linked to Melancholia," 3-14.

<sup>523</sup> Derby, "Manic-Depressive 'Exhaustion' Deaths," 443.

<sup>524</sup> 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.<sup>525</sup> 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.<sup>526</sup> 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.

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<sup>525</sup> Smith, “‘Visitation by God’,” 110.

<sup>526</sup> Michael Shepherd, “A Study of Major Psychoses in an English County,” *Maudsley Monographs No.3, Oxford University Press, 1957.*

Name, age (years), gender	Length of attack. Period of illness before plus after Admission. =Total (days)	Type of onset. Number of attack.	Precipitants mentioned in case notes.
EB,36, F	28+17=45	Sudden onset. First.	Worked in domestic service. Temperate. FH +ve Jilted by lover. Kept saying "He took me in." Accused but acquitted of being a kleptomaniac.
RE,53, M	7+100=107	Sudden onset. First.	Had a large family. Lost job. Became impoverished with insufficient food and clothing. Quiet, religiously inclined and temperate. "Grief at loss of situation."
MN,33, F	6+7=13	Sudden onset. First.	Four months pregnant but suckling infant of 16 months. FH +ve.
EB,50, F	? +11	? Second.	Serious illness of sister.
RM, 48, F	14+8=22	Sudden onset. Third.	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."
TB,48, M	7+6=13	Sudden onset. First.	Single labourer who has undergone "privation and had business worry." Attack said to be cause by "disappointment in love" - jilted by his first cousin.
AW,66, M	7+13=20	? First.	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.

MMcD,35, F	14=12=26	Sudden onset. First.	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.
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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.

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.<sup>527</sup> 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.<sup>528</sup> The reasons

<sup>527</sup> Daniel Tuke, *Dictionary of Psychological Medicine* (London, J. & A. Churchill, 1892); German Berrios and Niall Kennedy, "Erotomania: A Conceptual History," *History of Psychiatry* 13, no. 52 2002: 381-400; Peter Ellis and Graham Mellsop, "De Clérambault's Syndrome—A Nosological Entity?" *British Journal of Psychiatry* 146 1985: 90-3.

<sup>528</sup> F. St John Bullen, "Some Remarks on Insanity; Its Classification and Nomenclature," *Bristol Medico-chirurgical Journal* 30 (1883): 205-27; Clouston, *Clinical Lectures on Mental Diseases*, 535-45.

behind exhaustion being seen as a concomitant of the insanity of masturbation have been analysed by Hare and Laqueur.<sup>529</sup> 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.<sup>530</sup> Such reports strengthen historians’ view of the lethal power of emotions and their inhibitory effect on the cardiac cycle.<sup>531</sup> 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.

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<sup>529</sup> Edward Hare, “Masturbatory Insanity: The History of an Idea,” *Journal of Mental Science* 108 (1962): 439-55; Thomas Laqueur, *Solitary Sex: A Cultural History of Masturbation* (Princeton, NJ: Zone Books, 2003).

<sup>530</sup> Shulack, “Sudden “Exhaustive” Death,” 3-12.

<sup>531</sup> 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.<sup>532</sup> 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.<sup>533</sup> 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.<sup>534</sup> Malnourishment is also a frequent explanatory narrative for those who had exhaustion and, again, those who died from it.<sup>535</sup> 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.

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<sup>532</sup> Smith, "Welcome Release," 119-26; Smith, "'Visitation by God'," 113.

<sup>533</sup> Jones, "The Medical Treatment of Insanity," 875-8; Russell, "Mental Physicians and their Patients."

<sup>534</sup> Smith, "Welcome Release," 117-28; McKeown, *The Modern Rise of Population*, 134-60.

<sup>535</sup> Smith, "'Visitation by God'," 112; Marvin Adland, "Review, Case Studies," 38-69.



	Exhaustion deaths with post-mortem (after exclusion of cases with clear-cut physical disease and senile mania)	Exhaustion deaths with case notes examined
Number	57	34
Mean age (years)	52	49
M/F ratio	27M/30F	16M/18F
Mania/Melancholia	38/19	25/9
Number with reported emaciation on admission	–	14
Number reported as well-nourished on admission	–	15
Number with reported emaciation at death	32- 6 severely	20
Number reported as well-nourished at death	15	9
Number with specific report of “food refusal”	–	23
Number tube fed <sup>536</sup>	–	20

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

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<sup>536</sup> 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.<sup>537</sup>

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.<sup>538</sup> Smith comments that some Victorian alienists drew particular attention to the risks of food refusal associated with religious delusions.<sup>539</sup> 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.<sup>540</sup> 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.<sup>541</sup> 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,

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<sup>537</sup> Doody, Beveridge and Johnstone, "Poor and Mad," 887-97.

<sup>538</sup> Renvoize and Beveridge, "Mental Illness and the Late Victorians," 25; Klaf and Hamilton, "Schizophrenia — a Hundred Years Ago and Today," 822.

<sup>539</sup> Smith, "Welcome Release," 122.

<sup>540</sup> Doody, Beveridge and Johnstone, "Poor and Mad," 894.

<sup>541</sup> Hilary Marland, "Under the Shadow of Maternity: Birth, Death and Puerperal Insanity in Victorian Britain," *History of Psychiatry* 23, no. 1 (2012): 78-90.

coercive practice (hence its other designation, force-feeding), serving to emphasise the patient's lack of agency.<sup>542</sup> Larson postulated that exhaustion was a direct result of dehydration and fall in total blood volume rather than the diminished food intake.<sup>543</sup> 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.<sup>544</sup> 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.<sup>545</sup> Building on this evidence, this chapter explores this cardiac hypothesis via

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<sup>542</sup> Ian Miller, "'A Prostitution of the Profession?' Forcible Feeding, Suffrage and Medical Ethics, 1909-14," *Social History of Medicine* 26 (2014): 225-45

<sup>543</sup> Larson, "Fatal Cases of Acute Manic-Depressive Psychosis," 971-82.

<sup>544</sup> R. Locher, "On the Sudden Death of Mental Patients and the Acute Catatoniform Syndrome with Fatal Outcome," *Monatsschrift für Psychiatrie und Neurologie* 103 (1941): 278.

<sup>545</sup> Derby, "Manic-Depressive 'Exhaustion' Deaths," 447; Larson, "Fatal Cases of Acute Manic-Depressive Psychosis," 982.

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.

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

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.”<sup>548</sup> 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.<sup>549</sup> Wallis describes how the advent of devices to measure pulse (the sphygmograph) and, later, blood pressure and blood

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<sup>546</sup> Pulse: - Definitely abnormal:- 100/min and/or irregular; Probably abnormal:- rate over 90/min and/or weak, poor tone.

<sup>547</sup> Heart signs: - Definite:- Cardiac hypertrophy and/or murmur; Probable:- heart sounds abnormal and/or weak, muffled.

<sup>548</sup> Steven M. Stowe, “Seeing Themselves at Work: Physicians and the Case Narrative in the Mid-nineteenth-century American South,” *American Historical Review* 101 (1996): 60.

<sup>549</sup> Jennifer Wallis, “Bloody Technology: The Sphygmograph in Asylum Practice,” *History of Psychiatry* 28, no. 3 (2017): 297 –310.

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.<sup>550</sup> 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.<sup>551</sup> 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.<sup>552</sup> 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

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<sup>550</sup> Michael Finn, “The West Riding Lunatic Asylum and the Making of the Modern Brain Sciences in the Nineteenth Century” (PhD diss., University of Leeds, 2012), 105-7.

<sup>551</sup> 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.

<sup>552</sup> 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.<sup>553</sup> 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.<sup>554</sup> 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."<sup>555</sup>

#### **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

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<sup>553</sup> Adland, "Review, Case Studies," 38-69: Jarvie and Hood, "Acute Delirious Mania," 758-63.

<sup>554</sup> Wallis, *Investigating the Body*, 71.

<sup>555</sup> 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.<sup>556</sup> In a similar vein, John Ferguson warned against those who might “substitute the insanity of drugs for the insanity of disease.”<sup>557</sup> 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.”<sup>558</sup> More robustly, in 1907, Charles Hill, the President of the American Psychiatric Association, described current drug options as “a pile of rubbish.”<sup>559</sup> 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.”<sup>560</sup> 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.<sup>561</sup> The notable exception to this tentativeness is Scull whose overviews of the topic capture the ambivalence of alienists towards medication for the insane.<sup>562</sup>

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 *prima facie* 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

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<sup>556</sup> G. W Foster, “Common Features in Neurasthenia and Insanity,” 412.

<sup>557</sup> John Ferguson, “The Insanity Following Exhaustion,” 438.

<sup>558</sup> Daniel H. Tuke, *Chapters in the History of the Insane* (London: Kegan Paul, 1882), 486-7.

<sup>559</sup> Charles Hill, “Presidential Address: How we can best Advance the Study of Psychiatry,” *American Journal of Insanity* 64 (1907): 6.

<sup>560</sup> Charles Rosenberg, “The Therapeutic Revolution: Medicine, Meaning, and Social Change in Nineteenth-century America,” *Perspectives in Biology and Medicine* 20 (1977): 485.

<sup>561</sup>: Shepherd, *Institutionalizing the Insane*, 139.

<sup>562</sup> See e.g. Andrew Scull, “Somatic Treatments and the Historiography of Psychiatry,” *History of Psychiatry* 5, no. 17 (1994): 1-12.

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.<sup>563</sup> 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.”<sup>564</sup> 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.<sup>565</sup> 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.<sup>566</sup> 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

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<sup>563</sup> Havelock, “Presidential Address,” 104.

<sup>564</sup> A. S. Presley, “From Madhouses to Mental Hospitals: Does History Repeat Itself?” *Scottish Medical Journal* 28 (1983): 73.

<sup>565</sup> Wheeler, *Fair Mile Hospital*, 98.

<sup>566</sup> 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.”<sup>567</sup>

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.”<sup>568</sup> 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.”<sup>569</sup> 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.”<sup>570</sup> 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,

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<sup>567</sup> Turner, “A Diagnostic Analysis of the Casebooks of Ticehurst House Asylum, 1845–1890,” 61.

<sup>568</sup> Roy Porter, *A Social History of Madness. Stories of the Insane* (London: Weidenfeld & Nicolson, 1987); Sally Swartz, “Shrinking: A Post-Modern Perspective on Psychiatric Case Histories,” *South African Journal of Psychology* 26 (1996): 156.

<sup>569</sup> Peter Bartlett, “The Asylum, the Workhouse, and the Voice of the Insane Poor,” 421-32; Allan Ingram, *The Madhouse of Language: Writing and Reading Madness in the Eighteenth Century* (London: Routledge, 2013).

<sup>570</sup> 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.<sup>571</sup> 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.<sup>572</sup> 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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<sup>571</sup> Max Fink, Edward Shorter and Michael Taylor, "Catatonia Is not Schizophrenia: Kraepelin's Error and the Need to Recognize Catatonia as an Independent Syndrome in Medical Nomenclature," *Schizophrenia Bulletin* 36 (2009): 314-20.

<sup>572</sup> Stephan Mann, Stanley Caroff, Harry Bleier, W. K. Welz, et al, "Lethal Catatonia," *American Journal of Psychiatry* 143 (1986): 1374-81.

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.<sup>573</sup> In the current sample, one patient had many of the hallmarks of catatonia.<sup>574</sup> 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.<sup>575</sup> 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.<sup>576</sup> Lipowski comments that delirium was "the Cinderella" of English language psychiatry as it was "taken for granted, ignored and not considered worthy of study."<sup>577</sup> 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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<sup>573</sup> Edward Shorter, "What Historians and Clinicians Can Learn from the History of Medicine: The Example of Fatal Catatonia," *Medicina & Storia* 11 (2011): 5-17; Healy et al., "Mortality in Schizophrenia," 75-84.

<sup>574</sup> 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.

<sup>575</sup> H Palmer, "Scheid's Cyanotic Syndrome: A Case Record and Summary of the Original Monograph of K. Scheid," *British Journal of Psychiatry* 87 (1941): 635-8.

<sup>576</sup> Adamis Dimitrios, Adrian Treloar, Finbarr Martin and Alastair Macdonald, "A Brief Review of the History of Delirium as a Mental Disorder," *History of Psychiatry* 18, no. 4 (2007): 459-69.

<sup>577</sup> Zbigniew Lipowski, "Delirium, Clouding of Consciousness and Confusion," *The Journal of Nervous and Mental Disease* 145 (1967): 227.

terms which were applied developed even greater ambiguity.<sup>578</sup> Delirium is derived from the Latin *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.<sup>579</sup> 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 *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.<sup>580</sup> 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.<sup>581</sup>

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

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<sup>578</sup> German Berrios, “Delirium and Confusion in the 19th Century: A Conceptual History,” *British Journal of Psychiatry* 139 (1981): 439-49.

<sup>579</sup> Richard Krafft-Ebing, *Lehrbuch der Psychiatrie* (Stuttgart: Enke, 1878).

<sup>580</sup> Tuke, *Dictionary of Psychological Medicine*, 573.

<sup>581</sup> Berrios, “Delirium and Confusion in the 19th Century,” 445.

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.<sup>582</sup> 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

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<sup>582</sup> Jarvie and Hood, “Acute Delirious Mania,” 758-63; Harris et al, “The Morbidity and Mortality Linked to Melancholia,” 3-14.

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.<sup>583</sup> 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.<sup>584</sup> 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."<sup>585</sup> 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.<sup>586</sup> 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.<sup>587</sup> In his survey of the field, Shulack summarises the view that a toxemia induced the inability of blood regeneration and

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<sup>583</sup> Conolly Norman, "Acute Confusional Insanity," *Dublin Journal of Medical Science* 89 (1890): 506-18.

<sup>584</sup> Krafft-Ebing, *Lehrbuch der Psychiatrie*.

<sup>585</sup> Charles Mercier, "Kinds of Insanity," *Journal of Mental Science* 51 (1905): 72.

<sup>586</sup> Turner, "A Diagnostic Analysis of the Casebooks of Ticehurst House Asylum," 1-70.

<sup>587</sup> Theodore Deecke, "The Condition of the Brain in Insanity," *American Journal of Psychiatry* 37 (1881): 361-92.

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.<sup>588</sup> 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.<sup>589</sup> 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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<sup>588</sup> Shulack, "Sudden “Exhaustive” Death,” 6.

<sup>589</sup> Adam Al-Diwani et al., "The Psychopathology of NMDAR-antibody Encephalitis in Adults: A Systematic Review and Phenotypic Analysis of Individual Patient Data," *Lancet Psychiatry* 6 (2019): 235-46.

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.<sup>590</sup> 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.”<sup>591</sup> 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.”<sup>592</sup> 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.<sup>593</sup> Marland observes that these rates remained persistently high throughout the nineteenth century.<sup>594</sup> 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.

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<sup>590</sup> Bucknill and Tuke, *Manual of Psychological Medicine*.

<sup>591</sup> Marland, “Under the Shadow of Maternity,” 84.

<sup>592</sup> Susan Hogan, “The Tyranny of the Maternal Body: Madness and Maternity,” *Women’s History Magazine* 54 (2006): 26.

<sup>593</sup> Irvine Loudon, “Puerperal Insanity in the 19th century,” *Journal of the Royal Society of Medicine* 81 (1988): 76–9.

<sup>594</sup> Marland, “Under the Shadow of Maternity,” 80-1.



### 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 of 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.<sup>595</sup> 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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<sup>595</sup> 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.<sup>596</sup> 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

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<sup>596</sup> William F. Bynum and Michael Neve, “Hamlet on the Couch,” in *The Anatomy of Madness*, ed. William F. Bynum, Roy Porter and Michael Shepherd (London: Tavistock, 1985), 3 vols, I: 289-304.

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.<sup>597</sup> 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.<sup>598</sup> 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.<sup>599</sup> 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.<sup>600</sup> 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.

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<sup>597</sup> Shulack, "Exhaustion Syndrome," 466-75.

<sup>598</sup> Smith, "'Visitation by God'," 110.

<sup>599</sup> Derby, "Manic-Depressive 'Exhaustion' Deaths," 448.

<sup>600</sup> 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.”<sup>601</sup> 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.<sup>602</sup> 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.

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<sup>601</sup> Harris et al., “The Morbidity and Mortality linked to Melancholia,” 3-14; Adland, “Review, Case Studies,” 66.

<sup>602</sup> 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 changes 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.<sup>603</sup> Wallis devotes one and half pages of her seminal volume *“Investigating the Body in the Victorian Asylum”* to the topic.<sup>604</sup> 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.”<sup>605</sup> 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.<sup>606</sup> 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.<sup>607</sup> 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.<sup>608</sup> 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.

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<sup>603</sup> Adland, “Review, Case Studies,” 38-69; Renvoize and Beveridge, “Mental Illness and the Late Victorians,” 19-28; Samuel Kowal, “Emotions and Angina Pectoris: An Historical Review,” *The American Journal of Cardiology* 5 (1960): 421-7.

<sup>604</sup> Wallis, *Investigating the Body*, 70-1.

<sup>605</sup> Andrews, “Of the Termination of Insanity in Death,” 131.

<sup>606</sup> Doody, Beveridge and Johnstone, “Poor and Mad,” 891.

<sup>607</sup> Renvoize and Beveridge, “Mental Illness and the Late Victorians,” 23-4.

<sup>608</sup> Wallis, *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.<sup>609</sup> 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.<sup>610</sup> 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.<sup>611</sup> 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.<sup>612</sup> However, as Wallis stresses, a control group from the general population is needed before such assertions can be accepted fully.<sup>613</sup> 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 than in the West Riding of Yorkshire generally."<sup>614</sup> 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.<sup>615</sup>

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

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<sup>609</sup> Cecil Beadles, "On the Degenerative Lesions of the Arterial System in the Insane, with Remarks upon the Nature of Granular Ependyma," *Journal of Mental Science* 41 (1895): 32-50.

<sup>610</sup> Julius Mickle, "The Goulstonian Lectures on Insanity in Relation to Cardiac and Aortic Disease and Phthisis," *British Medical Journal* 1420 (1888): 575- 83.

<sup>611</sup> Drapes, "Phthisis and Insanity," 667-78.

<sup>612</sup> Thomas Duncan Greenlees, *A Contribution to the Study of the Diseases of the Circulatory System in the Insane* (Lewes: H. Wolff, 1885).

<sup>613</sup> Wallis, *Investigating the Body*, 71.

<sup>614</sup> J. Wilkie Burman, "Heart Disease and Insanity," *West Riding Lunatic Asylum Medical Reports* 3 (1873): 255.

<sup>615</sup> Blum, "The Enigma of the Brain," 108-24.

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.”<sup>616</sup> 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.<sup>617</sup> 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.<sup>618</sup> 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.<sup>619</sup> 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.”<sup>620</sup>

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<sup>616</sup> A. D. Morgan, "Some Forms of Undiagnosed Coronary Disease in Nineteenth-century England," *Medical History* 12 (1968): 356.

<sup>617</sup> Maurice Campbell, "Death Rate from Diseases of the Heart: 1876 to 1959," *British Medical Journal* 5356 (1963): 528; Alastair Robb-Smith and Hamish Tearloch, *The Enigma of Coronary Heart Disease* (Chicago, IL: Lloyd-Luke, 1967).

<sup>618</sup> Evan Jones and Evan Bedford, "Syphilitic Angina Pectoris," *British Heart Journal* 5 (1943): 107-20; Paul Klemperer, "The History of Coronary Sclerosis," *American Journal of Cardiology* 5 (1960): 94-107.

<sup>619</sup> Joshua Otto Leibowitz, *The History of Coronary Heart Disease* (Berkeley, CA: University of California Press, 1970); Fredrick Arthur Willius and Thomas Jan Dry, *A History of the Heart and the Circulation* (Philadelphia, PA: Saunders, 1948); Louis Acierno, *The History of Cardiology* (Carnforth, Lancashire: Parthenon Publishing, 1994).

