Brain

The skin, muscles, and bones of patients could all be studied during the patient's lifetime to some degree. The brain, though, could only be fully investigated after death even as its functions could be studied indirectly and its structural defects inferred via reflex and other tests. Looking at the brain of the general paralytic patient in the postmortem room, it was clear to asylum researchers that the condition had a serious impact on the brain substance: it was soft and often riddled with unusual lesions. Doctors seized upon the general paralytic brain as an object that had much to tell them not only about the course of general paralysis, but also about mental disease and neurophysiology more broadly. Lesions were meticulously mapped in diagrammatic form to investigate localisation of brain function, and microscopic examination of the brain tissue revealed the 'spider cell,' which promised to reveal something more of the disease's aetiology.

In seeking to make the brain readable for such investigation, or to preserve it for future study or teaching, however, the organ itself posed challenges. Dealing with this softened substance called for new methods of preservation and modes of investigation in order to make the brain properly legible. This chapter considers the challenges that asylum doctors faced when dealing with the brain as well as what their resulting investigations suggested, both about general paralysis's effect on the brain and the aetiology of the disease.

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Studying the Brain

James F. was admitted to the West Riding Asylum in May 1887. Unfortunately, he was no stranger to the institution, having been discharged just a few months earlier. His sister reported that since his discharge he had continued to wander the streets, often disappearing for days at a time. He refused to give any explanation for his disappearances and when he was at home was said to be going about frightening the local children by "making grimaces" at them. Entering the Asylum for a second time, James told the admitting doctor an elaborate story about a divorce, an illegitimate child, and a large quantity of jewellery that he possessed. Listening to the delusions of grandeur so characteristic of general paralysis and noting James's tremulous tongue, flabby muscles, and sluggish reflexes, the doctor diagnosed "Dementia, with General Paralysis."[1] During his time in the Asylum James's doctors could do little but watch him slowly succumb to the disease as he lost control over his movements and suffered an increasing number of seizures. Two years after his admission he was having fits every two to three weeks, his speech was slurred, and his patellar reflexes had disappeared completely. Upon James's death in July 1889, the West Riding doctors continued to examine his body for hints to the root of his condition. As one trace of James ended in a case-book record, another began in the postmortem book, where his major organs were examined and their appearances recorded in meticulous detail. Half of James's postmortem record was dedicated to his brain, particularly whether any unusual appearances could be seen on the organ's surface. Imagined as the root of James's disorder, the brain was the final part of his body to come under the doctor's gaze: it was examined closely, divided into sections, and the weight of each section carefully recorded.[2]

James's doctors were not unusual in their interest in his brain. What was notable at the time of their investigations, though, was the level of detail with which they were able to investigate the organ. Scholars had been musing upon the structure and functions of the brain for many years; in the seventeenth century Thomas Willis had suggested that the complexity of the convolutions of the brain could indicate an individual's intellectual ability. This analogy between intellect and physical structure could also be seen in Franz Joseph Gall and Johann Gaspar Spurzheim's early nineteenth-century phrenology. The rationale of phrenology was quite simple: the outer shape of the skull was used to make estimations of the structure of the brain inside. Gall and Spurzheim produced elaborately

detailed 'maps' of the brain, attempting to locate discrete psychological attributes in specific areas, including characteristics such as amiability or wittiness. Though intriguing, their conclusions rested on some questionable evidence. A frequently cited example was Gall's recollection of a classmate who had peculiar bulging eyes and an excellent verbal memory, leading him to the conclusion that the frontal lobe was the seat of language (the lobe in his classmate's case being so well-developed as to push the eyes outwards).[3] At the same time that Gall and Spurzheim were espousing their doctrine of phrenology, medical men in France were beginning to correlate clinical symptoms with the findings of pathological anatomy and physiological experiment. Here, the focus was less on personality traits or intelligence than the kinds of motor functions and anomalies met with in Chapter "Muscle". Paul Broca was said to have presented some of the most convincing evidence for a link between cerebral damage and loss of function at an 1861 meeting of the Paris Société d'Anthropologie. There, he presented the brain of a patient, nicknamed 'Tan' on account of the only syllable he could utter, who had suffered from language difficulties; his brain had marked frontal lobe damage.[4] Work like Broca's made a strong case for the place of pathological anatomy in localisation work, gaining a deeper understanding of the brain and mapping its functions. It did not take long for the French model to reach England, where it arrived into an atmosphere of enthusiastic innovation in medicine—the rise of anaesthesia and antiseptics, for example—and a healthy increase in professional journals that allowed researchers to disseminate their findings beyond their own institutions.