<sup>620</sup> 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.<sup>621</sup> Jarcho stresses this process was both difficult and slow.<sup>622</sup> 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.”<sup>623</sup> Later, Morgan showed convincingly that the fatty changes that Quain had observed were ischaemic in origin.<sup>624</sup> 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.”<sup>625</sup> 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

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<sup>621</sup> Leibowitz, *The History of Coronary Heart Disease*, 73-145.

<sup>622</sup> Jarcho, Review, 413.

<sup>623</sup> Richard Quain, "On Fatty Diseases of the Heart," *Medico-Chirurgical Transactions* 33 (1850): 121.

<sup>624</sup> 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.

<sup>625</sup> Archibald Billing, *Practical Observations on Diseases of the Lungs and Heart* (London: S. Highley, 1852), 73.

of the heart and warned that death from this disease was likely and likely to be sudden.<sup>626</sup> 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.<sup>627</sup>

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.<sup>628</sup> 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.<sup>629</sup> 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.<sup>630</sup> 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.<sup>631</sup> 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.<sup>632</sup>

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<sup>626</sup> Charles East, *The Story of Heart Disease* (Wappingers Falls, New York: Beekman Books Incorporated, 1958).

<sup>627</sup> Evan Bedford, "The Story of Fatty Heart. A Disease of Victorian Times," *British Heart Journal* 34 (1972): 23-8.

<sup>628</sup> J Bizot, "Recherches sur le Coeur et le Système Artériel Chez l'Homme," *Mémoires de la Société Médicale d'Observation* 1 (1837): 262.

<sup>629</sup> Samuel West, "Cases of Complete Obliteration of one Coronary Artery with Remarks upon the Coronary Circulation," *Transactions of the Pathological Society, London* 35 (1884):110.

<sup>630</sup> Rodney Finlayson, "Ischaemic Heart Disease, Aortic Aneurysms, and Atherosclerosis in the City of London, 1868–1982," *Medical History* 29 (1985): 151-68; Jeremiah Morris, "Recent History of Coronary Disease," *Lancet* 6645 (1951): 1-7; George Alexander Gibson, *Diseases of the Heart and Aorta* (Edinburgh: Pentland, 1898), Pentland’s Medical Series, 6, 652-744.

<sup>631</sup> Leon Michaels, "The Eighteenth-Century Origins of Angina Pectoris: Predisposing Causes, Recognition and Aftermath," *Medical History* 45 (2001): 156–63.

<sup>632</sup> James Herrick, "Clinical Features of Sudden Obstruction of the Coronary Arteries," *Journal of the American Medical Association* 59 (1912): 2015-22.

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.<sup>633</sup> 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.

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<sup>633</sup> It was intended to carry out a similar exercise within the Sunnyside records, but the Coronavirus pandemic precluded this.

	Fair Mile ≤ 55 years group	Fair Mile 56- ≤65 years group	Sunnyside ≤ 55 years group	Total group
n	17	13	10	40
Age at death (Years (SD))	44.5 (9)	60.4 (2)	51.2 (3)	51.3 (9)
Gender (M:F)	2:15	5:8	4:6	11:29
Mania: Melancholia	15:2	6:7	8:2	29:11

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.<sup>634</sup> 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.<sup>635</sup> Walton also shows that lack of kinship was a major driver to asylum incarceration, particularly in economically deprived females.<sup>636</sup> Many younger women in

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<sup>634</sup> Julianne Holt-Lunstad, Timothy Smith and Bradley Layton, "Social Relationships and Mortality Risk: A Meta-analytic Review," *PLoS Medicine* 7 (2010): 2-19.

<sup>635</sup> Adair, Melling and Forsythe, "Migration, Family Structure and Pauper Lunacy in Victorian England," 387-91.

<sup>636</sup> Walton, "Lunacy in the Industrial Revolution," 1-22.

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.”<sup>637</sup> Unmarried women may have felt marginalised, lonely, frustrated and unfulfilled possibly leading to insanity.<sup>638</sup> 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.<sup>639</sup> 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.

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<sup>637</sup> Melling and Forsythe, *The Politics of Madness*, 191.

<sup>638</sup> Edward Higgs and Amanda Wilkinson, "Women, Occupations and Work in the Victorian Censuses Revisited," *History Workshop Journal* 81 (2016): 17-38; Shepherd, *Institutionalizing the Insane*, 102.

<sup>639</sup> Alistair MacKinnon, "The Psychopathology of Ischaemic Heart Disease," *The Journal of the Royal College of General Practitioners* 16 (1968): 253. See also Don Carlos Peete, *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.<sup>640</sup> 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.<sup>641</sup> 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.

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<sup>640</sup> Harold Feil, "History of the Treatment of Heart Disease in the Nineteenth Century," *Bulletin of the History of Medicine* 34 (1960): 19-28.

<sup>641</sup> Jones, "Prognosis in Mental Diseases," 1578-83.

Asylum	Length of admission before death				
	Less than 1 week	Less than 4 weeks	4 weeks to 1 year	1 year to 5 years	More than 5 years
Sunnyside n=10	0	0	4	4	2
Fair Mile n=30	1	1	9	11	8
Total n=40	1	1	13	15	10

Table 5-2 Length of stay in the asylum prior to death in the vascular cause of death cohort.

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.

Point measure recorded	Emaciated (or similar)	Well nourished (or similar)	Not clear/not stated
At or near admission	8	10	22
At post-mortem	13	25	2

Table 5-3 Body habitus on admission and at death of the 40 patients from both asylums.

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.



Asylum	Group	Cause of death after post-mortem performed				
		Apoplexy/ Cerebral haemorrhage	Valvular heart disease	Morbis cordis/ Fatty heart	Cardiac failure	Other *
Sunnyside	≤ 55 years n=10	4	2	1	0	3
Fair Mile	≤ 55 years n=17	2	0	10	4	1
Fair Mile	56- ≤65 Years n=13	1	1	7	3	1
Total	n=40	7	3	18	7	5

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.”<sup>642</sup> Rokitansky noted that when the myocardium showed “dirty yellowish discolouration” and “became loose and flabby” then “very frequently” (my italics) there was “ossification of the coronary arteries.”<sup>643</sup> 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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<sup>642</sup> Samuel Black, “A Case of Angina Pectoris, with a Dissection,” *Memoirs of the Medical Society of London* 6 (1797): 41–9.

<sup>643</sup> Rokitansky, *A Manual of Pathological Anatomy*, 154, 159.

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.

	Cause of death/ Pathological finding	Clinical label	
		Mania n=29	Melancholia n=11
Cause of death ascription  n (% of clinical label)	Apoplexy	5 (17%)	2 (18%)
	Valvular Heart Disease	3 (10%)	0 (0%)
	Morbis cordis (fatty heart)	12 (41%)	6 (55%)
	Cardiac failure	6 (20%)	1 (9%)
	Other	3 (10%)	2 (18%)
Pathological change  n (% of clinical label)	Fatty heart pathology (n=26)	18 (62%)	8 (72%)
	Any atheroma (n=31)	20 (69%)	11 (100%)
	Hypertrophy of heart (n=19)	16 (55%)	3 (27%)

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.<sup>644</sup> 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.

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<sup>644</sup> Andrew Steptoe, "Depression and the Development of Coronary Heart Disease," in *Depression and Physical Illness*, ed. Andrew Steptoe (Cambridge: Cambridge University Press, 2007): 53-86; Riccardo Proietti et al., "Mental Stress and Ischemic Heart Disease: Evolving Awareness of a Complex Association," *Future Cardiology* 7 (2011): 425-37.

Pathological change reported in the brain	Number of reports (out of 40 cases)	Pathological change reported in vascular system			
		With atheroma of cerebral vessels (n out of 19 reports)	With fatty heart pathology (n out of 26 reports)	With fatty heart pathology and/or atheroma of any vessel (n out of 34 reports)	With heart hypertrophy (n out of 19 reports)
Brain softening	21	14	13	19	14
Shrunken grey matter or cortex	17	14	12	16	8
Oedema of white and/or grey matter	18	11	14	11	8
Congestion and/or hyperaemia	13	7	8	9	4

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.

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

its probable ischaemic or haemorrhagic origin.<sup>645</sup> 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*.<sup>646</sup> 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

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<sup>645</sup> Mayerl et al., "Atherosclerosis Research," 96-103.

<sup>646</sup> 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.<sup>647</sup> 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.<sup>648</sup> Bouillard, in 1840, demonstrated the direct relationship between preceding rheumatic fever and the subsequent development of rheumatic heart valve pathology.<sup>649</sup> 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

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<sup>647</sup> Evan Bedford, William Hartston and Robert Drew, "An Early Account of Rheumatic Heart Disease by Joseph Brown (1784–1868)," *Medical History* 20 (1976): 76-9.

<sup>648</sup> Matthew Baillie, *The Works of Matthew Baillie, MD, to which is Prefixed an Account of his Life Collected from Authentic Sources by James Wardrop, Surgeon Extraordinary to the King* Vol. 1 (London: Longman, 1825); William Charles Wells, "On Rheumatism of the Heart," *Transactions of the Society for Improving Medical and Chirurgical Knowledge* 3 (1812): 373-424. Wells was an American physician who trained in Edinburgh and who had spent time with John Hunter in Leyden and London.

<sup>649</sup> Jean Bouillard, *Traité Clinique du Rhumatisme Articulaire* (Paris: J.B. Baillière, 1840).

part of their contractile and distensible powers.”<sup>650</sup> 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.<sup>651</sup> 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.<sup>652</sup> 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

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<sup>650</sup> Matthew Baillie, *The Morbid Anatomy of Some of the Most Important Parts of the Human Body* (London: J. Johnson & G. Nicol, 1793), 261.

<sup>651</sup> Richard Quain, "Cerebral Apoplexy: With Some General and Statistical Observations on the Causes of the Disease, and the Relative Frequency of its Occurrence at Different Periods of Life," *London Journal of Medicine* 11 (1849): 27.

<sup>652</sup> Mayerl et al, "Atherosclerosis Research," 97-8.



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 and vessel disease in the population at large and in those with a label of mental illness.<sup>653</sup> 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.<sup>654</sup> 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.”<sup>655</sup> Kirstie Blair argues, that the heart lends itself such interpretations “constantly blurring the boundaries between cultural supposition and apparent medical fact.”<sup>656</sup> 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 aetiological factor in coronary disease.<sup>657</sup> 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.<sup>658</sup> 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

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<sup>653</sup> Engel, "Sudden and Rapid Death during Psychological Stress," 771-83.

<sup>654</sup> Leibowitz, *The History of Coronary Heart Disease*, 165.

<sup>655</sup> Fay Bound Alberti, *Matters of the Heart: History, Medicine, and Emotion* (Oxford: Oxford University Press, 2010), 17.

<sup>656</sup> 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.

<sup>657</sup> John Ryle and W. T. Russell, "The Natural History of Coronary Disease: A Clinical and Epidemiological Study," *British Heart Journal* 11 (1949): 370-89.

<sup>658</sup> Kowal, "Emotions and Angina Pectoris," 421-7.

Bartholomew's Hospital in London, was clear that "death has followed mental excitement more frequently than bodily excitement."<sup>659</sup> 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."<sup>660</sup> These conclusions chime with other commentators' views on the topic and the current findings.<sup>661</sup> 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.<sup>662</sup> 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

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<sup>659</sup> Peter Latham, *Lectures on Subjects Connected with Clinical Medicine* (London: Longmans, 1846), 413.

<sup>660</sup> MacKinnon, "The Psychopathology of Ischaemic Heart Disease," 253.

<sup>661</sup> Leibowitz, *The History of Coronary Heart Disease*, 165; Holt-Lunstad, "Social Relationships and Mortality Risk," 2-19.

<sup>662</sup> Marjorie Levine-Clark, "Dysfunctional Domesticity: Female Insanity and Family Relationships Among the West Riding Poor in the Mid-nineteenth Century," *Journal of Family History* 25 (2000): 341-61; Melling and Forsythe, *The Politics of Madness*, 191; Smith, "Family, Community and the Victorian Asylum," 109-24.

interruptions to it, and, on the other, are expressions of it and (e) the inheritance of an ambitious or conscientious personality pattern.”<sup>663</sup>

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 Russell 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.<sup>664</sup>

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.<sup>665</sup> 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.<sup>666</sup> 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 Laënnec in 1838, Quain in 1850 and Herxheimer in 1901, repeatedly

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<sup>663</sup> Ryle and Russell, "The Natural History of Coronary Disease," 386.

<sup>664</sup> *Ibid*, 387.

<sup>665</sup> Acierno, *The History of Cardiology*, 148-51; Louis Gallavardin, *La Degenescence Graisseuse du Myocarde* (Paris: JB Baillière, 1900).

<sup>666</sup> 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.<sup>667</sup> 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.<sup>668</sup> 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.<sup>669</sup> 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.<sup>670</sup> The French surgeon and pathologist Jean Lobstein introduced

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<sup>667</sup> René Laennec and John Forbes, *A Treatise on the Diseases of the Chest and on the Mediate Auscultation* (New York: S. Wood & Sons, 1830); Gotthold Herxheimer, "Über Fettfarbstoffe," *Deutsche Medizinische Wochenschrift* 27 (1901): 607-9.

<sup>668</sup> Bedford, "The Story of Fatty Heart," 27.

<sup>669</sup> Randall Thompson et al, "Atherosclerosis across 4000 years of Human History: the Horus study of four Ancient Populations," *Lancet* 381 (2013): 1211-22.

<sup>670</sup> 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.<sup>671</sup> 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.<sup>672</sup> 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.<sup>673</sup> The historian Schiller called this a victory of “solidism over humoralism.”<sup>674</sup> 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

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<sup>671</sup> Jean Frederic Lobstein, *Traité d’Anatomie Pathologique* (Paris: Levrault, 1829). The synonymous term atherosclerosis also came to be used frequently after this time.

<sup>672</sup> Rudolf Virchow, *Cellular Pathology: As based upon Physiological and Pathological Histology* (London: John Churchill, 1860), 162-4; Mayerl et al, “Atherosclerosis Research,” 97-8.

<sup>673</sup> Mayerl, “Atherosclerosis Research,” 97-8.

<sup>674</sup> Francis Schiller, “Concepts of Stroke before and after Virchow,” *Medical History* 14 (1970): 130.

may be related to his personality, Quain also did not acknowledge others' work on this theme.<sup>675</sup> 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.<sup>676</sup>

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.<sup>677</sup> 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.<sup>678</sup> 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.<sup>679</sup> One of Edinburgh Medical School's renowned physicians, George Alexander Gibson (1854-1913), published his widely acclaimed textbook *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.<sup>680</sup> 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

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<sup>675</sup> Morgan. "Some Forms of Undiagnosed Coronary Disease" 347. Morgan describes Quain as prone to waffle and the DNB is even more damning, noting, in 1896, that Quain's character was marred by "the violence of his party feelings, his jealousy and the readiness with which he imputed improper motives to all that differed from him". D'Arcy Power, "Sir Richard Quain," *Dictionary of National Biography*, 47 (1896): 90-1. Accessed May 12, 2022, [https://en.wikisource.org/wiki/Dictionary\\_of\\_National\\_Biography,\\_1885-1900/Quain,\\_Richard](https://en.wikisource.org/wiki/Dictionary_of_National_Biography,_1885-1900/Quain,_Richard).

<sup>676</sup> Carl Weigert, "Uber die Pathologischen Gerinnungsvorgange," *Virchow Archive of Pathological Anatomy* 79 (1880): 87.

<sup>677</sup> George Balfour, *Diseases of the Heart and Aorta* (Philadelphia, PA: Lindsay & Blakiston 1876).

<sup>678</sup> William Osler, *Lectures on Angina Pectoris and Allied States* (New York: Appleton, 1897).

<sup>679</sup> Leon Michaels, "Aetiology of Coronary Artery Disease: An Historical Approach," *British Heart Journal* 28 (1966): 258-264.

<sup>680</sup> Gibson, *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.<sup>681</sup> Thereafter, however, there was a rapid rise in the clinical manifestations of coronary atheroma (angina and coronary thrombosis).<sup>682</sup> 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.<sup>683</sup> 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.<sup>684</sup>

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.<sup>685</sup> 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.”<sup>686</sup> 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

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<sup>681</sup> Registrar-General. Annual Report of Births, Deaths, and Marriages in England and Wales, H.M.S.O., London, 73, 1910.

<sup>682</sup> Leon Michaels, "Aetiology of Coronary Artery Disease," 258-60.

<sup>683</sup> Herrick, "Clinical Features of Sudden Obstruction of the Coronary Arteries," 2015-22; James Herrick, "Thrombosis of the Coronary Arteries," *Journal of the American Medical Association* 72 (1919): 387-90.

<sup>684</sup> James Herrick, "An Intimate Account of my Early Experience with Coronary Thrombosis," *American Heart Journal* 27 (1944): 1-18; James Herrick, *A Short History of Cardiology* (Springfield, IL: Charles C Thomas, 1942).

<sup>685</sup> Finlayson, "Ischaemic Heart Disease," 151-168; Morris, "Recent History of Coronary Disease," 1-7.

<sup>686</sup> 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.<sup>687</sup> 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.<sup>688</sup> 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.<sup>689</sup> 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.<sup>690</sup> 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,

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<sup>687</sup> John Lindsay Steven, "Cases of Spontaneous Rupture of the Heart, and Remarks on the Pathology of the Condition, with Special Reference to Fatty Degeneration and Disease of the Coronary Arteries," *Glasgow Medical Journal* 22 (1884): 413- 27; Arnold Levene, "Spontaneous Rupture of the Heart: A Historical Review," *Postgraduate Medical Journal* 38 (1962): 334-7.

<sup>688</sup> Finlayson came to the view that it was the per capita consumption of manufactured cigarettes (which rose linearly from zero in 1890) that followed the prevalence of coronary deaths during the twentieth century "with almost relentless consistency" which explained this most. In the United Kingdom, the consumption of cigarette tobacco increased almost twentyfold from 0.2 lb. per head per annum in 1900 to 3.9 lb. in 1960. See Barbara Forey et al., *International Smoking Statistics. A Collection of Historical Data from 30 Economically Developed Countries, 2nd edition* (Oxford: Oxford University Press, 2002).

<sup>689</sup> Alistair MacKinnon, "The Origin of the Modern Epidemic of Coronary Artery Disease in England," *The Journal of the Royal College of General Practitioners* 37 (1987): 174-6.

<sup>690</sup> J. A. Gorringe, "A Fresh Look at what Everybody Knows about Ischaemic Heart Disease: Discussion Paper," *Journal of the Royal Society of Medicine* 77 (1984): 390.



becomes less paradoxical in the light of this dissociation.<sup>691</sup> 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.<sup>692</sup> 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."<sup>693</sup> 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 Beadles' study and the current one is that he found atheroma "where

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<sup>691</sup> Geoffrey Rose and Michael Marmot, "Social Class and Coronary Heart Disease," *Heart* 45 (1981): 13-9.

<sup>692</sup> Beadles, 'On the Degenerative Lesions of the Arterial System in the Insane,' 32-50.

<sup>693</sup> 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.”<sup>694</sup> 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.<sup>695</sup> 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.”<sup>696</sup> 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.”<sup>697</sup> Ultimately, however, Beadles came down on the side of the prevalent toxæmia 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.<sup>698</sup>

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.”<sup>699</sup> 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.<sup>700</sup> The coronary arteries, particularly the left one,

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<sup>694</sup> Beadles, ‘On the Degenerative Lesions of the Arterial System in the Insane,’ 32, 37, 38.

<sup>695</sup> F. St John Bullen, “A Review of the Influence of Reflex and Toxic Agencies in the Causation of Insanity and Epilepsy,” *Journal of Mental Science* 41 (1895): 187-202.

<sup>696</sup> Fred M. T. Skae, “Vacuolation of the Nuclei of Nerve Cells in the Cortex,” *British Medical Journal* 1742 (1894): 1075

<sup>697</sup> Beadles, ‘On the Degenerative Lesions of the Arterial System in the Insane,’ 43, 44.

<sup>698</sup> Noll, “The Blood of the Insane,” 395-418.

<sup>699</sup> William Mott, “Obituary: William Julius Mickle,” *British Medical Journal* 2977 (1918): 103.

<sup>700</sup> Mickle, “The Goulstonian Lectures on Insanity,” 575- 83.

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.”<sup>701</sup> 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.<sup>702</sup> 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.<sup>703</sup>

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.<sup>704</sup> 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

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<sup>701</sup> Ibid, 580.

<sup>702</sup> 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).

<sup>703</sup> 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.

<sup>704</sup> William Baly, “Rupture of the Heart, Consequent on Fatty Degeneration, Associated with Softening of the Brain,” *Transactions of the Pathological Society, London* 3 (1851): 264-69.

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.<sup>705</sup> 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.<sup>706</sup> 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.<sup>707</sup> 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."<sup>708</sup> 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

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<sup>705</sup> Suzanne Segerstron and Gregory Miller, "Psychological Stress and the Human Immune System: A Meta-analytic Study of 30 years of Inquiry," *Psychological Bulletin* 130 (2004): 601-30.

<sup>706</sup> Smith, "Welcome Release," 117-28; Ernst, "The Limits of Comparison," 404-18.

<sup>707</sup> Szreter, "Industrialization and Health," 75-86.

<sup>708</sup> Helen McCarthy, *Double Lives: A History of Working Motherhood* (London: Bloomsbury Publishing, 2020).

the consequence of an inflammatory process, which may have been, in turn, a consequence of frequent and unrestrained infections and/or stress.<sup>709</sup> 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.

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<sup>709</sup> Virchow, *Cellular Pathology*, 162; Krishnaswamy, "The Inflammation Paradigm and Coronary Artery Disease," 246-56; Mayerl et al, "Atherosclerosis Research," 97-8.

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.”<sup>710</sup> 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.”<sup>711</sup> 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.<sup>712</sup> 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,

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<sup>710</sup> Smith, “‘Visitation by God’,” 113.

<sup>711</sup> Scull, *Museums of Madness*, 192.

<sup>712</sup> Adair, Melling and Forsythe, “Migration, Family Structure and Pauper Lunacy in Victorian England,” 373-401.

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.<sup>713</sup>

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.<sup>714</sup> 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.<sup>715</sup> 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.<sup>716</sup> To explain this observation, most historians consider that poor physical health and emaciation on admission to the asylum

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<sup>713</sup> Elaine Murphy, "The Administration of Insanity in England 1800 to 1870," in *The Confinement of the Insane: International Perspectives, 1800–1965*, ed. Roy Porter and David Wright (Cambridge: Cambridge University Press, 2003), 334-49.

<sup>714</sup> Wallis, *Investigating the Body*; Wright, Jacklin and Themeles, "Dying to Get Out of the Asylum," 591–621; Melling and Forsythe, *The Politics of Madness*; Smith, "Welcome Release," 117-28; Michael and Hirst, "Recording the Many Faces of Death at the Denbigh Asylum," 40-51; Smith, "'Visitation by God'," 109-10.

<sup>715</sup> Cutler, Deaton and Lleras-Muney, "The Determinants of Mortality," 97-120; Wright, Jacklin and Themeles, "Dying to Get Out of the Asylum," 591–621; Jones, "Prognosis in Mental Diseases," 1578-83.

<sup>716</sup> 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.<sup>717</sup> 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.<sup>718</sup> 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).<sup>719</sup> 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.<sup>720</sup> 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.<sup>721</sup> 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

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<sup>717</sup> Scull, *Museums of Madness*.

<sup>718</sup> Szreter, "Economic Growth, Disruption, Deprivation, Disease, and Death," 693-728.

<sup>719</sup> Melling and Forsythe, *The Politics of Madness*; Adair, Melling and Forsythe, "Migration, Family Structure and Pauper Lunacy," 338.

<sup>720</sup> Smith, "Welcome Release," 119-21.

<sup>721</sup> Wright, Jacklin and Themeles, "Dying to Get Out of the Asylum," 599-600; Renvoize and Beveridge, "Mental Illness and the Late Victorians," 19-28.

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.<sup>722</sup> 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.<sup>723</sup>

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.<sup>724</sup> 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.<sup>725</sup> 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.<sup>726</sup> 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

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<sup>722</sup> Ernst, “The Limits of Comparison,” 404-18; Szreter, “Author Response: Debating Mortality,” 705-9.

<sup>723</sup> Robertson, “Sudden Deaths from Trivial Causes,” 110-24; Gregg, “Lethal Power of Emotions,” 30; Engel, “Sudden and Rapid Death during Psychological Stress,” 771-83.

<sup>724</sup> Shepherd and Wright, “Madness, Suicide and the Victorian Asylum,” 175-96

<sup>725</sup> Smith, “Insanity and the ‘Civilising Process’,” 250-68; Smith, “Behind Closed Doors; Lunatic Asylum Keepers,” 301-27.

<sup>726</sup> 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.<sup>727</sup> This association is also evident in developing countries and in displaced populations such as refugees.<sup>728</sup> 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.<sup>729</sup> 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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<sup>727</sup> Waltraud Ernst, "Crossing the Boundaries of 'Colonial Psychiatry'. Reflections on the Development of Psychiatry in British India, c. 1870–1940," *Culture, Medicine, and Psychiatry* 35 (2011): 536-45.

<sup>728</sup> Michael Toole and Ronald Waldman, "Prevention of Excess Mortality in Refugee and Displaced Populations in Developing Countries," *Journal of the American Medical Association* 263 (1990): 3296-302.

<sup>729</sup> Shephard, "Tuberculosis in Time," 1024-5; Ashley Wilsey, "Half in Love with Easeful Death," 1-39.

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-mortems 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.<sup>730</sup> 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.<sup>731</sup> 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.<sup>732</sup> 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.

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<sup>730</sup> Shortt, *Victorian Lunacy*, 61; Kodikara, Cunningham and Pollanen, "Excited Delirium Syndrome," 252-4; Ranson, "Excited Delirium Syndrome," 667-72.

<sup>731</sup> Shulack, "Exhaustion Syndrome in Excited Psychotic Patients," 466-75; Smith, "'Visitation by God'," 109-110.

<sup>732</sup> 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), toxæmia and neurasthenia.<sup>733</sup> 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."<sup>734</sup> 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.<sup>735</sup> This study makes the key observation, found in

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<sup>733</sup> Wallis, *Investigating the Body*, 70; Noll, "The Blood of the Insane," 395-418.

<sup>734</sup> Harris et al, "The Morbidity and Mortality linked to Melancholia," 3-14; Adland, "Review, Case Studies," 66.

<sup>735</sup> 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 to 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.<sup>736</sup> 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.<sup>737</sup> 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

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<sup>736</sup> Krishnaswamy, "The Inflammation Paradigm and Coronary Artery Disease," 246-56; Mayerl et al., "Atherosclerosis Research," 96-103.

<sup>737</sup> Andrews, "Death and the Dead-house," 9; Wallis, *Investigating the Body*; Cullen, "Post-mortem in the Victorian Asylum," 284-6; Hurren, "'Abnormalities and Deformities'."

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.<sup>738</sup> 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.<sup>739</sup> 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.<sup>740</sup> 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

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<sup>738</sup> Smith, "Welcome Release"; Scull, *The Most Solitary of Afflictions*; Digby, "The Changing Profile of a Nineteenth-century Asylum," 739-48; Andrews, "Death and the Dead-house," 12.

<sup>739</sup> Wallis, "The Bones of the Insane," 196-211.

<sup>740</sup> 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).<sup>741</sup> 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."<sup>742</sup> 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.<sup>743</sup> 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.<sup>744</sup> 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

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<sup>741</sup> Hervey, "A Slavish Bowing Down;" Andrews, "They're in the Trade".

<sup>742</sup> Cullen, "Post-mortem in the Victorian Asylum," 285; Andrews, "Case Notes, Case Histories," 267.

<sup>743</sup> Blustein, "The Brief Career of 'Cerebral Hyperaemia'," 24-51.

<sup>744</sup> 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.<sup>745</sup> 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.<sup>746</sup> By the mid- to later parts of the century,

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<sup>745</sup> Plum, "Prospects for Research on Schizophrenia," 384-8.

<sup>746</sup> 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.”<sup>747</sup> 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.<sup>748</sup> The majority of the doctors performing post-mortems in the two series discussed here were graduates of either Glasgow or Edinburgh Universities,

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<sup>747</sup> Charles Rosenberg, "Contested Boundaries: Psychiatry, Disease, and Diagnosis," *Perspectives in Biology and Medicine* 49 (2006): 413.

<sup>748</sup> Nystrom, "Post-mortem Examinations," 164-72.

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."<sup>749</sup> 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.<sup>750</sup> 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.<sup>751</sup> 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."<sup>752</sup> 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 asylumdom. 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

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<sup>749</sup> Ray, "Models of Madness," 251.

<sup>750</sup> Cullen, "Post-mortem in the Victorian Asylum," 286-7.

<sup>751</sup> Buklijas, "The Laboratory and the Asylum," 311-25.

<sup>752</sup> 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.<sup>753</sup> 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.<sup>754</sup> 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

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<sup>753</sup> Andrews, “Case Notes, Case Histories,” 267.

<sup>754</sup> 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 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.<sup>755</sup> 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.<sup>756</sup> 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.<sup>757</sup>

Luckin remarks that researchers whose "primary training has been medical" may oversimplify findings and take insufficient account of the social and economic context.<sup>758</sup> 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."<sup>759</sup> Houston

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<sup>755</sup> Scull, *The Most Solitary of Afflictions*, 18-48.

<sup>756</sup> Andrew Scull, *Madness in Civilization* (New Jersey: Princeton University Press, 2015); Murphy, "The Administration of Insanity," 334.

<sup>757</sup> Murphy, "The Administration of Insanity," 336; Michel Foucault, *History of Madness*, Translated by Jonathan Murphy and Jean Khalifa (London/New York: Routledge, 2006).

<sup>758</sup> Luckin, "Death and Survival in the City," 53-61.

<sup>759</sup> Keith Wrightson, *Earthly Necessities: Economic Lives in Early Modern Britain* (Yale, New Haven, CT: Yale University Press, 2000), 15.

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.<sup>760</sup> 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.”<sup>761</sup> 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.<sup>762</sup> 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.”<sup>763</sup> 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.”<sup>764</sup> 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.”<sup>765</sup> 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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<sup>760</sup> Houston, “A Latent Historiography,” 289-310.

<sup>761</sup> Mark Micale and Roy Porter, *Discovering the History of Psychiatry* (New York: Oxford University Press, 1994), 6.

<sup>762</sup> McCarthy et al., “Lives in the Asylum Record,” 358-79; McGovern, “The Myths of Social Control and Custodial Oppression,” 3-23.

<sup>763</sup> Davey, “Lectures on Insanity,” 581.

<sup>764</sup> Jones, “Prognosis in Mental Diseases,” 1579.

<sup>765</sup> Smith, “Visitation by God,” 113.

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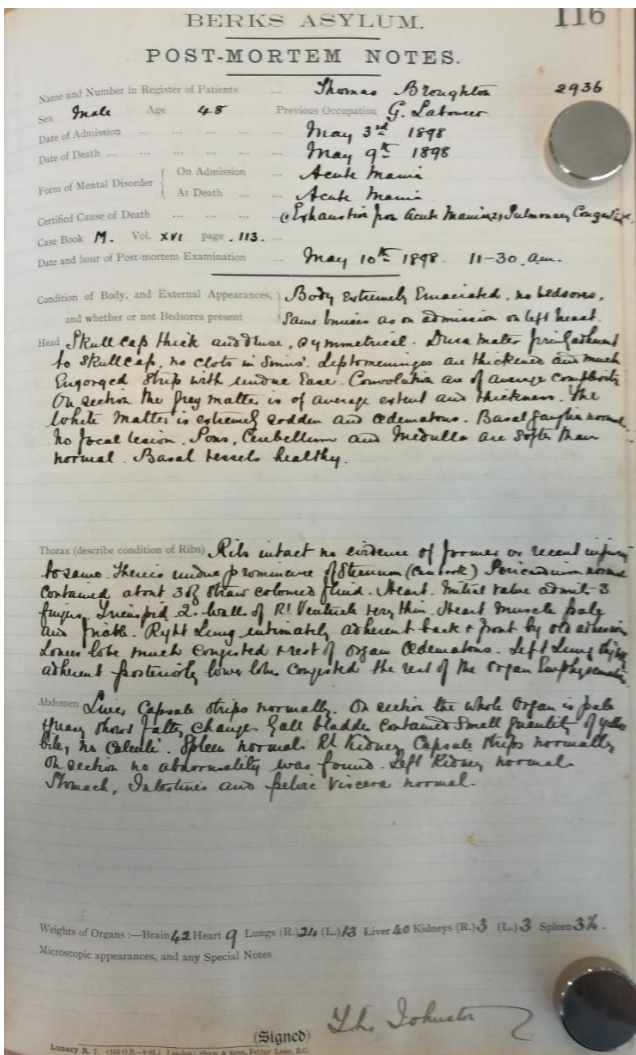
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## Appendix A. Photographs of 6 post-mortem reports from Post-mortem Reports book of Fair Mile asylum.<sup>766</sup>

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

<sup>766</sup> 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.

Contained about 30g straw coloured fluid. Heart. Initial valve admit 3 fingers. Incispid. L. wall of R! Ventricle very thin. Heart muscle pale and friable. Right lung intimately adherent back + front by old adhesions.

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.

BERKS ASYLUM.  
POST-MORTEM NOTES.

13

Name and Number in Register of Patients	...	Joseph Randall
Sex	M	Age 40
Date of Admission	...	May 26 <sup>th</sup> 1902
Date of Death	...	May 30 <sup>th</sup> 1902
Form of Mental Disorder	On Admission	Acute Mania
	At Death	acute Mania
Certified Cause of Death	...	Exhaustion from Acute Mania
Case Book	...	Vol. xxvii page 205
Date and hour of Post-mortem Examination	...	May 31 <sup>st</sup> 1902 11-30 AM

Condition of Body, and External Appearances, and whether or not Bedsores present

The body is well nourished  
No bedsores bruises or marks of

Head

*videlicet*  
Skull cap is thick & dense. Dura mater very adherent to the skull cap also congested. Basal sinuses enlarged. The Leptomeninges are thickened & very congested. They strip with undue ease. The convolutions are of average complexity but show some wasting. On section the grey matter is wasted & very congested. The white matter is congested & the puncta vasculosa are well marked. Basal ganglia are congested. The pons, medulla & cerebellum are soft, congested, & oedematous. Basal vessels atrophied.

Thorax (describe condition of Ribs) Ribs intact no evidence of former or recent injury to same Pericardium normal. Heart initial & aortic valves are atrophied, also the first part of aorta coronary arteries atrophied. Heart muscle pale & friable. There are numerous recent pleural adhesions over the lower left lung Basis of both lungs are acutely congested.

Abdomen Liver Capsule is adherent to Diaphragm over the left lobe. The organ is congested & Macle's fatty change. The Gall bladder contains a small quantity of dark bile, no calculi. Spleen soft & congested. Both kidneys are in a state of acute congestion. The Malpighian bodies are well marked also the atrophy of the medulla. The stomach Large & small intestines normal. Pelvic viscera normal.

Weights of Organs.—Brain 52 Heart 11/2 Lungs (R.) 19 (L.) 14 Liver 11 Kidneys (R.) 1/2 (L.) 4 Spleen 4

Microscopic appearances, and any Special Notes

(Signed) A. P. Lennon-Brown

Lansley St. 7, (A. 31, 140) G.S.—411) London: 114 & 115, Peter Lane, E.C.

Detail of report of Brain and Thorax

Head *intences*  
 Skull cap is thick & dense. Dura Mater very adherent to the skull cap also congested. Basal sinuses engorged. The Leptomeninges are thickened & very congested. They strip with undue ease. The convolutions are of average complexity but show some wasting. On section the grey matter is wasted & very congested. The white matter is congested & has puncta vasculosa are well marked. Basal ganglia are congested. The pons, medulla & cerebellum are soft, congested, & atheromatous. Basal vessels atheromatous.

Thorax (describe condition of Ribs) Ribs intact no evidence of former or recent injury to some Pericardium normal. Heart mitral & aortic valves are atheromatous, also the first part of aortic coronary arteries atheromatous. Heart muscle pale & friable. There are numerous recent pleural adhesions over the lower left lung Basis of both lungs are acutely congested.

Points of note:- Grey matter “wasted and congested”. White matter “congested and punctate vasculosa well marked”. Basal vessels “atheromatous”. Aortic valves and coronary arteries “atheromatous”. Heart muscle “pale and friable.”

Case 3 Post-mortem report from case GH (see Chapter 3)

171 BERKS ASYLUM. POST-MORTEM NOTES.

Name and Number in Registry of Patients *Gertrude Elizabeth Harris.*  
 Sex *F* Age *27* Previous Occupation *Shool-Teacher.*  
 Date of Admission *December 9, 1900*  
 Date of Death *December 10, 1900*  
 Part of Mental Disorder *On Admission*  
 At Death *Acute mania*  
 Certified Cause of Death *Acute Mania*  
 Case Book *F* Vol. *XXI* page *257*  
 Date and hour of Post-mortem Examination *December 12, 1900. 11 A.M.*

Condition of Body and External Appearances, and whether or not Diseases present *The body is well preserved. There has been no injury in right arm. The*

Head *no evidence of any disease of the brain.*  
*On removing the skull the appearance of two small tumours on inner surface of outer table of frontal region, and these tumours are in form of small nodules, they are dark grey & attached in color to calvaria. They appear to have been in existence for some time. The tumours are of the nature of meningiomas, they are firm & white. The tumour on the right side is situated in the middle of the frontal region, and is about 1/2 inch in diameter. There are no markings of blood vessels in the tumour. On section the tumour is found to be composed of a dense mass of cells, and is surrounded by a thin layer of connective tissue. It contains the vessels and nerves of the brain.*

Thorax *Heart - Both valves healthy. The lungs are normal. R. lobe of heart is filled with fresh blood. L. lobe is almost entirely empty. The coronary arteries are normal. The aorta is normal. The pulmonary artery is normal. The pulmonary veins are normal. The pleurae are normal. The diaphragm is normal. The stomach is normal. The intestines are normal. The liver is normal. The spleen is normal. The kidneys are normal. The bladder is normal. The uterus is normal. The ovaries are normal.*

Abdomen *Liver - Capsule is normal and white and average size. The surface appears normal. The gall bladder is normal. The stomach is normal. The intestines are normal. The spleen is normal. The kidneys are normal. The bladder is normal. The uterus is normal. The ovaries are normal.*

Weights of Organs:- Brain *17 1/2* Lungs *(R) 11 1/2 (L) 11 1/2* Kidneys *(R) 5 (L) 6 1/2* Spleen *7 1/2*  
 Microscopic appearances, and any Special Notes

(Signed) *P. L. Jones*

Leadb. R. 7, (S. 16, 161) S. 16-162 London: Wm. & Son, Printers, Ltd.



Detail of Report of Head

Head are no lacerations or marks of violence

On removing the scalp there are marks of two small lacerations on inner surface of scalp anteriorly in frontal region, and third small one (one laceration in occipital region, they are dark green & blackish red in color and contain clotted blood. They apparently have been in existence for three to six days. On the hairy surface of the scalp no bruising is visible - The skull cap is extremely thin and brittle especially in parietal region which is about 1/12 inch in thickness. There is fracture of skull. Dura mater is healthy. Longitudinal sinus contains black clot. On removing the dura there is an extensive haemorrhage of great occurrence between the arachnoid and pia mater; it occupies the occipital and parietal regions on both sides.

Thorax (describes condition of Ribs) Brain as a whole is of fair consistency, gyri are of average complexity - On section grey matter is markedly hyperemic white matter is also hyperemic. Ventricles & pons healthy, there is no focal lesion. Cerebellum pons and medulla healthy - Basal ganglia healthy.