The concept of localisation of function clearly appealed to the physiological method for its supporting evidence, and was closely tied to the earlier doctrine of phrenology. By the end of the 1840s, though, "the full-time, pure physiologist, who … devoted all his time to research on the nervous system" may have distanced himself from the (by then somewhat outmoded) theories of Gall.[5] Instead, as L. Stephen Jacyna argues, he found in physiological psychology "a partly novel way of continuing an old argument."[6] The work of researchers such as Marshall Hall, on the 'reflex arc' of the spinal cord, had made the distinct separation of mind and matter increasingly difficult. The reflex model of function as articulated by Thomas Laycock and Wilhelm Griesinger, for example, precipitated the kind of tests described in "Muscle" that turned the patient's body into "a machine of its own revealing."[7] The reflex model provided the tools through which alienists could imagine the somatic origins of mental disorder.[8] It

underwent an important development in the work of John Hughlings Jackson who—influenced by Herbert Spencer (in turn influenced by Gall)—applied evolutionary logic to neurological disorders and achieved widespread renown for his work on epilepsy in the 1870s.[9] To Jackson mind and body were parallel yet separate, a concept expressed in his "doctrine of concomitance"; this idea allowed the nervous system to be understood from both a psychological and a physiological point of view.[10] Historians have emphasised the contribution of Jackson's work as primarily conceptual, rather than practical (though it drew strongly on principles from the natural sciences).[11] Even as a basic conceptual framework, Jackson was able to use his theory to remind researchers of the importance of the structure and function of the nervous centres to normal mental and bodily processes. In extending and modifying the theory of evolution with his notion of a "dissolution" that could affect the brain's functions, he provided a neat, if somewhat vague, explanation of the mechanics of mental decline (though Jackson's concern was the sensory-motor functioning of the brain, it was difficult to do away entirely with the psychological aspect as studied by Gall).

Jackson's work strongly influenced David Ferrier; the latter's investigations into cerebral localisation at the West Riding referred back explicitly to Jackson's epilepsy studies as he attempted to induce seizures in animals using electric current. James Crichton-Browne, who had invited Ferrier to conduct his research at the West Riding Asylum, encapsulated at once the various influences described above. The French model of matching clinical symptoms to pathological anatomy was central to his approach, and his family background in phrenology (his father, W.A.F. Browne, lectured on the subject) had convinced him of the links between material change to the brain and mental disease, leading him to view gross pathology as the "staple of [asylum] research."[12] Browsing the contents of the in-house journal the *West Riding Lunatic Asylum Medical Reports*, one can see that this was an interest held by other staff and visiting doctors to the Asylum, with papers on everything from brain weight to cranial injuries and insanity.[13] Successive superintendents maintained Crichton-Browne's focus: William Bevan Lewis was a particularly keen observer, producing various articles on pathological technique as well as a substantial guide to histological methods, *The Human Brain*, in 1882.

The rationale behind this kind of asylum work in the nineteenth century—insanity as a disease of the brain—meant that the alienist profession was one that could be cautiously optimistic about its ability to relieve the

symptoms of, and possibly even cure, mental disease via physical methods. We might view the popularity of such an approach as being in its practical —somewhat self-serving—basis, as holding out the promise of professional autonomy for alienist science. Equally, though, it was an approach that reflected contemporary scientific concerns and, in the case of general paralysis, a degree of desperation. In 1876 Crichton-Browne asked whether "our professional descendants may look back with pity and censure upon the helpless attitude that we have been content to assume in the presence of general paralysis?"[14] It was a devastating disease and its rapid, vicious spread suggested—more so than other mental diseases—that the condition had a physical basis in the brain. Here, then, was a vague sense of hope: that there was a physical condition of the general paralytic brain that could be uncovered by careful investigation and thus form the basis of preventative or curative treatment.