Throat Pulmonary

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"

BERKS ASYLUM. 186

POST-MORTEM NOTES.

Name and Number in Register of Patients ... *Maria Volt 2857*

Sex *P* Age *53* Previous Occupation *Housewife*

Date of Admission ... *December 1. 1897*

Date of Death ... *February 22 1905*

Form of Mental Disorder: On Admission ... *Delusional Insanity*  
At Death ... *Delusional Insanity*

Certified Cause of Death ... *Primitiva. Zebra Heart*

Case Book *P* Vol. *XIII* page *490*

Date and hour of Post-mortem Examination ... *February 23. 1905. 11.30. A.M.*

Condition of Body, and External Appearances, and whether or not Bedsores present: *The body is very well nourished, there are no tumours, bedsores or marks of violence.*

Head: *Skull cap is thick and dense. Dura mater normal, no clot in sinuses. Septum in jugular bulb present, the jugular vein average size. Protona on a whole is fairly firm. Ears are spacious and healthy. The entire grey matter is pale. White matter is fairly white and clean. The brain. Basal ganglia are pale. There is no free brain. Ventricular appendage is healthy. Cerebellum pons and medulla appear normal. Basal meninges healthy.*

Thorax (describe condition of Ribs): *Reheptate, there is evidence of old, or recent, injury to the ribs. Pericardium is adherent to sternum, it contains about 3j fluid. Myocardium is soft and flabby showing fatty change. L. Ventricle is hypertrophied. There is commencing atheroma of aortic valve. This valve and cusps are normal. Both lungs are adherent to pleurae by recent adhesions. The organs themselves are collapsed and emphysematous at the apex.*

Abdomen: *Liver. Capsule is on the outside cavity. On section the organ shows well marked fatty change. Gall bladder contains dark bile calculi. Spleen is soft and small. Both kidneys show adherent capsules. They are markedly enlarged. The left also shows commencing fatty change. On the inner side of the stomach, the mucosa is atant and the present a punctiform appearance. They may possibly be due to typhoid some 2 or 3 weeks ago. The stomach, pancreas and the liver and pelvic viscera are healthy.*

Weights of Organs:—Brain *14* Heart *12* Lungs (R) *13* (L) *11* Liver *8* Kidneys (R) *6* (L) *5* Spleen *14* g.

Microscopic appearances, and any Special Notes

(Signed) *E. J. Denny*

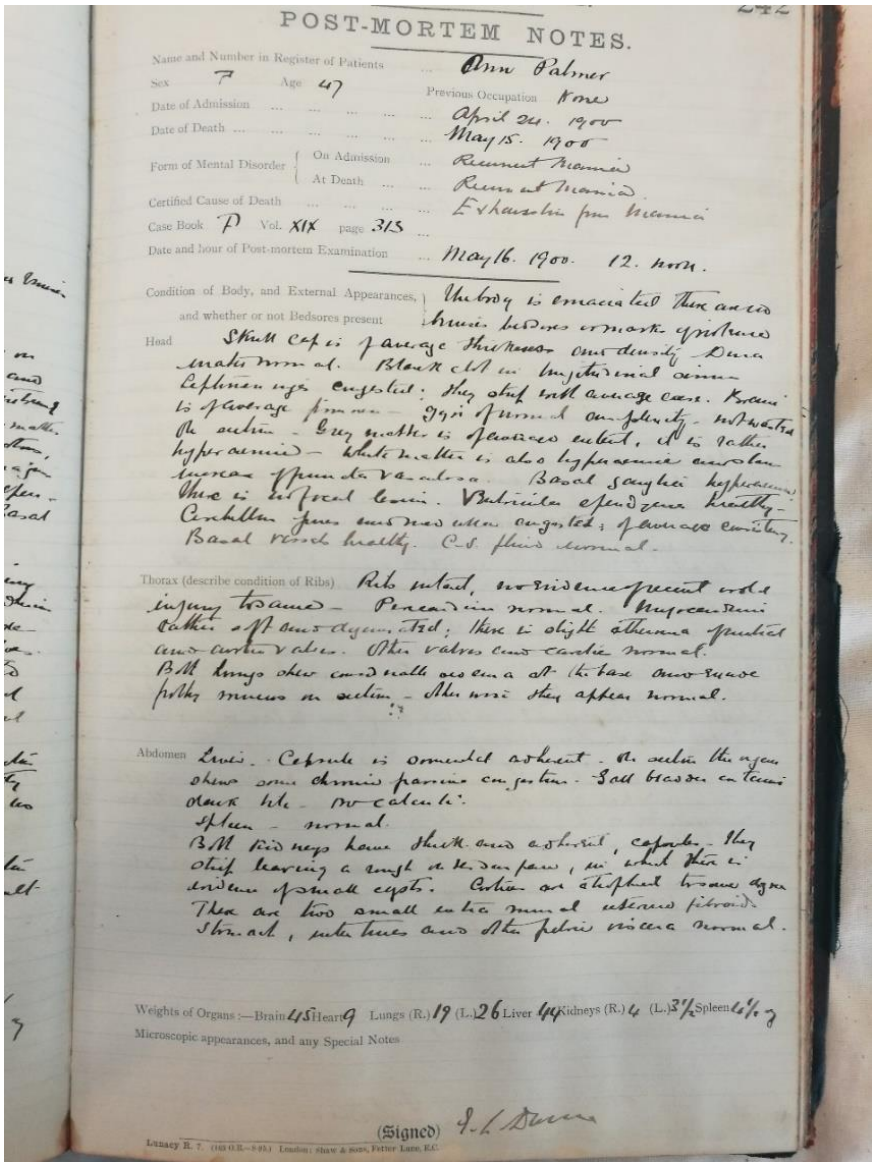
LONDON: H. K. LAYTON, 36, SOUTH BROADWAY, LONDON, E.C. 4. PRINTED BY SHAW & SON, FETTER LANE, E.C.

Detail of heart and vessels report.

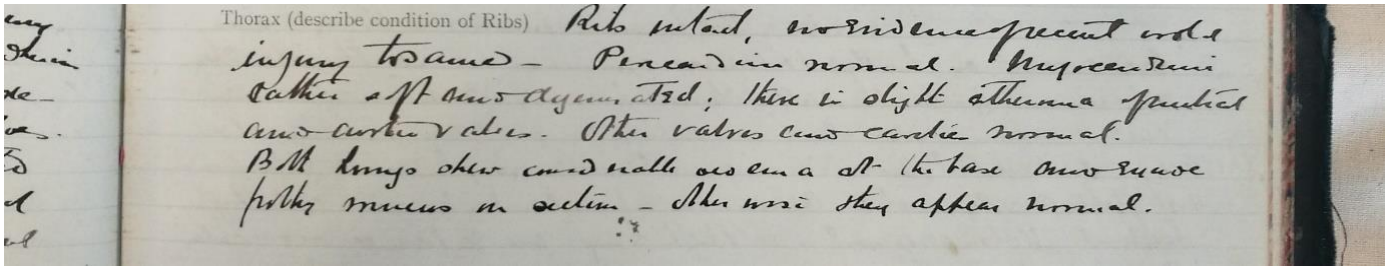
*about 3j fluid. Myocardium is soft and flabby showing fatty change. L. Ventricle is hypertrophied. There is commencing atheroma of aortic valve. This valve and cusps are normal. Both lungs are adherent to pleurae by recent adhesions. The organs themselves are collapsed and emphysematous at the apex.*

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

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.

BERKS ASYLUM. 46

POST-MORTEM NOTES.

Name and Number in Register of Patients ... *Frederick John Gibbs*  
 Sex *Male*. Age *42*. Previous Occupation *Antique dealer*.  
 Date of Admission ... *January 6<sup>th</sup> 1894*.  
 Date of Death ... *November 26<sup>th</sup> 1896*.  
 Form of Mental Disorder { On Admission ... *Melancholia*  
 At Death ... *Melancholia*  
 Certified Cause of Death ... *Pneumonia*  
 Case Book *Clinic* Vol. *I* page *70*  
 Date and hour of Post-mortem Examination ... *November 27<sup>th</sup> 11-15 am.*

Condition of Body, and External Appearances, and whether or not Bedsores present. *Body is fairly well nourished. No bedsores, bruises or marks of violence.*

Head *Skull cap thick and dense especially on frontal region. Symmetrical Dura mater is slightly adherent over the table. Its cloth in sinus. Lep-tomeninges are much thickened and congested strips with average ease. Brain is extremely soft. Convolutionis are of average complexity. On section the grey matter is markedly hyperaemic. White matter is sodden and oedematous. No focal lesion. A small aneurism about the size of a pea was found on the left middle cerebral artery. Small atheromatous patches on basal vessels. The Pons, Cerebellum and Medulla were all extremely soft.*

Thorax (describe condition of Ribs) *Ribs intact: there is no evidence of recent or former injury to same. Pericardium normal. Endocardium stained with blood. Myo-cardium friable. Mitral valve admits lips of 2 fingers. Tricuspid admits two. Aortic valves slight atheroma. Rh lung shows upper lobes in state of red hepatate lower lobe congested. Left lung congested throughout. Pleura thickened and adherent everywhere by old adhesions.*

Abdomen *Liver capsule strips with ease. On section no abnormality is seen. Gall bladder contained small quantity of dark bile. No calculi. Spleen large size and dark colour showing congestion. Rt Kidney, capsule adherent. On section there is some cortical atrophy otherwise no pathological change. Left Kidney capsule strips easily, also some atrophy of cortex. The walls of the bladder are much thickened. Other pelvic organs are normal.*

Weights of Organs — Brain *47* Heart *8½* Lungs (R.) *34* (L.) *17½* Liver *46* Kidneys (R.) *3½* (L.) *3½* Spleen *6½* g

Microscopic appearances, and any Special Notes

(Signed) *L. H. Johnston*

Lansley St. 7. (112 D.R. 614). Location: Shaw & Sons, Peter Lane, E.C.

Detail of Head and Thorax report

*Brain is extremely soft. Convolutionis are of average complexity. On section the grey matter is markedly hyperaemic. White matter is sodden and oedematous. No focal lesion. A small aneurism about the size of a pea was found on the left middle cerebral artery. Small atheromatous patches on basal vessels. The Pons, Cerebellum and Medulla were all extremely soft.*

Thorax (describe condition of Ribs) *Ribs intact: there is no evidence of recent or former injury to same. Pericardium normal. Endocardium stained with blood. Myo-cardium friable. Mitral valve admits lips of 2 fingers. Tricuspid admits two. Aortic valves slight atheroma. Rh lung shows upper lobes in state of red hepatate*

Points of note:- Hyperaemic grey matter. White matter "sodden and oedematous". Patches of atheroma on basal vessels. Heart muscle friable. Atheroma of aortic valves.



**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  $\pm$  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).

Clinical Subgroup (Ave age at death )	Cause of death	Ages at Death (years)	n	Cause of death sub-group  Number of cases and age at death of Subgroup (years Mean ± SD)
MANIA n=165 (58 years)  Those with Senile Mania shown with a A *  Group without senile mania n=139 (55 years)	Exhaustion	64,64*,51,64,62,48,79*,55,47,72*,75*,47,71*,86*,66,69*,73*,40,54,44,36,68*,63,71*,78*,74	26	Exhaustion  n=26 62 ± 13
	Cerebral softening	79,x,55,64,71,63,66,40,70,63*,68,42,78,60	14	Brain  Conditions  n=39 70±11
	Senile Decay	73,70*,78*,85*,85,76,78,83,70,70,79,70,68,79,76*,66,75,72,71,81	20	
	Cerebral Atrophy	60,47,68	3	
	Senility	74*,82*	2	Vascular disease  n=36 58±14
	Morbis Cordis	71,80,66,70,64,71*,43,74,58,53,63,67*,7250,53,49,58	17	
	Heart Disease	63,41,74,41,34,60,67,59	8	
	Cardiac failure	26,34,53,61,78	5	
	Apoplexy	76,74*,27,54,68	5	
	Syncope	45	1	Infections  n=54 47±15
	Phthisis Pneumonale	42,34,x,46,23,48,40,32,51,40,38,33,48,32,36,53,39,26,45,30,47,49,22,31	24	
	Tuberculosis	44	1	
	Pneumonia	73,76,66,70,62*,69,69,52,49,52,28,73*,44,59	14	
	Pyelonephritis	49,59	2	
	Bronchitis	53	1	
	Septicaemia	61	1	
	Enteric fever /Colitis	24,38,34,55,39	5	
	Dysentery	47,65*,71,52	4	

	Abscess of liver	62	1	Others n=10 56±14
	Meningitis	24	1	
	Cancer (Rectum, duodenum, uterus(2), pancreas, liver, sarcoma)	52,72,38,44,71,69,67	7	
	Volvulus	53	1	
	Peritonitis	63	1	
	Suffocation	33	1	
MELAN- CHOLIA n=70 (57 years) Those with senile melancholia shown with a * Group without senile melancholia (56 years)	Exhaustion	60,58,54,44,50,64,44	7	
	Cerebral softening	62,60,60,51,61,63,47,63	8	Brain Conditions n=16 66±10
	Senile Decay	72,72,70*,75,81*	5	
	Senility	63,70,88	3	
	Morbis Cordis	74,66,45,74,59,65*,62	7	Vascular disease n=17 62±8
	Heart Disease	71,66,66,54	4	
	Cardiac failure	59,59,47	3	
	Apoplexy	72,60	2	
	Lung gangrene	57	1	
	Phthisis Pneumonale	36,24,52,36,56,25,38,40	8	Infections n=25 50±14
	Pneumonia	42,55,44,56,78,66,55,40,37,78*,68	11	
	Enteric fever /Colitis	49	1	
	Dysentery	63*,38,57	3	
	Meningitis	59	1	
	Empyema	53	1	
	Cancer (bladder)	55	1	Others



	Shock after operation	59	1	n=5 58±3
	Peritonitis	55	1	
	Spinal Atrophy	58	1	
	Bright's Disease	62	1	
DEMENTIA n=169 (66 years) Those with secondary dementia shown with a ‡	Exhaustion	76‡	1	Exhaustion n=1 76
	Cerebral softening	60,67,78,84,63,65,56,65,64,64‡,68,50,66,67,45	15	Brain Conditions n=88 74±9
	Senile Decay	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	65	
	Cerebral Atrophy	75,66,67,52,68	5	Vascular disease n=21 61±10
	Senility	79,68,83	3	
	Morbis Cordis	49,76,64,61,56‡,74,68,56,77	9	
	Heart Disease	71,68‡,62‡	3	
	Cardiac failure	64,63,42,46,64	5	Infections n=52 54±16
	Apoplexy	52,48,55,72‡	4	
	Phthisis Pneumonale	52,26‡,41,56,59,56,41‡,27,22‡,27,57‡,62,56, 54,41,37,54,69,62,66,41	21	
	Tuberculosis	37	1	
	Pneumonia	60,33,63,73,72,69,28,49,76,29,56,70, 62,77,64,65‡,68,66‡,63,70,66,49,27	23	
	Bronchitis	X,	1	
	Enteric fever/Colitis	38	1	
	Dysentery	31‡,49,80,82	4	

	Empyema	30‡	1	Others n=7 65±15
	Cancer (Bladder, stomach, sarcoma)	50, 82, 50‡	3	
	Subdural	65	1	
	Unknown	49,80,82	3	
IDIOCY n=72 (39 years)	Exhaustion	29,23,70	3	Exhaustion n=3 40±21
	Cerebral softening	55,56,60	3	Brain Conditions n=12 63±9
	Senile Decay	66,72,77,70,70,71	6	
	Cerebral Atrophy	50,48, x	3	Vascular disease n=7 41±15
	Morbis Cordis	65,43	2	
	Cardiac failure	50,28,15	3	
	Apoplexy (check)	39,47	2	
	Phthisis Pneumonale	26,32,18,38,16,27,13,19,17,48,x,16,46,17, 22,19 ,17,33,32	19	Infections n=34 32±16
	Tuberculosis	14	1	
	Pneumonia	34,20,28,48,78,46,51,50,22,48	10	
	Enteric fever/Colitis	18	1	
	Pyelonephritis	49	1	
	Dysentery	52,47	2	
	Marasmus	16,29,13,17	4	
	Epilepsy	64, 39	2	Others n=16 32±16
	Status epilepticus	54, 15	2	
	Peritonitis	21	1	
	Diabetes	23	1	

	Bright's disease	42	1	
	Cancer (bowel)	44	1	
	Shock	43	1	
	Ataxia	51	1	
	Unknown	20,17	2	
EPILIPSY n=61 (45 years)	Exhaustion	52,43,24,22,26,59,23,21,55,24,29	11	Exhaustion n=11 34±14
	Cerebral softening	71,53,66	3	Brain
	Senile Decay	85,84,79,49,61	5	Pathology
	Cerebral Atrophy	60	1	n=9 68±12
	Morbis Cordis	72	1	Vascular disease
	Cardiac failure	56,64,29, x,19,69	6	n=9 53±18
	Apoplexy (check)	56,57	2	
	Phthisis Pneumonale	55,61,32,26,56,31	6	
	Pneumonia	33,31,43,59,48,41,53	7	Infections
	Enteric fever /Colitis	34	1	n=16 44±12
	Dysentery	41, 67	2	
	Epilepsy	27,29,35,39,43,38	6	Others
	Status epilepticus	15,37,23,35,36,31	6	n=16 35±12
	Epileptic dementia	38,70	2	
	Emphysema	14	1	
	Ca (Jaw)	42	1	

**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**

Cause of death of death category	Examples (in order of frequency in each sub-group)
INFECTIONS	<p>Tuberculosis (TB) - Phthisis pulmonale, acute TB, TB, Acute miliary TB</p> <p>Pneumonia - Bronchopneumonia, Acute Pneumonia, Empyema, Pleurisy, Bronchitis</p> <p>Gastro-intestinal Infections - Enteric fever, Dysentery.</p> <p>Other Infections - Meningitis, Septicaemia</p>
OTHER	<p>Various cancers.</p> <p>Intestinal obstruction, Volvulus, Perforation, Peritonitis, Rupture of bowel, Ulcerative colitis, Hydatid cysts.</p> <p>Choking</p> <p>Suicide – Asphyxia, Laceration of throat.</p>
VASCULAR	<p>Heart disease, Heart failure, Morbis Cordis, Valvular disease of heart, Apoplexy, Cerebral haemorrhage, Rupture of aneurysm, Congestion of lungs.</p>
BRAIN CONDITIONS	<p>Brain atrophy, Cerebral softening, Senile decay, Brain congestion, Senility</p>
EXHAUSTION	<p>Exhaustion from mania, Exhaustion from melancholia</p>

## 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.

Number Initials Age at Death Sex	Clinical diagnosis on admission	Occupation	Cause of death	External post- mortem findings	Internal post-mortem findings	Length of stay group* Number and length of Attack
1. ET 17 F	Acute delirious mania	Domestic servant.	Acute delirious mania	Well nourished “tending to stout” Dark fluid on upper thirds of thighs as “though commencing to be gangrenous”	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.	B First
2. JK 40 M	ME	-	Phthisis	Tall and very emaciated	Brain NAD Heart valves normal. Heart muscle “flabby and hypertrophied” Lungs – cavities and adhesions.	E ?

					Consolidation in upper segments with congestion in lower parts	
3. GL 52 M	Delusional insanity	-	Apoplexy	Well nourished— “very corpulent” Bedsore	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.	E ?
4. JC 40 M	Recurrent mania	-	Acute military TB	Well nourished	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++	E ?
5. JT 32 F	ME	-	Duodenal ulceration	Well nourished	Brain NAD Heart valves normal. Heart muscle normal apart from some	C ?

					Thickening. Patches of atheroma in aorta. Lungs – apical scars otherwise NAD Perforations in duodenum AND “bowel” Exudation++	
6. JR 55 M	ME	Joiner	Asphyxia	Well nourished Ligature marks	Pia and cerebral tissue congested. Trachea is compressed. Lungs “gorged” with blood. Heart muscle is hypertrophied. Valves OK	B First 6 years
7. MM 43 F	ME		Hydatid cyst Pleurisy	Much emaciated	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.	E ?

					Cysts in liver one of which was put in the museum.	
8. JP 50 F	Delusional mania	Housekeeper	Brain Atrophy	Much emaciated	Subdural fluid++ Bullae under pia. Generalised convolutional thinning esp. at the fissure of Rolando. Ventricles much dilated. Vessels at base of brain were atheromatous. arteries and aorta were atheromatous. Heart valves normal. . Lungs – mild oedema	C First 14 weeks
9. AL 55 F	Delusional insanity	-	Ca stomach	Body of aged woman emaciated	Pia etc normal Brain was firm and large. Vessels at base of brain were atheromatous Heart muscle is “somewhat flabby” Lungs old TB and were somewhat oedematous. Stomach showed Ca masses in and near stomach. Liver showed secondaries.	E ?



10. WM 38 F	ME With delusions	-	Phthisis Pneumonale	Much emaciated	Subdural fluid++ Bullae under pia. Convolutions atrophied "slightly". Coronary arteries and aorta were "slightly" atheromatous. Lungs riddled with tubercular granulations with some cavitation	D ?
11. GJ 35 F	Acute mania with delirium	Domestic servant	Pneumonia	A little emaciated	Sub-arachnoid fluid++ Brain substance was "pale". Heart valves normal. . Lungs – pneumonia at base with consolidation	A First 3 months
12. DM 27 M	ME	-	Phthisis Pneumonale	Much emaciated	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.	D ?

					Heart valves normal. Lungs showed TB cavities, nodules, and old scars.	
13. MAF 24 F	ME	-	Phthisis Pneumonale	Abdomen much distended	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.	C ?
14. RMCK 35 F	Acute mania with delirium	-	Phthisis Pneumonale	A little emaciated	Sub-arachnoid fluid++ Brain substance was "pale". Heart valves normal. . Lungs – pneumonia at base with consolidation	D ?
15. AA 25 F	MA	-	Septic Pneumonia	Much emaciated	Grey matter was "pale" and slightly thinned Atheroma of valves. Lung lobes consolidated and "gangrenous" with	E ?

					some tubercular nodules	
16. JS 51 F	MA	Servant and knitter	Ca uterus	Much emaciated	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 ?

					consolidation and pleural thickening and calcification.	
20. MO 55 F	MA	Domestic	Exhaustion from mania	Well nourished	Pia etc normal Brain substance was congested. Cyst behind fissure of Rolando irregular "longstanding" Vessels at base of brain were atheromatous Heart hypertrophied and dilated. Mitral valve was incompetent. Epicardial fat Lung oedematous at base.	D First 4 years
21. SC 48 F	MA	Knitter and washer	Exhaustion from mania	Much emaciated	Pia etc. normal Brain substance normal Valves and muscle normal. Some epicardial fat. Lungs had tubercular foci and a cavity.	C First 5 weeks
22. JG 32 F	Delusional mania	-	Phthisis Pneumonale	Much emaciated	Vessels at base of brain were atheromatous otherwise NAD. Heart valves normal.	D ?

					Pericardium thickened. Lungs:- tubercular mass and cavity formation with consolidation and pleural thickening.	
23. RB 52 F	Delusional mania	Farm servant's wife	Valvular disease of heart	Well nourished	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.	D Second Few days
24. JMcA 44 M	ME	-	Phthisis Pneumonitis	Fairly well nourished	Pia etc. normal Brain substance normal	C ?

					Valves normal. Hypertrophy of L ventricle. Lungs had tubercular foci and a cavity.	
25. RB 39	MA with delusions	-	Phthisis and pericarditis. Miliary TB	Well nourished	Pia etc. thickened. Otherwise, NAD Pericardium thickened++ fibrous nodules in heart "bread and butter appearance". Hypertrophy of L ventricle. Aorta was diffusely atheromatous. Lungs:- tubercular cavity formation with caseation and pleural thickening with miliary nodules.	E ?
26. AN 32 M	MA	-	Intestinal obstruction	Abdomen distended	Brain - NAD Hypertrophy of L ventricle Valves were competent. Volvulus of colon	E ?
27. HI 54 F	MA	Knitter and crofter	Pneumonia	-	Congestion lateral sinuses. Valves normal. Heart hypertrophied and dilated.	D ?

					Muscles flabby and soft. Lungs consolidated with some adherence.	
28. WW 37 F	ME	None	Exhaustion from melancholia	Very emaciated Bedsore	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".	C First 3 months
29. SD 51 F	MA	-	Advanced TB	Very much emaciated	Minimal examination, "body needed for burial". Caseation in lungs.	D ?
30. GC 41 F	MA	-	Pneumonia	Well nourished Bruises over legs and arms "size of 5/ piece	Brain showed "slight congestion". Valves normal. Heart normal "to all appearances" No atheroma. Lungs consolidated. No sign of TB.	A ?
31. MM 54 F	MA	-	Pneumonia	Rather emaciated	Congestion of brain and pia etc. and some softening. Heart was flabby and L ventricle	D ?

					hypertrophied. Epicardial and pericardial fat Mitral valve calcified and all valves showed "atheroma" Serosanguinous fluid in lungs and much congested and consolidated.	
32. MB 43 F	ME with stupor		Phthisis Pneumonialis	Emaciated	Brain- NAD Pleural adherent. TB nodules in both lungs++. R lung riddled with cavities. Pericardial fluid. Fatty infiltration of heart.	E ?
33. DS 53 F	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. Aortic valve - NAD	C ?
34. AC 43 M	ME	Farmer	Exhaustion from melancholia/gangrene of lungs	Emaciated	Convolutions normal and well developed. On section puncta are more numerous	C First Some months



					<p>and conspicuous than normal. Basal arteries NAD.</p> <p>Valves normal.</p> <p>Atheromatous patches of aorta and aortic valve. Cusps of aortic valve thickened.</p> <p>Myocardium pale.</p> <p>Lungs congested and oedematous with “gangrene” at bases</p>	
35. JR 45 F	MA	Knitter	Pneumonia	Well nourished	<p>Brain - NAD</p> <p>Heart was flabby and L ventricle hypertrophied.</p> <p>Epicardial and pericardial fat</p> <p>Mitral valve thickened and aortic valve and aorta showed “commencing atheroma”.</p> <p>R lung shows congestion and pneumonia.</p>	A 5 <sup>th</sup>
36. JY 50 M	MA	-	TB and Cerebral softening	Emaciated. Several scars on the back	Generalised diffuse softening of brain particularly of white matter in	D ?

					<p>corpus callosum</p> <p>Pleural adherent. TB nodules in both lungs++ with cavities. Bronchial glands are enlarged and caseous. Gelatinous infiltration of heart and L ventricle hypertrophied.</p>	
37. SW 53 F	MA	-	Phthisis and cardiac disease	Emaciated.	<p>Brain NAD</p> <p>TB nodules in both lungs++ with cavities ranging from size of pea to that of walnut and numerous caseous nodules. Heart is atrophied, pale and wasted.</p>	C ?
38. JC 48 F	MA	Housewife	Congestion of brain	Thin	<p>Brain convoluted normally.</p> <p>Grey and white matter "pinkish"</p> <p>Otherwise, NAD</p> <p>Thorax etc - not examined</p>	B First 1 week
39. WD 24 F	MA	-	Phthisis	Emaciated. The penis has been amputated.	<p>Brain - NAD</p> <p>TB nodules in both lungs++ with cavities and nodules.</p> <p>Heart NAD</p>	D ?
40. ID	MA	-	Acute TB	Emaciated.	Brain - NAD	D ?

34 F					TB nodules in both lungs++ with cavities and nodules. Aortic and mitral valve incompetent. Milk spot right ventricle.	
41. JM 52 M	MA	-	Phthisis	Emaciated with bedsores	Brain substance pale. TB nodules in both lungs++ with cavities. Cardiac muscle pale and flabby with gelatinous degeneration. Aortic incompetence	C ?
42. CS 37 F	MA	Domestic servant.	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.	B First attack 1 week
43. JG 42 M	MA	-	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	C ?

					state of “acute yellow softening” Heart dilated and hypertrophied. Aortic valve incompetent. Calcification round coronary arteries Mitral valve stenosed and thickened.	
44. EA 37 F	ME	-	Phthisis	Emaciated	Brain - NAD TB nodules in both lungs++ with cavities and nodules. Myocardium is pale and flabby.	E ?
45. MT 44 M	MA	-	Exhaustion Labelled TB	Emaciated. Sinuses leading to bare bone on tibia Femur and toe. Several abscesses.	Brain substance pale. Nodular masses scattered throughout the lungs of caseous material. Aortic valve is incompetent and there is commencing atheroma of aorta.	E ?
46. JS 25 M	ME	-	TB peritonitis Phthisis	Much Emaciated	Brain and grey matter thin and pale. Heart is anaemic and wasted. Valves OK. TB nodules and caseation.	D ?

					Abdomen filled with purulent fluid. Intestines covered with TB nodules. Liver enlarged with waxy degeneration	
47. MG 46 F	Acute Mania (puerperal)	-	Pneumonia	Well nourished	Brain - NAD Fibrous deposit on epicardium and R ventricle. Aortic valve incompetent – rest NAD Lung congested with hepatisation. Frothy exudate	C ? 2nd attack
48. MM 55 F	ME	Housewife	Congestion of lungs	Emaciated	Brain - NAD Valves are incompetent. There is atheroma of aorta. Congestion of lungs	D First 6 weeks
49. BD 45 F	MA		Exhaustion from colostomy	Emaciated. Colostomy L groin	Brain substance pale. Large white patch on R ventricle, otherwise NAD. Large abscess in abdomen. Contiguous with pelvis and acetabulum, which has tubercular disease.	A ?

					Liver enlarged with waxy and fatty disease.	
50. AG 54 F	ME	-	Cerebral haemorrhage	Well nourished	<p>“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</p> <p>Lungs emphysematous but otherwise NAD.</p> <p>Aortic valve is competent but has patches of sclerosis at coronary arteries.</p> <p>Mitral valve is sclerosed and thickened and there are calcareous patches on it.</p>	E 2 <sup>nd</sup> 4 days
51. JH	MA	-	Cerebral apoplexy	Well nourished	<p>Large extravasation from pons through corpus striatum and optic thalamus.</p> <p>Acute softening of around haemorrhage</p>	D ?

					<p>and in corpus callosum.</p> <p>Arteries at base are atheromatous.</p> <p>Heart hypertrophied.</p> <p>Valves competent.</p> <p>Numerous patches of atheroma on aorta above valve. Small patch of atheroma on mitral valve.</p> <p>Lungs congested.</p>	
52. DS 46 M	MA	-	Intestinal obstruction from volvulus	Somewhat emaciated	<p>Brain "somewhat shrunken" with deep sulci.</p> <p>Valves are competent. There is atheroma of aorta.</p> <p>Some congestion of lungs</p>	E ?
53. AMcK 43 F	MA	-	Ca breast	<p>Greatly emaciated.</p> <p>Cancerous nodules all over trunk and head.</p> <p>Large Ca ulcer over scar of amputated R breast</p>	<p>Brain - NAD</p> <p>Heart - NAD.</p> <p>Nodes in lung, which are collapsed.</p> <p>Purulent lymph.</p>	E ?
54. BC 29 M	ME	-	Phthisis	Much emaciated.	Brain anaemic but no atrophy	D ?

					Lungs infiltrated with TB nodules. Heart normal if anaemic.	
55. JB 54 M	ME	-	Phthisis	Greatly emaciated	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.	D
56. Alexander Bateman 53 M	ME	-	Pneumonia	Poorly developed adult.	Excess fluid in subarachnoid space. Grey matter atrophied. Oedema round ventricles. Atheroma of basal arteries. Signs of pericarditis. Valves are competent and myocardium normal. There is no atheroma of aorta. Congestion of lungs with red and grey hepatisation.	E ?
57. AR 37	MA		Phthisis	"III" developed adult.	Grey matter anaemic and thin.	E ?



F					Signs of pericarditis. Valves are competent and myocardium normal. There is no atheroma of aorta. Lungs infiltrated with TB nodules and cavities.	
58. DB 21 F	MA	-	Phthisis	Much emaciated	Brain shows atrophy with softening Lungs infiltrated with TB caseous nodules. Heart normal if anaemic. Cardiac muscle pale and flabby. Thickening of mitral cusps.	D ?
59. MM 26 M	ME	-	Phthisis	Some bedsores	Brain shows atrophy with softening R lung infiltrated with TB nodules and cavities. Signs of pericarditis. Cardiac muscle NAD.	E ?
60. AF 35 M	ME	-	Phthisis	Much emaciated	Brain pale and anaemic with softening. Cardiac muscle pale and flabby There is atheroma of aorta.	C ?

					R lung infiltrated with TB nodules and pus-filled cavities	
61. JS 53 F	ME	-	Rupture of bowel	"Ill" constructed adult.	Considerable atrophy of L frontal lobe. Lateral ventricles enlarged++ No softening. Signs of pericardial fluid. L ventricle hypertrophied. Thickening of mitral cusps. Slight congestion of lungs. Liver congested. Massive distension of large bowel. Three inches of gangrenous bowel with a rupture. Evidence of scybala.	C ?
62. SS 50 F	MA	-	Pneumonia	Much emaciated.	Brain- Not examined ("examination conducted in haste") Heart NAD. Fracture of rib with evidence of pleurisy. Evidence of consolidation at base.	A ?
63. JR 53 M	ME	-	Exhaustion from melancholia and Phthisis Pneumonale	Very much emaciated.	Grey matter pale and anaemic, soft and friable.	D ?

					Signs of pericardial fluid. Cardiac muscle pale and flabby with mucoid degeneration. R lung infiltrated with TB nodules and cavities. Little functional lung.	
64. WG 48 M	Acute delirious mania	-	Pneumonia	Well developed	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	A ?
65. CY 49 F	MA	Domestic servant	Cardiac Disease	Very oedematous	Brain substance was very oedematous. Ventricles enlarged. Otherwise, NAD.	C Single. Second attack. Sudden onset... a

					<p>Aortic valve was competent but showed atheroma.</p> <p>Tricuspid valve dilated.</p> <p>Myocardium was slightly fatty.</p> <p>Lungs were very oedematous and showed <b>old</b> TB (clinical notes said phthisis at one point)</p>	day or two.
66. PG 39 M	MA	-	Pneumonia	Well developed	<p>Not examined - "Friends waiting to take him".</p> <p>Left ventricle dilated and L ventricle myocardium thinned.</p> <p>Valves NAD</p> <p>Pleura adherent.</p> <p>Both lungs congested.</p>	B ?
67. DMcL 30 M	?	Garden labourer	Cut throat	<p>Well developed.</p> <p>Large wound from near mastoid to midline severing muscles, jugular vein and I carotid artery; also, a second wound through first</p>	<p>Brain - NAD</p> <p>Heart - NAD</p> <p>Lungs – old TB</p>	C 2nd attack. 5 weeks.

68. JN 50 F	Mania History of alcohol	Crofter	Congestion of lungs	Not very much emaciated	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	C First attack of about 6 months
69. MI	MA	Agricultural labourer	Cardiac Disease	Very stout	Brain NAD Pleural effusions. Lungs congested. L and R ventricular hypertrophy. All valves incompetent. Liver- acutely congested. Ascites.	D Single. First attack
70. James Jack 35 M	MA	Tool finisher	Phthisis Pneumonale	Emaciated somewhat	Brain - NAD. Heart NAD Lungs TB cavities ++	D ?
71. JE 45 M	Delusional melancholia	Ploughman	Phthisis Pneumonale	Very much emaciated	Brain - NAD Heart dilated and walls thin. L ventricle hypertrophied	D ?

					Lungs – collapsed, caseation material. Abscesses and pus.	
72. GB 43 M	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.

Number Initials Age at Death Sex	Clinical diagnosis on admission	Occupation	Cause of death	External post- mortem findings	Internal post-mortem findings	Length of stay group Number and length of Attack
1. SP 40 F	MA	Housewife	Morbis Cordis	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	E First
2. HB 36 M	ME	Labourer	Phthisis	Extremely emaciated	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.	B ?
3. ML 22 F	MA	Dressmaker	PP	Very much emaciated	Brain - Congested and "hyperaemic" but NAD Heart small and pale Pericardial fluid++.	D ?

					Suppurating TB foci in both lungs	
4. SB 42 F	MA	Housewife	Phthisis	Very much emaciated	Brain - NAD but vessels atheromatous. Lumbar spine had mucous -purulent mass Heart normal Lungs show phthisis and cavities	D ?
5. MP 34 F	MA	Cook	Phthisis	Very much emaciated	Brain softened but otherwise NAD Valves normal. Myocardium flabby with some atheroma. Lungs showed signs of TB.	D ?
6. MW 52 F	MA	None	Ca rectum	Emaciated	Brain anaemic, white matter oedematous Heart was small size and myocardium soft and friable. Valves normal. Lungs congested and secondary Ca in one lung.	E ?
7. TT 52 M	ME	Agricultural Labourer	Advanced TB	Very much emaciated	Brain shrunken and anaemic, white matter oedematous Pericardial thickening and fluid++. Heart pale and friable. Valves dilated. Patches of atheroma. TB masses and cavities	E ?
8. MD 53 F	MA	None	Heart disease	Very much emaciated	Grey and white matter congested Myocardium soft and friable. Valves OK.	C First 6 weeks



					Atheroma of vessels. Pleural adhesions and congestion of lungs.	
9. FG 42 M	ME	Antique dealer	Pneumonia	Well nourished	Brain soft and hyperaemic. White matter oedematous. Atheromatous patches on basal vessels. Valves somewhat dilated. Pericardial blood. Myocardium soft and friable. Atheroma of some vessels R Lung congested with "red hepatisation"	D ?
10. HH 26 M	MA	Soldier	Cardiac failure	Well nourished	Congestion of brain and grey and white matter dark and congested. Mitral valve somewhat dilated. Some pericardial fluid. Pleural effusion. R lung collapsed.	D First 3.3 years.
11. FH 51 M	MA	Labourer	Exhaustion	Much emaciated. Bruise over l iliac region.	Brain soft. Both grey and white matter congested and oedematous. Atheromatous patches on basal vessels Heart large. Hypertrophy of l ventricle but also pale and friable. Mitral valve somewhat dilated. Congestion of both lungs.	A ?

12. MK 42 F	MA	School Mistress	Heart Disease 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.	E First (3days)
13. SH 46 F	MA	Housewife	Phthisis	Emaciated	Brain pale. White matter oedematous Pericardium thickened. Valves normal. Myocardium soft and friable. Lungs consolidated with signs of TB foci.	E ?
14. JS 23 M	MA	None	Phthisis	Rather emaciated	Brain pale. White matter oedematous Pericardium thickened and contained fluid. Valves normal. Myocardium pale and flabby. Lungs consolidated with cavities.	C ?
15. HR 48 M	MA	Labourer	Phthisis	Very much emaciated	Brain pale. White matter oedematous NAD. Basal vessels show atheroma. Heart small and flabby. Lungs show TB tubercle and cavities".	D ?
16. EP 40 F	MA	Charwoman	Phthisis	Emaciated	Brain pale. Myocardium flabby and degenerated. Vessels show atheroma	D ?

17. TB 48 M	MA	Labourer	Exhaustion	Extremely Emaciated	Meninges engorged. White matter oedematous Valves slightly dilated. R ventricle thin. Muscle pale and friable. Lungs congested.	A First (1 week)
18. BB 32 F	MA	None	Phthisis	Rather emaciated	Grey matter pale. White matter oedematous Heart was flabby and degenerated. Both lungs much congested and consolidated with cavities and TB deposits.	E ?
19. GB 36 M	ME	Fitter	Phthisis	Very much emaciated	Grey matter soft. White matter oedematous Heart small. Some pericardial fluid. Lungs show apical consolidation and gangrene in parts.	C ?
20. MK 34 F	MA	Domestic Servant	Cardiac failure in recovery from typhoid	Emaciated	Not examined Flaccid heart. Lungs not congested. Large bowel ulcers healing	? ?
21. EW 51 F	MA	Housewife	Phthisis	Emaciated	Heart was flabby and degenerated. Lungs congested with purulent cavities.	E ?
22. RH 55 F	MA	?	Cerebral softening	Emaciated	Grey matter pale. White matter oedematous. Atheroma of basal arteries. Myocardium shows fatty degeneration. Valves normal.	E ?