A variety of work took place in the West Riding Asylum that aimed to uncover such a condition and to make the brain a legible object of scientific enquiry. David Gooding, Trevor Pinch, and Simon Schaffer refer to the 'work' needed to make an object an instrument ("a reliable transmitter of nature's messages"), and I use the term here to refer to the means of visualising brain disease that contributed to theories about the character and aetiology of general paralysis.[15] Whilst I do not consider scientific knowledge a straightforwardly determinative force, the productive effects of scientific labour are important when considering perceptions of general paralysis in the late nineteenth century. The parts of the body that could be surfaced by new forms of technology or scientific work were altered in this period and had a direct impact upon the pathological changes that were considered proof of the disease.

Studies of the brain in the late nineteenth-century asylum aimed to uncover the essential lesions of disease: visceral alterations to the brain substance. Investigations into the pathological nature of conditions such as general paralysis took place alongside efforts to make the recording and classification of pathological information more consistent. Like the meticulous recording of muscle strength or the breaking strain of ribs, the physical properties of the brain became one part of a wider quantitative exercise. The large volume of patients in the country's asylums, many of them chronic cases, made the need for an efficient bureaucratic apparatus increasingly urgent if doctors were to deal effectively with this growing population. In 1870, Montrose Asylum's James C. Howden emphasised that a standard system of recording lesions at postmortem should be

instituted so that comparisons could be made between different asylum populations, and he tabulated 235 cases according to the lesions present. John Batty Tuke at Fife and Kinross Asylum quickly took Howden's advice to heart and included a table of 75 cases in that asylum's 1871 annual report. Three years later, W.G. Balfour put together another table of lesions as found in 390 patients at Colney Hatch.[16] Unfortunately, these efforts were not the beginning of a widely accepted system, as researchers continued to use their own classification schemes or terminology according to their personal interests and theories. As a result, data from one population could often be interpreted in different ways. At the West Riding Francis Simpson criticised the conclusion of his colleague William Lloyd Andriezen that sclerosis of the brain (an overgrowth of cells) was common in epileptic idiocy and imbecility.[17] Although Andriezen's research "[led] the reader to suppose that [sclerosis] was present in nearly every case out of fourteen examined by him," when Simpson checked the postmortem records he found that Andriezen had performed 15 autopsies, only six of which were of the epileptic type he had described.[18] Simpson suggested that the high number of abnormalities during Andriezen's time at the Asylum was mere coincidence rather than evidence of characteristic changes in imbecility and idiocy. Andriezen's selection of case studies for his article was to some extent a consequence of contemporary publishing practices. It was much more attractive to write and publish perfect illustrative cases that justified one's work (and that of one's institution) than it was to relate ambiguous findings. Detailed case reports and studies "transformed uncertainty" and made sense of the sometimes anomalous results of lengthy investigations that had been carried out according to the individual researcher's or institution's specifications.[19]

The difficulty of instituting standardised systems didn't discourage asylum doctors from recording postmortem findings in great quantity and detail, however. There was, as we saw in Chapter "Bone", broad agreement about the value of detailed postmortem records, with the postmortem an important test of patient care as well as a means of closely investigating the pathological features of general paralysis. In 1889, West Riding Pathologist Frederick St. John Bullen assessed the results of 1565 postmortems carried out at the Asylum over a period of 11 years by a number of people, including three successive superintendents (James Crichton-Browne, Herbert Major, and William Bevan Lewis). Though Bullen acknowledged that not all had followed the same system, he was still able to present a wealth of material on the condition of the brains under

study.[20] Looking at the Asylum's postmortem records that Bullen drew upon, it is clear that staff were taking a steadily more systematic approach to recording their findings. Between 1880 and 1900, postmortem records evolved from a continuous block of text to text split into separate sections (head, thorax, and so on), later supplemented with the printed certificates described in Chapter "Bone", which reminded the recorder of the need to note any bedsores or broken bones.