23. WF 40 M	MA	Labourer	Phthisis	Very much emaciated	Grey matter pale. White matter "sodden, pale and oedematous" Heart small, pale and flabby. Some pericardial fluid. Valves somewhat dilated. Lungs -adherent with purulent cavities.	D ?
24. EW 49 M	MA	Labourer	Pyelonephritis	Emaciated	NAD brain Heart enlarged. Valves dilated. Ventricle walls much hypertrophied Some congestion lower lobe. Passive congestion of liver. Large renal abscess.	A ?
25. JS 55 M	ME	Butcher	Ca bladder	Extremely Emaciated	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 NAD other than Ca mass in bladder	D ?
26. AH 55 F	MA	None (Widow)	Exhaustion	Poorly nourished	Grey matter hyperaemic. White matter "sodden and oedematous" with "punctate vasculosa". Atheroma of basal arteries.	A First (some days)

					Some pericardial pus. Myocardium shows soft and friable. Atheroma of arteries. Valves normal. Lungs are coated with purulent lymph. Some congestion at bases.	
27. LS 47 F	MA	Laundress	Exhaustion	Poorly nourished	Grey and white matter hyperaemic. Myocardium and pericardium normal. Some atheroma of valvular vessels. Valves normal. Some congestion at bases.	A First (2 weeks)
28. SG 53 F	MA	None (Widow)	Cardiac failure	Rather emaciated	Grey matter pale. White matter oedematous Some pericardial fluid. L Heart contracted. R ventricle is thin and myocardium shows fatty degeneration. ? mitral valve dilated, rest NAD. Lungs emphysematous.	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 ?
30. HD 55 F	ME	Labourer	Peritonitis	Fairly nourished	Grey matter less firm than normal. White matter somewhat oedematous.	A ?

					<p>Atheroma of basal arteries. Some pericardial pus.</p> <p>Myocardium is soft and rather degenerated.</p> <p>Atheroma of valvular arteries.</p> <p>Valves normal.</p> <p>Some emphysema and congestion at bases.</p> <p>Pus in pelvis and throughout on lymph nodes. No obvious perforation but sloughing ulcer in rectum.</p>	
31. EB 53 F	Recurrent MA	None	Volvulus of colon.	Well nourished	<p>Grey matter slightly hyperaemic</p> <p>Myocardium and pericardium normal.</p> <p>Valves normal.</p> <p>Some congestion at base.</p> <p>Large volvulus of sigmoid colon with some evidence of peritonitis.</p>	D ?
32. JS 33 F	Delusional insanity	Cook	Phthisis	Much emaciated	<p>Grey matter and white matter pale.</p> <p>Some pericardial fluid otherwise NAD.</p> <p>TB cavities filled with pus.</p>	D ?
33. AP 47 F	Recurrent Mania	None	Exhaustion	Emaciated and bruises and bedsores	<p>Grey matter slightly hyperaemic. White matter hyperaemic with punctate vasculosa.</p> <p>Myocardium soft and degenerated.</p>	B Second (five weeks)

					Atheroma of valvular arteries. Some congestion at bases.	
34. HL 52 F	ME	Domestic Servant	Pneumonia	Well nourished	Gyri are wasted especially centrally. Myocardium is soft and rather degenerated. Valves normal. Some congestion at base with pus.	C ?
35. MR 48 F	Delusional insanity	Housewife	Phthisis	Emaciated	Grey and white matter anaemic Myocardium is soft and rather degenerated. Lungs:- adherent and congested	D ?
36 MH 32 F	MA	None	Phthisis	Emaciated	Grey matter and white matter pale. Some pericardial fluid otherwise NAD. TB cavities filled with pus.	C ?
37. SAK 41 F	MA	None	Heart Disease	Fairly nourished	Grey matter slightly hyperaemic. White matter normal Myocardium soft and degenerated. Atheroma of valvular arteries and first part of aorta. Lungs NAD	C First (6 days)
38. EG 24 F	MA	None	Enteric fever	Well nourished	Grey matter pale. White matter soft and slightly oedematous Heart and lungs NAD. R iliac fossa filled with pus and bowel dilated	D ?

39. JS 55 M	ME	Labourer	Pneumonia/Morbis Cordis	Emaciated	Brain soft and sulci gaping. Grey matter average thickness but pale. White matter pale. Atheroma of basal arteries. L Heart is dilated and hypertrophied. Mitral valve shrunken. Lungs:- adherent. R and L lung show grey and red hepatisation	C ?
40. WS 35 M	MA	Labourer	Acute TB	Emaciated	Grey matter and white matter pale Some pericardial fluid. Myocardium pale and anaemic Numerous active tubercles and TB cavities.	C ?
41. EW 38 F	MA	Nursery Governess	Enteric fever	Fairly nourished	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.	D ?
42. ES 51 F	ME	Housewife	Cerebral softening	Fairly well nourished	Brain is shrunken. Gyri are wasted, sulci gaping. Grey matter pale. White matter is sodden and slightly oedematous.	D ?



					Atheroma of basal arteries. Myocardium fatty and degenerated. Atheroma of mitral and aortic arteries	
43. MW 49 F	MA	Housewife	Pneumonia	Fairly nourished	Brain soft and gyri are wasted. Grey and white matter hyperaemic. Pericardium, myocardium and valves all normal. R lung show red hepatisation.	B ?
44. HH 44 F	ME	None	Broncho-Pneumonia	Emaciated	Brain soft. Gyri are wasted, sulci gaping. Grey matter pale. White matter is slightly oedematous Heart large. Hypertrophy of ventricles. Dilated ventricles. Lung consolidated with exudates.	C ?
45. HW	Recurrent Mania	Prostitute	Ca uterus	Rather Emaciated	Brain soft and gyri are wasted. White matter is slightly oedematous Heart large. Hypertrophy of L ventricle. Dilated ventricles. Incompetent mitral valve. Lungs collapsed and oedematous. Liver-nodules of amyloid degeneration. Ca uterus infiltrating walls of uterus. ?scirrhous in nature	C ?

46. FC 25 F	ME	Domestic Servant	Phthisis	Rather Emaciated	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.	D ?
47. AM 47 F	Recurrent Mania	None	Dysentery	Emaciated	Pericardium, myocardium and valves all normal. R lung show red hepatisation.	E ?
48. OL 43 F	MA	Dressmaker	Morbis Cordis	Well nourished	Brain soft. Gyri are wasted. Grey matter pale. White matter is slightly oedematous Atheroma of basal arteries Pericardium contains fluid. Hypertrophy of L ventricle. Dilated ventricles. Incompetent mitral and tricuspid valves. Some oedema of lungs.	D Second (1 month)
49. FH 54 F	ME	Housewife	Exhaustion	Rather Emaciated	Brain soft and gyri are wasted. Sulci gaping. Rest hard to read. Heart small. Pericardium contains fluid. Hypertrophy of L ventricle. Myocardium somewhat degenerated.	C First (5 weeks)

					Valve and cavities normal. Lungs congested and oedematous. Some emphysema.	
50. AL 47 F	ME	Dressmaker	Cerebral softening	Emaciated	Brain soft and gyri are wasted. Sulci gaping. Grey matter pale. White matter is sodden and oedematous. Hypertrophy of L ventricle. Valves and cavities NAD. Slight congestion of lungs.	D ?
51. AG 40 F	MA	Domestic Servant	Cerebral softening	Emaciated	Brain soft and gyri are wasted. Sulci gaping. Grey matter pale. White matter is sodden and oedematous Hypertrophy of L ventricle. Valves and cavities NAD. Slight congestion of lungs.	E ?
52. SW 38 F	ME	Domestic Servant	Phthisis	Well nourished	Grey matter and white matter pale. Pericardium, myocardium and valves all normal. Numerous tubercular foci and cavities with pus.	D ?
53. CW 53 F	MA	Housewife	Phthisis	Emaciated	Grey matter and white matter extremely pale Pericardium and valves all normal. Myocardium soft and friable. Numerous tubercular	D ?

					nodules and cavities and pleural involvement	
54. MAI 44 F	ME	None (workhouse)	Exhaustion	Emaciated	Brain enlarged. Gyri are flattened. Sulci gaping. Grey matter congested. White matter is oedematous. Ventricles are dilated "hydrocephalus". No focal lesion. Myocardium soft. Valves and cavities NAD. Slight congestion of lungs.	B First (4 days)
55. TH 40 M	ME	None	Phthisis	Emaciated	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	E ?
56. JR 40 M	MA	Bricklayer	Exhaustion	Well nourished	Convolutions well marked but show some wasting. Grey matter is congested. White matter is congested with punctate vasculosa well-marked. Atheroma of basal arteries.	A First (1 week)

					Atheroma of mitral and aortic valves. First part of aorta atheromatous. Myocardium pale and friable. Lungs acutely congested.	
57. JD 54 M	MA	Groom	Exhaustion	Emaciated	Brain soft and gyri are wasted. Grey matter hyperaemic. White matter is sodden and oedematous. Atheroma of basal arteries. Pericardium normal. Herat dilated. Myocardium soft. Mitral valve dilated. Atheroma of mitral and aortic valves. First part of aorta atheromatous. R lung shows middle lobe hepatisation. L base congested.	B Second (3 months)
58. EE 39 F	MA	Domestic Servant	Phthisis	Emaciated	Grey and white matter pale. White matter is oedematous. Myocardium soft. Valves and cavities NAD. Adherent lungs. Tubercular foci and cavities++	E ?
59. MM 26 F	MA	None	Acute pulmonary TB	Emaciated	Grey and white matter pale. Pericardial sac has fluid. Myocardium anaemic soft and friable. All ventricles dilated.	D ?

					Tubercular deposits and cavities++ (Size of grapes)	
60. MH 40 F	ME	Housekeeper	Morbis Cordis	Emaciated	Grey and white matter hyperaemic. Pericardium adherent. Myocardium soft flabby and degenerated. Atheroma of mitral and aortic valves. Lungs congested and "engorged with blood".	D First (6 weeks)
61. EH 38 F	ME	Housewife	Dysentery	Emaciated	Grey and white matter hyperaemic. Pericardium normal. Myocardium soft otherwise NAD Lungs NAD Whole of ileocecal area and large bowel covered with sloughing ulcers	B ?
62. LH 50 F	ME	Seamstress	Exhaustion	Emaciated	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.	C First (6 weeks)
63. EH 50 M	MA	Clerk	Exhaustion from acute mania/ Morbis Cordis	Emaciated	Gyri are shrunken. Grey matter thin, soft and congested. White matter soft and	D First (3 months)

					<p>congested and oedematous.</p> <p>Atheroma of basal arteries.</p> <p>Atheroma of mitral and aortic valves and are shrunken.</p> <p>Myocardium hypertrophied.</p> <p>Lungs emphysematous and congested at bases.</p>	
64. RW 52 F	Recurrent mania	Housewife	Pneumonia	Fairly nourished	<p>Brain soft and gyri are wasted. Sulci gaping.</p> <p>Grey and white matter pale.</p> <p>Pericardium adherent.</p> <p>Myocardium fatty and degenerated otherwise NAD.</p> <p>Atheroma of mitral and aortic valves.</p> <p>R lung shows red hepatisation.</p> <p>Emphysema of L lung with congestion at base.</p>	D ?
65. FMD 33 F	MA	Dressmaker	Suffocation from impacted bread in larynx.	Well Nourished. Bread the size of a "cob-nut" found in trachea above vocal cords.	Normal.	C ?
66. KB 28 F	MA	Domestic Servant	Pneumonia	Well Nourished.	<p>Brain shape and consistency normal. Gyri OK. Grey matter hyperaemic.</p> <p>Pericardium normal.</p> <p>Myocardium soft and degenerated. Valves and cavities NAD.</p>	B ?

					Red hepatisation in R lung	
67. SB 45 F	ME	Housewife	Phthisis	Emaciated. Bruise on the trochanter.	Grey and white matter hyperaemic Pericardium normal. Myocardium soft and degenerated. Valves and cavities NAD. Both lungs had TB foci and cavities.	
68. AY	MA	Housewife	Syncope (labelled as choking)	Emaciated. Signs of previous 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.	D ?
69. SM 54 F	ME	None	Cardiac disease	Well Nourished.	Brain soft and gyri are wasted. Sulci gaping. Grey matter pale. White matter sodden and oedematous. Atheroma of basal arteries. Myocardium soft flabby and degenerated. Atheroma of aortic and mitral valves. Chambers dilated. Some consolidation of L lower lobe.	C First (3 months)
70. EB 36 F	MA	Tailoress	Exhaustion	Emaciated.	Brain shape and consistency normal. Grey and white matter extremely congested	C First (1 month)



					Pericardium, myocardium, valves and cavities NAD.  R Lung shows acute congestion, less at base of L lung.	
71. MW 55 F	MA	Nurse	Colitis	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.	E ?
72. HL 39 F	MA	None	Colitis	Emaciated.	Grey and white matter hyperaemic otherwise NAD  Pericardium, , valves and cavities NAD  Myocardium somewhat softened.  Lungs NAD  Liver slight congestion. Large bowel shows sloughing ulcers. Pelvis filled with pus.  Haemorrhagic mucosa.  Lymphoid infiltration.  Mesenteric glands enlarged and soft.	D ?

73. EP 52 F	MA	Charwoman	Dysentery	Fairly Emaciated.	Brain and gyri are wasted and shrunken. Sulci gaping. Grey matter pale. White matter sodden and oedematous. Atheroma of basal arteries. Myocardium soft flabby and degenerated. Atheroma of aortic and mitral valves. Lungs emphysematous with some congestion at bases. Small bowel thickened. Caecal area has petechial haemorrhages and ulcers.	D ?
74. JP 30 M	MA	Hotel worker	Phthisis	Emaciated.	Brain soft. Grey matter congested. White matter soft and oedematous. Pericardium thick. Myocardium soft and some signs of fatty degeneration. Lungs show cavities and tubercular deposits	D ?
75. OM 42 M	MA	Sadler	Cerebral softening	-	Gyri are wasted. Brain soft. Grey matter anaemic and thin. White matter soft and oedematous. Atheroma of basal arteries. Myocardium soft friable. Atheroma of aortic and mitral valves.	D ?

					Lungs - old calcareous? at apex with fibrous change.	
76. MH 47 F	MA	None	Phthisis	Emaciated	Grey and white matter pale. Myocardium soft flabby and degenerated. Tubercular consolidation and cavities++	D ?
77. FH 44 F	ME	Housewife	Pneumonia	Fairly Emaciated	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.	B ?
78. KL 49 F	MA	Housekeeper	Phthisis	Emaciated	Grey matter anaemic otherwise NAD Pericardium normal. Myocardium soft and flabby. Valves and chambers normal. Lungs show cavities and tubercular foci.	E ?
79. FF 55 F	ME	Housewife	Pneumonia	Emaciated	Grey matter on R side much congested. L NAD. Pericardial fluid. Myocardium soft flabby and degenerated. Valves normal. Ventricles dilated. R lung show red hepatisation and pleural adhesions.	D ?

80 RG 22 M	MA	Farmer's son	Phthisis	Fairly Emaciated	Grey matter anaemic and white matter pale. Pericardial fluid ++ otherwise NAD. Tubercular consolidation and cavities++	C ?
81. EE 44 F	MA	Domestic servant	TB	Fairly Emaciated	Grey matter anaemic. White matter oedematous. Myocardium normal. Valves and chambers normal. Both lungs show cavities and tubercular foci.	E ?
82. AB 53 F	ME	Chapel caretaker	Empyema (labelled as pneumonia)	Emaciated.	Brain soft and gyri are wasted. Sulci gaping. Grey matter pale. White matter sodden and oedematous. Pericardial fluid ++ Myocardium normal. Atheroma of mitral valve. Other valves and chambers normal. R lung show red hepatisation. R pleural space contains several ounces of fetid pus.	D ?
83. MS \$\$ F	MA	Domestic Servant	Fibro- sarcoma of uterus. Septicaemia	Fairly nourished. Large abdominal wound (?surgical) with strangulation??	Brain soft and gyri are wasted. Sulci gaping. Grey matter pale. White matter sodden and oedematous. Heart NAD Lungs emphysematous with pleural collapse.	E ?

					Fetid pus in abdominal cavity. Large tumour from stump of uterus	
84. EP	ME	Charwoman	Cardiac failure Subdural haemorrhage	Rather Emaciated	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.	B ?
85. GH 27 F	MA	School teacher	Cerebral haemorrhage	Fairly nourished Bruises R arm and scalp. No skull fracture or "signs of violence"	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	A ?

					marked hypertrophy otherwise NAD.	
86. MB 54 F	MA	Domestic Servant	Cerebral haemorrhage and softening	Emaciated.	Haemorrhage in meningeal space of middle fossa of several days' duration. Brain soft and gyri are wasted and pale. Sulci gaping. White matter sodden and oedematous. Atheroma of basal arteries. L ventricle shows marked hypertrophy Atheroma of aortic valve. Other valves and chambers normal. Lungs emphysematous. Healed TB foci.	D First 2 weeks
87. AS 34 M	MA	Market trader	Enteric fever.	Fairly nourished	Sulci gaping. Otherwise, NAD. L ventricle shows hypertrophy Mitral valve soft and friable. Lungs show congestion at bases.	E ?
88. MW 53 F	Delusional insanity	Housewife	Fatty heart and bronchitis.	Well nourished.	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.	E First 12 days

					Some basal pleural adhesions otherwise NAD.	
89. AM 24 F	MA	None	Meningitis and pneumonia (labelled as such)	Emaciated. Ulcers on legs and jaw.	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.	C ?
90. SS 44 M	MA	Farm labourer	Pneumonia	Fairly nourished with no bruises or signs of violence.	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.	D ?
91. SAS 40 F	ME	Housewife	Pneumonia	Fairly nourished with no bruises or signs of violence	Grey and white matter congested with vessels markedly engorged. Basal vessels healthy. Heart NAD. Lungs show red hepatisation.	B ?
92. AB 37 F	ME with stupor	None	Pneumonia	Fairly nourished with no bruises or signs of violence	Brain soft. Gyri OK. Grey and white matter congested. Basal vessels healthy. Pericardium normal. Myocardium soft and flabby. All chambers	A ?

					dilated. R lung show red hepatinisation.	
93. ET 44 F	ME and stupor	Domestic Servant	Ulcerative colitis	Fairly nourished with no bruises or signs of violence.	“The head was not examined at the request of friends”  Myocardium soft and flabby. All valves and chambers normal.  Congestion of ileocecal valve and widespread ulcers in large bowel with punctiform haemorrhage	C ?
94. KK	MA	Governess	Cerebral softening and heart disease (labelled as latter)	Somewhat emaciated	Brain soft and gyri are wasted. Sulci gaping.  Grey matter wasted and white matter oedematous.  Atheroma of basal arteries.  Pericardium thickened.  Myocardium soft and flabby. Aortic valve and aorta are calcareous.  Lungs congested at bases. Healed TB foci.	E Single 14 months First
95. AA 31 F	MA	Governess	Phthisis	Somewhat emaciated.  Heart NAD. Lungs show numerous tubercular foci and cavities.	Brain soft. Both grey and white matters are pale and anaemic.  No atheroma of basal arteries	E ?
96. RR 49 F	MA	Laundress	Fatty heart and cerebral softening. (labelled as latter)	Fairly nourished with no bruises or signs of violence.	Brain soft and gyri especially parietal are wasted. Sulci gaping.  Grey matter wasted and white matter oedematous.	D ?



					<p>Atheroma of basal arteries.</p> <p>Pericardium normal.</p> <p>Ventricles shows hypertrophy</p> <p>Valves healthy but chambers dilated. Lungs congested at bases.</p>	
<p>97.</p> <p>JC</p> <p>55</p> <p>F</p>	MA	Charwoman	Fatty heart	Obese with umbilical hernia	<p>Brain soft and gyri are wasted. Sulci gaping.</p> <p>Grey matter wasted and white matter oedematous. No atheroma of basal arteries.</p> <p>Myocardium soft and flabby. L ventricle shows hypertrophy. Aortic valve shows atheroma. Other valves and chambers normal. Lungs emphysematous and congested at bases.</p>	E ?
<p>98.</p> <p>CC</p> <p>45</p> <p>M</p>	MA	Labourer	Cardiac failure (morbus cordis)	Fairly nourished with no bruises or signs of violence	<p>Grey matter pale and white matter oedematous. Lateral ventricle enlarged with fluid.</p> <p>No atheroma of basal arteries.</p> <p>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</p>	E ?

					aorta is atheromatous. Other valves healthy. Lungs NAD.	
99. SW 43 F	ME	Housewife	Phthisis	Somewhat emaciated	Brain of normal consistency. Both grey and white matters are pale and anaemic. No atheroma of basal arteries. Heart NAD. Lungs show numerous tubercular foci and cavities.	E ?
100. HM 44 F	MA	Housewife	Exhaustion	Somewhat emaciated	Brain soft. Grey matter hyperaemic. White matter normal. No atheroma of basal arteries. Myocardium soft and degenerated. Ventricles and cavities normal. R Lung congested at base.	C 1 week 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.
9. Cause of death now changed to Bright’s disease. Evidence of kidney pathology at post-mortem.

## **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 Certificate 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 feed 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<sup>st</sup> 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.

Number Initials Age	Date of death	Date of admission Length of stay category.	Mental state	Cause of death	Main findings at post- mortem	Main clinical findings
1. GL 52	13th Oct 1895	20th Nov 1888 E	MA	Apoplexy	Well nourished—“very corpulent” Bedsore 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	-
2. RB 52	10th Dec 1897	13th April 1895 D	MA	Valvular disease of heart	Well nourished Pia opalescent Brain substance was	Married. Farm servant’s wife. Three children 1st attack, of a few days’ duration



					<p>congested and showed many vascular "markers".</p> <p>Vessels at base of brain were atheromatous</p> <p>Heart was hypertrophied and dilated. Aortic and pulmonary valves were incompetent.</p> <p>Aorta and coronary vessels were diffusely atheromatous.</p> <p>Hypertrophy and fatty degeneration of ventricular muscles.</p> <p>Lung congestion.</p> <p>Epicardial fat</p> <p>Liver congested</p>	<p>Not epileptic, suicidal or dangerous.</p> <p>Certificates. "quite changed"</p> <p>Thinks "someone has seen her and is suffering from those effects"</p> <p>Says she has seen an angel.</p> <p>Had a similar slight attack a few years ago but made an excellent recovery.</p> <p>Suddenly became restless and excited and violent. Deluged child with water under the impression it was a baptism</p> <p>Sister committed suicide and cousin was an inmate of Sunnyside.</p> <p>Bodily condition:-</p> <p>Indifferent. Pulse regular but 1st mitral sound abnormal.</p> <p>Described as being in state of profound melancholia with occasional impulsive violence. Restless delusional condition July 20th. Maniacal condition "shouts, preaches and cries".</p> <p>Continues in similar way. Develops a discharging sinus on her neck.</p> <p>Continues delusional and mis-identifies people.</p> <p>Suddenly had fit and died.</p>
3. DS 53	15th Feb 1892	18th Feb 1881 C	MA	Cardiac valvular disease	<p>Well nourished</p> <p>Very dropsied</p> <p>Softening in internal capsule Pleural fluid</p> <p>Both lungs Oedematous.</p> <p>L ventricle and other cavities hypertrophied.</p> <p>Mitral valve incompetent.</p> <p>Aorta NAD</p> <p>Liver enlarged...chronic venous congestion.</p>	-

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”

						<p>Physical:- Bodily condition. Indifferent</p> <p>CVS and other systems NAD</p> <p>Mental state:- Profound melancholia but has "dementia probably of a passing character"</p> <p>Wanders about, looks nervous and afraid.</p> <p>Refuses food, stomach tube passed.</p> <p>Not improved, quiet and seldom speaks.</p> <p>August:- outburst of motor excitement</p> <p>Labouring under marked delusion, she was fighting for her life.</p> <p>Gradually sinking, refuses food, and health precarious.</p> <p>Died July 1st following year</p>
6. AG 54	9th Aug 1893	5th Feb 1874 E	ME	Cerebral haemorrhage	<p>Well nourished.</p> <p>"Bullae" of fluid in R cortex near Sylvian fissure and parietal lobe.</p> <p>I L cortex there is attenuation and much softening. All arteries are thickened and calcareous. Diagram of lesions</p> <p>Lungs emphysematous but otherwise NAD.</p> <p>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.</p> <p>Liver normal but kidneys "much contracted".</p>	<p>Married. 2nd attack. Stated to be 4 days insane.</p> <p>Physical Examination. NAD apart from numerous petechial spots over body.</p> <p>Mentally:-Many delusions. Thinks house is full of men and wild animals.</p> <p>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</p> <p>Violent at times and said by mother to be unmanageable.</p> <p>Previous admission had been for melancholia when she had delusions of suspicion. Now much worse.</p> <p>Remained the same for month of February. Bursts in to hysterical fits of laughter now and then and also demands her release.</p> <p>Over next few years remains general OK but subject to outbursts of excitement with the same delusions as above. Often attacks other harmless</p>

						patients who she thinks are "souffing" her.
7. JH 53	23rd Oct 1893	19th Dec 1891 D	MA	Cerebral apoplexy	Well nourished. Large extravasation from pons through corpus striatum and optic thalamus. Acute softening of around haemorrhage and in corpus callosum. Arteries at base are atheromatous. Heart hypertrophied. Valves competent. Numerous atheromatous? on aorta above valve . Small patch of atheroma on mitral valve. Lungs congested. Liver congested	
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

					Liver-chronic venous engorgement. Ascites.	1899 Improved mentally but poorer physically. In heart failure with oedema of legs Died
9. JN 50	14th Feb 1894	23rd June 1893 C	MA	Congestion of lungs	Not very much emaciated 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 N Liver soft and friable	1st attack, not epileptic, not suicidal Described as having a dissolute intemperate life. Husband a sailor away a lot but recently back and running small croft. No children Attack started 6 months ago. Took to bed. Careless and untidy. No alcohol since then. No FH On admission Easily excited, Habits dirty Wanders around naked. Has imaginary ailments Physical Pulse 104 regular. Heart displaced. Murmur at tricuspid valve. Mental Agitated and excited. Memory poor Disorientated. No clear evidence of delusions or hallucinations. Semi-incoherent. Developed marked oedema. died
10. MI 52	20th Aug 1892	8th April 1891 D	MA	Cardiac Disease	Very stout Brain NAD Pleural effusions. Lungs congested. L and R ventricular hypertrophy. All valves incompetent Liver-acutely congested. Ascites.	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

						<p>More recently sleep less good.</p> <p>Mental state on admission</p> <p>Many delusions of suspicion. People around her have designs on her.</p> <p>Thought attendants meant to drown her. Continues restless and hypochondriacal.</p> <p>Developed two poisoned wounds on finger and legs.</p> <p>Health became feeble. Very breathless</p> <p>Dropsy rapidly increasing. Southeys tubes in abdomen to draw fluid.</p> <p>Growing weaker</p>
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**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.

No. Initials Age	Date of death	Date of admission Length of stay category	Ment al state	Cause of death	Main findings at post- mortem.	Main clinical findings
1. SP 40	8 <sup>th</sup> Nov 1895	29/7/1882 D	MA	Morbis. cordis	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.	Case notes missing Admission Register Cause : Heredity Health indifferent First attack of 4 weeks duration
2. MD 53	25th Aug 1896	C	MA	Bronchitis in register relabelled as heart disease after PM	Very much emaciated Grey and white matter congested Myocardium soft and friable. Valves OK. Atheroma of vessels. Pleural adhesions and congestion of lungs  Liver congested	Climacteric, bodily illness 2nd class, morbis cordis, feeble First attack of 6 weeks duration 52-year-old single lady of no occupation. First attack of about 6 weeks in duration. Can give little information. Episode of pneumonia about 6 weeks ago at onset. Climacteric. Not in workhouse. No FH

						<p>Feeble woman of anaemic complexion, many teeth missing.</p> <p>Body poorly nourished musculature poor.</p> <p>Pulse 100/min, poor. Mitral systolic murmur.</p> <p>Mental state.</p> <p>Restless and agitated.</p> <p>Much talk to herself, incoherent.</p> <p>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.</p> <p>Sleeps poorly but better with chloral.</p> <p>Food intake poor as she says she cannot afford it.</p> <p>Intermittently agitated requiring hyoscine. Multiple bruises and haematomas.</p> <p>Episode of diarrhoea with fluctuant temp.</p> <p>Remains resistive.</p> <p>Temperature begins to fluctuate.</p> <p>Sounds at bases</p> <p>Dies 25th Aug 1896</p>
3. MW 53	22 <sup>nd</sup> Feb 1905	1/12/1897. E	MA	Bronchitis and fatty heart	<p>Well nourished. Grey matter and white matter pale. Pericardium normal. Myocardium soft and flabby and shows fatty change. L ventricle shows hypertrophy</p> <p>Aortic valve shows atheroma. Other valves and chambers normal.</p> <p>Some basal pleural adhesions otherwise NAD.</p>	<p>Married , Reading</p> <p>Heredity and drink</p> <p>2<sup>nd</sup> Class, feeble</p> <p>12 days First</p> <p>Housewife.</p> <p>First attack of 12 days duration.</p> <p>Normally intelligent</p> <p>Second marriage 7 years ago, never lived happily with him. One miscarriage by first husband.</p> <p>Living comfortably. Intemperate and not taking food well.</p> <p>One brother was insane.</p>



					<p>Liver fatty change.</p> <p>Changes of old typhoid?</p>	<p>Climacteric.</p> <p>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.</p> <p>Refused food since. Excitable at times.</p> <p>Physical; well nourished. Pulse 76 small. 1st heart sound faint. Two very small bruises on forearms. No ataxia.</p> <p>Mental. Marked delusions. Memory and orientation poor.</p> <p>Thinks her husband wants to poison her so he can marry another woman. Continues to express delusions.</p> <p>Sleeps and eats quite well.</p> <p>Begins to take more unintelligibly. 1908.. much the same. Bodily health good.</p> <p>Feb Develops fluctuating fever.</p> <p>Sounds at R apex</p> <p>Given medication but sinks into coma and dies.</p>
4. KK 53	18 <sup>th</sup> Aug 1905	E	MA	Cerebral softening and heart disease (labelled as latter)	<p>Somewhat emaciated with no etc.</p> <p>Brain soft and gyri are wasted. Sulci gaping.</p> <p>Grey matter wasted and white matter oedematous.</p> <p>Atheroma of basal arteries.</p>	<p>Single, Farringdon. Transferred from Bethlem Hospital</p> <p>Heredity and climacteric</p> <p>2<sup>nd</sup> Class, thin and feeble</p> <p>14 months</p> <p>First</p> <p>Single governess. Causes are said to be heredity, shock and the climacteric. mother's death one year</p>

					<p>Pericardium is thickened. Myocardium soft and flabby. Aortic valve and aorta are calcareous. Lungs congested at bases. Healed TB foci. Abdo - NAD</p>	<p>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.</p>
5. HH 26	16 <sup>th</sup> Feb 1897	13 <sup>th</sup> April 1894 D	MA	Cardiac failure	<p>Well nourished Congestion of brain and grey and white matter dark and congested. Mitral valve somewhat dilated. Some pericardial fluid. Pleural effusion. R lung collapsed. Liver passive congestion</p>	<p>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</p>

						<p>strangle himself. Admitted to another Asylum initially.</p> <p>Physical.</p> <p>Palate rather vaulted. Body well nourished, musculature good. Pulse 72/min. Heart and lungs NAD.</p> <p>Intermittently abusive and aggressive. At other times dull and vacant though laughs loudly and raucously for no apparent reason.</p> <p>Works well. Sleep and health good.</p> <p>Begins to collect and hoard rubbish</p> <p>Said to be full of delusions. Tied a ligature round his penis "to benefit his eyesight".</p>
6. MK 41	29th March 1897	30th July 1881 E	MA	Heart disease and Addison's disease	<p>Much emaciated</p> <p>Brain soft. Both grey and white matter congested and oedematous.</p> <p>Some pericardial fluid.</p> <p>Mitral valve somewhat dilated. Myocardium soft.</p> <p>Atheroma of some vessels.</p> <p>Liver passive congestion and nodular.</p> <p>.</p>	<p>Single, Reading</p> <p>Unknown</p> <p>Health indifferent</p> <p>3 days</p> <p>First Single school mistress aged 25.</p> <p>Transferred from Shrewsbury Asylum where she has been resident since 1880.</p> <p>No FH</p> <p>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.</p> <p>Physically: - Indifferent health and reduced condition. Heart and lungs healthy.</p> <p>Mentally: - Peculiar manner and deportment. Subject to delusions and hallucinations of sight and sound. Voices like telephones appear</p>

						<p>to her at night and that the spirits of her mother and father appear to her at night and converse with her.</p> <p>Intermittently excitable and abusive but most of the time industrious.</p> <p>Pattern as above continues.</p> <p>Intermittently accuses others of stealing possessions and threatens to kill them.</p> <p>Health varies between good and indifferent. Eats badly.</p>
7. MK 34	10th Sept1898	?	MA	Cardiac failure in recovery from typhoid	<p>Rather emaciated</p> <p>Head - Not examined</p> <p>Flaccid heart.</p> <p>Lungs not congested.</p> <p>Large bowel : ulcers healing</p>	-
8. SG 51	16th April 1899	26 <sup>th</sup> Jan 1897 D	MA	Cardiac failure	<p>Rather emaciated</p> <p>Grey matter pale. White matter oedematous</p> <p>Some pericardial fluid. L</p> <p>Heart contracted. R ventricle is thin and myocardium shows fatty degeneration. ? mitral valve dilated, rest NAD.</p> <p>Lungs emphysematous.</p> <p>NAD</p>	<p>51-year-old widow, of no occupation. First attack, duration 3 months.</p> <p>Husband died 18 years ago.</p> <p>Two grown up children, one died in infancy.</p> <p>Not been in workhouse, living comfortably. Refuses food on occasions. Is intemperate.</p> <p>No FH</p> <p>Gradually getting worse over past 3 months.</p> <p>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.</p> <p>Her meat has been poisoned by vitriol.</p> <p>Quite robust and well nourished, superficial capillaries injected.</p>

						<p>Head tremulous. Reflexes brisk with a tendency to clonus.</p> <p>Mental:- Numerous ideas of persecution. Food poisoned. Pillows charged with electricity. Man named ??? is responsible for all this and he knows her thoughts.</p> <p>Sleeps and eats quite well but remains deluded. Says Catholic priests told her that laurel leaves were a good thing to poison yourself with. A suicide ticket has been issued for her.</p> <p>Later note says delusions and hallucinations unchanged.</p> <p>Says telephone wires are under the table and affecting her.</p> <p>Said to be in good bodily health.</p> <p>Delusions and halls continue.</p> <p>Developed spike in temp. Said to have influenza but recovered well.</p> <p>Syncope and died suddenly despite treatment 2 weeks later.</p>
9. SAK 41	11th Feb 1901	9th Dec 1891 E	MA	Heart disease	<p>Fairly nourished</p> <p>Grey matter slightly hyperaemic. White matter normal.</p> <p>Myocardium soft and degenerated.</p> <p>Atheroma of valvular arteries and first part of aorta.</p> <p>Lungs NAD</p> <p>Liver- nodules of fatty degeneration.</p>	<p>Married, Wokingham</p> <p>Lactation</p> <p>Exhausted, 2nd class 6 days First Married to farm labourer. Third attack of 5 days duration.</p> <p>Cause said to be exhaustion from lactation from too frequent pregnancies.</p> <p>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.</p> <p>Normally intelligent and temperate.</p> <p>No FH. Said to be dangerous to</p>

						<p>others and suicidal – attempted to jump out of window.</p> <p>Physical. Ruddy complexion. Tongue dry with thick yellow fur. Pulse small, regular and feeble. Systolic bruit at base and pulmonic area. Breasts full of milk.</p> <p>Mental state. Rambling and incoherent Attention poor. Talking to herself all the time.</p> <p>Given belladonna and strychnine plus others unreadable.</p> <p>Sleeps well sometimes but at others awake all night. Remain incoherent and talkative. Making no progress. Occasionally more tractable.</p> <p>Described as a very noisy chronic.</p> <p>Notes continued Vol 1 p 126.</p>
10. OL 43	23rd Aug 1901	12th July 1899 D	MA	Morbis Cordis	<p>Well nourished</p> <p>Brain soft. Gyri are wasted. Grey matter pale.</p> <p>White matter is slightly oedematous</p> <p>Atheroma of basal arteries.</p> <p>Pericardium contains fluid.</p> <p>Hypertrophy of L ventricle. Dilated ventricles. Incompetent mitral and tricuspid valves. Some oedema of lungs.</p> <p>Liver-coarse cirrhosis</p>	<p>Married, Previous episode 2nd class Morbis cordis 1 month. Second Married dressmaker. Second attack, one month's duration.</p> <p>She has been married twice, has 3 live children and 2 dead, cause unknown.</p> <p>Two months since her last confinement. Child died. In poor general health. Attack began suddenly.</p> <p>Admission order. Talks incoherently, cannot keep to subject for more than a second or two. Talks about the Lord and conversations with him. Says her eye is to be taken out. Can become very excited.</p> <p>Physical. Feeble . Pulse too small, mitral systolic murmur.</p> <p>Mental. Mildly excited. Voluble and talk incoherent with shifting themes.</p>

						<p>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.</p> <p>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.</p> <p>March 8th, 1900. Sudden onset of pre-cordial chest pain. Heart racing. Given digitalis.</p> <p>Settles but in June gets marked "cardiac dyspnoea". Becomes markedly oedematous. Increasing "failure of cardiac compensation". Dies. Said to have morbis cordis (valvular).</p>
11. MH 40	14th Sept 1902	2nd Oct 1900 D	ME	Pericarditis/ Morbis Cordis	<p>Emaciated</p> <p>Grey and white matter hyperaemic.</p> <p>Otherwise, NAD</p> <p>Pericardium adherent.</p> <p>Myocardium soft flabby and degenerated.</p> <p>Atheroma of mitral and aortic valves.</p> <p>Lungs congested and "engorged with blood".</p> <p>Liver hyperaemic.</p> <p>.</p>	<p>Housekeeper</p> <p>Widowed, Reading</p> <p>Drink and morbis cordis. 2nd class, heart disease. 6 weeks. First Husband died 8 years ago of TB. First attack of 6 weeks duration.</p> <p>One child alive, one died in infancy and one died of TB 3 months ago.</p> <p>No FH. Has weak heart and says she has a FH of that.</p> <p>Drinking to excess but temperate for last month.</p> <p>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</p>

						<p>increasing restlessness and use of bottle.</p> <p>Physical. Sallow complexion. Pulse 80/min weak. Soft systolic mitral murmur.</p> <p>Bruises on thighs.</p> <p>Mental. Restless. Thinks that everyone is disappearing and that burglars are in the house. Thinks she should protect everyone. Restless and refuses food.</p> <p>Continues very restless. Tears at clothes. Said to be very depressed several times</p> <p>Aug 1902. Episode of pre-cordial pain. Pulse rapid and soft. Rub heard over area. Temperature elevated at 100. Poultice applied to area. Temp remained elevated. Pulse rapid and weak. Given brandy 3 times a day. Died.</p> <p>Clinical cause of death given as pericarditis and morbis cordis.</p>
12. AY 45	8th June 1903	29 <sup>th</sup> Oct 1901 D	Chronic MA	Syncope (labelled as choking)	<p>Emaciated. Signs of tracheostomy .</p> <p>Trachea filled with hard pulp.</p> <p>Brain soft and gyri are wasted. Sulci gaping. Grey matter pale. White matter sodden and oedematous.</p> <p>Left ventricle hypertrophied. Mitral and tricuspid valve slightly incompetent.</p> <p>Occasional TB foci.</p>	<p>Married housewife</p> <p>Second attack</p> <p>Duration 18 months, 2nd class (morbis cordis)</p> <p>Not suicidal</p> <p>Married 4 years. 3 living children, one died, 6 months since the last one</p> <p>Living comfortably, clean, good habits</p> <p>No FH</p> <p>History hard to read.</p> <p>Says someone is stealing her food.</p>



					<p>Liver NAD</p> <p>.</p>	<p>People watch her. Something about stomach contents and her breast. Probable delusions.</p> <p>Feeble, physically. Feeble musculature.</p> <p>Grey complexion</p> <p>Pulse 80/min.</p> <p>Aortic systolic bruit.</p> <p>Mental state: delusions and hallucinations. Auditory and olfactory</p> <p>Someone has bewitched her. Hears voices. Thinks her neighbour is involved.</p> <p>Continues much the same. Has brief fever following attack of facial erysipelas</p> <p>Sleeps well but has delusions and hallucinations. Thinks there is poison in her bed and people coming to kill her.</p> <p>Passed bright blood per rectum which did not recur.</p> <p>Uterus is much enlarged.</p> <p>Weak and anaemic</p> <p>Liver is possibly enlarged but no growth.</p> <p>Sudden syncope when having breakfast. Doctor called and artificial respiration tried. Lot of food in mouth.</p> <p>Cause of death Syncope while having breakfast (verdict of Coroner jury)</p>
13. SM 54	30th Jul 1903	15th Oct 1902 C	ME	Cardiac disease.	<p>Well Nourished.</p> <p>Brain soft and gyri are wasted. Sulci gaping. Grey matter pale. White matter sodden and</p>	<p>Married, Windsor</p> <p>Heart disease</p> <p>3rd class. 2 weeks</p> <p>First 53-year-old married housewife.</p> <p>First attack, duration 14 days. Wife of a policeman. No children .</p>

					<p>oedematous. Atheroma of basal arteries.</p> <p>Myocardium soft flabby and degenerated.</p> <p>Atheroma of aortic and mitral valves. Chambers dilated.</p> <p>Some consolidation of L lower lobe.</p> <p>Well-marked nutmeg change.</p>	<p>Indifferent health. Rheumatic fever 5 years ago. Suffers from fainting fits.</p> <p>Normally cheerful, temperate.</p> <p>FH. Mother committed suicide.</p> <p>Became depressed, stopped eating food.</p> <p>Lost interest in habits or surroundings.</p> <p>Depressed with a dreadful feeling.</p> <p>Sees people around her when there is no one. Says she can see a rabbit with one eye and electricity coming from fingers.</p> <p>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.</p> <p>Feeble old woman. Poorly nourished. Poor musculature. Pulse 64/min of poor volume. Vessels "atheromatous". Heart action feeble, apex beat imperceptible.</p> <p>Systolic bruit.</p> <p>Mental state. Very depressed.</p> <p>Memory poor. Feels frightened.</p> <p>Remains very depressed , constantly weeping and saying, "don't send me away".</p> <p>Heart action weaker and dies.</p> <p>Cardiac disease.</p>
14. GH 27	10 <sup>th</sup> Dec 1904	9 <sup>th</sup> Dec 1904 A	MA	Cerebral haemorrhage	<p>Fairly nourished</p> <p>Bruises R arm and scalp.</p> <p>No skull fracture or "signs of violence"</p>	<p>School teacher</p> <p>Single, Farrington</p> <p>Over study</p>

				<p>Skull cap said to be thin.</p> <p>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.</p> <p>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.</p> <p>Ribs intact.</p> <p>Lungs tinged with blood L ventricle shows marked hypertrophy otherwise NAD.</p> <p>NAD</p>	<p>"Heredity neurotic". Single school teacher in her first attack of 14 days duration.</p> <p>Normally in good general health. Intelligent and temperate.</p> <p>No FH</p> <p>Said to be over anxious in preparing for an examination.</p> <p>Admission order. Visited four times by doctor. Getting increasingly restless. Has delusion that doctor is someone else.</p> <p>History from father, also a school master. Changed completely since a trivial boating accident in the summer.</p> <p>Physical. Pale complexion. Pupils dilated. Heart 100/min of fair tone. Heart and lungs normal.</p> <p>Mental. Rambles incoherently. Misidentifies place. Says she has only one foot and that she came here by motor from India.</p> <p>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.</p> <p>Cause of death cerebral haemorrhage ("Verdict of Coroners Jury")</p>
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15. MB 54	27th Jan 1905	12th Dec 1901 E	MA	Cerebral haemorrhage and softening	Emaciated. Haemorrhage in meningeal space of middle fossa of several days' duration. Brain soft and gyri are wasted and pale. Sulci gaping. White matter sodden and oedematous. Atheroma of basal arteries. L ventricle shows marked hypertrophy Atheroma of aortic valve. Other valves and chambers normal. Lungs emphysematous. Healed TB foci. Abdo:-NAD	Single, Wandsworth Asylum. Cause unknown. 2nd class 9 years 6 months First class Case notes missing
16. JC 55	6th Jan 1906	24th June 1898 E	MA	Fatty heart	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	Married, Windsor. Unknown 8 months (?climacteric) 2nd Class. Obese and umbilical hernia 48-year-old married charwoman. Admitted Causefield Asylum one year earlier. Admission order there talks of acting strangely talking to herself and voices. Can be excitable and abusive. Well nourished. Pulse 72 of low volume. 1st heart sound muffled. Mental state. Has hallucinations. Talks to Indian Princess and reads a prayer to her. Says the nurse is the Duchess of York and the doctor is Prince of Peace and Lord of Lords. Laughs and smiles a lot in response to voices.

						<p>Calls herself Princess Coles. Said to have delusions of exaltation. Excited , decks herself out. Bodily health good.</p> <p>Remains exalted but becoming weaker. Dies suddenly.</p>
17. CC 45	9 <sup>th</sup> Jan 1906	28th June 1890 E	MA	Cardiac failure (morbus cordis)	<p>Fairly nourished with no bruises or signs of violence.</p> <p>Grey matter pale and white matter oedematous. Lateral ventricle enlarged with fluid.</p> <p>No atheroma of basal arteries.</p> <p>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.</p> <p>Liver congested.</p> <p>Otherwise, NAD</p>	<p>Single, Bradfield.</p> <p>Unknown</p> <p>Heart disease</p> <p>2nd Class</p> <p>Not known</p> <p>30-year-old single labourer.</p> <p>Admitted as a criminal lunatic and then transferred to Pauper register on 28/6/1890.</p> <p>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.</p> <p>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.</p> <p>At other times he is sullen and morose. Said to be spiteful and insulting but "fortunately it is seldom he deigns to speak".</p> <p>Put a shirt button up his penis to stop it being "injured" and also toenails in his ears which became</p>

						inflamed. Also rubs faeces on himself. Health said to be good.
18. JA 63	14th Jan 1897	12th July 1895 D	MA	Valvular Heart Disease	Fairly well nourished Gyri wasted, brain soft. Grey matter pale. White matter oedematous. Basal vessels atheromatous. Heart large and both ventricles hypertrophied. Mitral and tricuspid valves incompetent. Aortic and mitral valves calcareous and atheromatous. Congestion of lungs. Liver nutmeg change.	Single domestic. 3rd attack of 14 days duration. Inmate of Littlemore in 1869 for a year, Admitted to asylum in 184 and discharged "recovered" in 1891. Temperate and industrious. In workhouse since Jan 1895. No FH. Rather feeble but well nourished. Heart regular but loud systolic bruit Maniacal, rambling and incoherent. Talks of unrelated topics which couldn't be followed. Constantly laughing to herself. Remains in this state for many months. Improves but complaining of physical symptoms a lot. Develops oedema and irregular heart rhythm. Complains of precordial pain. Given digitalis and improves but subsequently collapses and digitalis stopped. Then improves for a bit before showing signs of cardiac failure (oedema of legs). Noted to have precordial friction sounds. Kept in bed, pleural effusion drained and rallies. Pleural effusion returns, become weaker and dies.
19. PL 59	23 <sup>rd</sup> July 1897 7	26th June 1897 B	MA	Heart Disease	Fairly well nourished Brain soft. White matter sodden and oedematous.	Single, of no occupation. First attack of several weeks.

					<p>Basal vessels atheromatous.</p> <p>Heart large. Myocardium hypertrophied, flabby and friable. All cavities dilated.</p> <p>Atheroma of aortic and mitral valves.</p> <p>Lungs NAD.</p> <p>Liver enlarged and fatty.</p> <p>Congestion of intestines.</p>	<p>Living comfortably but stated to be unintelligent.</p> <p>Threatened suicide by drowning.</p> <p>Not refused food. Not in workhouse.</p> <p>Swollen and varicose legs for some time and present attack is said to relate to worry about this.</p> <p>History of tearing off her clothes and attempting to jump out of a window.</p> <p>Feeble, but well nourished. Diastolic murmur in aortic area. Pulse 100/min, small.</p> <p>Varicose ulcers on feet.</p> <p>Said to be both demented and deluded but no details given. No evidence of hallucinations.</p> <p>Restless with poor sleep. Given medication (? name) to no effect.</p> <p>Develops diarrhoea and becomes weaker. Passes a bloody stool and dies.</p>
20. EY 57	27 Oct 1900	27th April 1900 C	ME	<p>Pulmonary haemorrhage and gangrene.</p>	<p>Fairly well nourished</p> <p>Gyri shrunken to slight degree. Grey matter hyperaemic.</p> <p>Basal vessels not atheromatous.</p> <p>Myocardium flabby and soft. With fatty degeneration. Atheroma of aortic and mitral valves. Blood clots in lungs.</p> <p>Liver fatty degeneration.</p>	<p>Widow of no occupation. In first attack of 7 weeks duration. Suicidal.</p> <p>Son is a patient in asylum, no other FH.</p> <p>Fairly intelligent. Temperate, good habits, not in Union.</p> <p>Actively suicidal for 7 weeks since she became very depressed.</p> <p>Nil specific physically.</p> <p>Mentally very melancholic and vacant.</p> <p>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.</p> <p>Food refusal and fed with the pump.</p> <p>Develops phlebitis.</p>

						Develops haemoptysis and a pleural effusion. Fluid withdrawn but became foetid when drain left in. Died
21. LP 60	16th Feb 1902	11th Jan 1902 C	MA	Cardiac failure	Emaciated  Brain soft, gyri wasted. White matter oedematous. Basal vessels atheromatous  Left ventricle much hypertrophied. Myocardium shows slight fatty change. Valves NAD. Some congestion of lungs. Liver fatty degeneration.	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.
22. ES 58	23 Dec 1902	13th Nov 1902 C	MA	Fatty Heart	Well nourished Brain soft. Very wasted, sulci prominent. White matter congested and oedematous. Basal vessels atheromatous. 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



					<p>Congestion of lungs. Liver congested</p>	<p>beliefs that he has to print millions of letters. Became spiteful to parents and blind sister.</p> <p>Physical Well nourished. NAD.</p> <p>Bruises on chest and arms.</p> <p>Noisy and abusive. Loses self-control easily. Strange ideas not well described. Poor memory.</p> <p>Given diet and purges.</p> <p>Continues noisy and thought disordered.</p> <p>Develops fever – said to be influenza- and becomes weak and dies.</p>
23. BA 59	20th April 1903	10th Sept 1901 D	ME	Cardiac failure	<p>Emaciated</p> <p>Gyri wasted. Grey matter oedematous and anaemic.</p> <p>Basal vessels very atheromatous.</p> <p>Heart small. Myocardium brown, soft and oedematous. Atheroma on 1st part and mitral valve. Coronary arteries very atheromatous.</p> <p>Tricuspid v dilated.</p> <p>Lungs oedematous.</p> <p>Liver congested</p>	<p>Carpet planner, married with 7 children. First attack.</p> <p>Always been eccentric. Temperate.</p> <p>Not worked for two years.</p> <p>Threatened his family with knives. Also threatened suicide.</p> <p>From admission record. Great change, once intelligent and quiet, now assertive and combative.</p> <p>Strange beliefs:- thought his mistress was Holland's gin and that he was knocked over by Puck.</p> <p>Physically, poorly nourished and feeble. Heart sounds weak, pulse poor tone.</p> <p>Mentally, anxious and emotional.</p> <p>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.</p> <p>Remains deluded re sister-in-law.</p>

						<p>Becomes more rational but still deluded re plot and he sees many were in it.</p> <p>Intermittently very depressed and was briefly in a cataleptic state.</p> <p>Had another bout of severe depression with catalepsy and in the middle of this a burst of excitement.</p> <p>Lost interest in surroundings. Sudden syncope and died.</p>
24. GJ 59	21st May 1903	20th July 1901 D	ME	Morbis cordis	<p>Emaciated</p> <p>Brain soft, convolutions thinned. Grey matter hyperaemic. White matter congested, soft and oedematous.</p> <p>Basal vessels atheromatous</p> <p>Myocardium soft pale and fatty.</p> <p>Valves dilated especially the tricuspid.</p> <p>1st part of aorta atheroma. Aortic valves thickened. LV dilated and hypertrophied. Lungs show evidence of bronchitis and congestion.</p> <p>Liver congested</p>	<p>Married with 10 children.</p> <p>Living comfortably, never in workhouse, but has not worked for past 6 months.</p> <p>Supposed cause is worry.</p> <p>Temperate, good behaviour.</p> <p>Despondent for some time, fearful he would do his family harm.</p> <p>Refusing food.</p> <p>Tried to kill himself by hanging.</p> <p>Says his jaw has "gone" and his face is split into halves.</p> <p>Actively suicidal.</p> <p>Emaciated on admission.</p> <p>Marked arcus senilis. Heart weak and irregular. Pulse 65/min, poor tone, vessels thick.</p> <p>Looks depressed. Says he has no stomach. Back passage blocked by a body that comes down from his chest.</p> <p>Course. Sleep poor, food intake poor, hypochondriacal, "very deluded about his viscera".</p> <p>Continues this way for many months. Recurrent attacks of bronchitis with "great cardiac embarrassment".</p>

						Loses strength, dies.
25. ET 65	15th Feb 1904	26th Feb 1903 C	ME	Not stated	<p>Not nourished</p> <p>Brain soft, gyri wasted.</p> <p>Grey and white matter oedematous. Basal vessels atheromatous</p> <p>Myocardium soft and friable.</p> <p>Atheroma of aortic and mitral valves.</p> <p>Lungs congested.</p> <p>Commencing cirrhosis of liver. Congestion of gut</p>	<p>Housewife.</p> <p>Normally an intelligent woman.</p> <p>6 months in Union.</p> <p>Attempted suicide by drowning.</p> <p>First attack followed her husband's death. Became depressed then disinhibited and troublesome.</p> <p>Refuses food.</p> <p>Fair health. Pulse 84/min, normal.</p> <p>Thyroid enlarged.</p> <p>Depressed and agitated. Keeps saying "oh dear".</p> <p>Memory and orientation very poor.</p> <p>Developed a tachycardia, raised temp and loose stools and died.</p> <p>Annie Rowles</p> <p>Married dressmaker in second attack of 3 months duration.</p> <p>One child. Intelligent and temperate.</p> <p>Threatened to drown herself.</p> <p>Food refusal.</p> <p>Strong FH of insanity.</p> <p>In Littlemore 16 years ago</p> <p>Out of work for some time, said to be cause of attack.</p> <p>Has become depressed and says she cannot stand this any longer.</p> <p>Repeatedly talks of drowning herself.</p> <p>Sees her face as damaged. Sleep very poor.</p> <p>Mentally, she reports that everything she did went wrong.</p> <p>Hears voices talking to her which distresses her. Did attempt to drown herself. Expression is melancholic.</p> <p>No findings on physical examination.</p> <p>Remains agitated and very depressed.</p>

						Developed a high temperature and died.
26. WC 61	8th Oct 1901	17th June 1901 C	MA	Cardiac failure	<p>Very obese</p> <p>Sloughing ulcer on leg with fracture underneath.</p> <p>Gyri wasted and sulci gaping. Brain soft.</p> <p>Grey matter pale. White matter oedematous.</p> <p>Basal vessels atheromatous.</p> <p>Myocardium flabby and soft. With fatty degeneration. Cavities dilated. Valves NAD.</p> <p>Congestion of lungs.</p> <p>Liver fatty degeneration</p>	<p>Married, no children, plate layer on GWR.</p> <p>2nd attack of two months duration.</p> <p>No family history, temperate.</p> <p>Become noisy and talkative and threatening people with his crutch.</p> <p>Making accusations against everyone and takes up boxing attitudes saying the devil is after him.</p> <p>Shouts and swears and is often unintelligible.</p> <p>Physically , well nourished. Heart sounds normal but feeble. Indurated ulcer on leg below knee.</p> <p>Mentally, cheerful and laughs and breaks into song.</p> <p>Persecutory delusions re mother-in-law.</p> <p>Course. Remains deluded and noisy but cheerful. Ulcer healing.</p> <p>Feel out of bed and fractured femur</p> <p>Develops a temperature and dies.</p>
27. AR 62	9th Aug 1904	30th June 1904 C	ME	Fatty Heart	<p>Well nourished</p> <p>Gyri wasted; sulci prominent.</p> <p>Grey matter thin and congested. White matter congested and oedematous. Basal vessels atheromatous.</p> <p>Myocardium soft , friable and very fatty. LV dilated.</p> <p>Atheroma of 1st part of aorta and of aortic valves.</p> <p>Mitral valve dilated.</p> <p>Congestion of lungs.</p>	<p>Married dressmaker in second attack of 3 months duration.</p> <p>One child. Intelligent and temperate.</p> <p>Threatened to drown herself.</p> <p>Food refusal.</p> <p>Strong FH of insanity.</p> <p>In Littlemore 16 years ago</p> <p>Out of work for some time, said to be cause of attack.</p> <p>Has become depressed and says she cannot stand this any longer.</p> <p>Repeatedly talks of drowning herself.</p> <p>Sees her face as damaged. Sleep very poor.</p>

					Liver nodular change.	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. Developed a high temperature and died.
28. WK 59	20th Dec 1904	9thDec 1901 D	MA	Morbis cordis	Fairly well nourished Brain soft- otherwise NAD Myocardium soft. Aorta dilated and clotted. Valves all incompetent. Marked hypertrophy Lung shows evidence of pneumonia. Abdo NAD	Unknown 2nd class First 57 months. Case notes missing
29. SC 63	4th March 1905	29th May 1883 E	ME	Morbis cordis	Fairly well nourished Brain soft. Grey matter hyperaemic. White matter soft and hyperaemic. Basal vessels atheromatous. Myocardium hypertrophied. Anterior cusp of mitral valve atheroma. Lungs congested. Liver congested	Case notes missing
30. LL 60	26th Oct 1905	26th April 1898 E	ME	Cerebral haemorrhage	Well nourished. Brain soft, gyri wasted. Large yellow softening on R side affecting large parts of brain. Basal vessels atheromatous.	53, widow, stewardess. Transferred from Portsmouth. Probable second attack. Rambling and incoherent. Continually says "I can't" or "I couldn't". Blames everything on a Mr. Coleman. Says he robbed her of

					<p>Myocardium hypertrophied.</p> <p>Atheroma of aortic and mitral valves.</p> <p>Lungs congested.</p> <p>Congestion of liver.</p>	<p>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.</p> <p>Has epileptic fit.</p> <p>Develops fever and changes at lung base.</p> <p>Said to have had a cerebral haemorrhage as cause of death later but no neurological changes described.</p>
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