In 1899, preprinted books for recording the results of postmortem investigations took on a more standardised format, with a specific number of lines allocated to heart, lungs, brain, and other organs and systems.[21] These record books are a clear contrast to the blank book of earlier years in which doctors were free to write as much as they wished and suggest, in their proscriptive layout, that prior investigations had highlighted the importance of specific features of the body and brain. Leafing through the postmortem books, however, it is clear that this more rigid layout garnered much less information than the free-form book of previous years, with many doctors completing each line or section with nothing more than a short sentence or one or two words. The layout of the books had a further function, though, acting as a guide or checklist for how the postmortem was to be carried out and which parts of the body were to be accorded special attention. From "External appearances" such as bruises, the record guided the observer from the head—the density of the skull, brain adhesions, state of the vessels—downwards, to the neck, thorax, and abdomen (each of these meriting much less space than the brain). How had the West Riding's doctors come to decide, via the increasing range of methods and technologies at their disposal, which pathological features were most important and worthy of record?

Viewing and Reading the Brain

As clinical and pathological methods developed in the second half of the nineteenth century, the parts of the body that could be studied in greater detail multiplied. And with these new ways of seeing, the physical features that were considered to be evidence of mental disease changed. The dynamometer ("Muscle") correlated low muscle strength with reduced mental ability, and the breaking strain test ("Bone") added softened bones to the pathological profile of general paralysis. Whilst new techniques and practices were readily applied to the study of skin, muscles, and bones, the brain remained the pinnacle of the investigative enterprise. Here was an

object that could act as a model for other conditions due to the staggering array of mental and physical symptoms that general paralysis generated. The brain in general paralysis was attacked in much the same way as the other bodily fabric discussed so far. Like the skin riddled with sores, the muscles replaced by fat, or the bone that was softened and greasy, the fabric of the brain was gradually broken down in a way that all too clearly mirrored the disease's devastating mental and physical effects. James F.'s brain appeared "reduced," contained clots and opaque areas, and was surrounded by a large amount of cerebrospinal fluid; the brain of a female general paralytic patient, Ellen W.H., was covered with thickened membranes that had stuck to the surface of the brain.[22]

In 1876, Crichton-Browne listed the typical features seen in the brains of general paralytic patients at postmortem and emphasised that his West Riding colleagues—upon seeing such changes—could "determine with accuracy … whether or not a brain submitted to them, of the history of which they knew nothing, had belonged to a patient affected by general paralysis."[23] In this he concurred with other researchers such as Berlin psychiatrist Carl Friedrich Otto Westphal, who claimed to find no anomalies in any insane brains at postmortem *except* in cases of general paralysis. In Westphal's opinion "it would be impossible to designate amongst a hundred miscellaneous brains those which have belonged to insane persons, if the cases of general paralysis had been eliminated."[24] Staff could, it seems, diagnose the disease purely on the basis of pathological appearances, with general paralysis at postmortem becoming a definite physical issue divorced from its clinical and social aspects. As Gayle Davis highlights, the diagnosis of general paralysis was a process, with different diagnoses possibly being made at a patient's admission, during their stay, and after death.[25] Certainly, there are several records at the West Riding in which general paralysis was only diagnosed towards the very end of a patient's life, and ultimately confirmed at postmortem.

The disease was evidenced, said Crichton-Browne, by softening and atrophy of the brain substance, a 'flattened' appearance of the whole organ, a thickening of the arachnoid (one of three membranes surrounding the brain and spinal cord), staining of the dura mater (the membrane outside the arachnoid and closest to the skull), and adhesion of the pia mater (the membrane closest to the brain) to the cerebral substance.[26] William Julius Mickle, arguably the most prolific writer on general paralysis, echoed Crichton-Browne's observations: a thickened arachnoid, discolouration, atrophy, and adhesions—though he also sounded a note of caution that "it

[would] not do to make too much of one factor in the morbid histology" of the disease.[27] By 1913, the essential lesions of general paralysis were little changed in Emil Kraepelin's *General Paresis*: cloudiness and thickening, adhesions, a "sinking-in of the brain substance," and a decrease in weight.[28] In the late nineteenth century a confident link between general paralysis and syphilis had not yet been made (though many suspected it), so that the presence of a gumma on the brain (a similar kind of growth to that affecting the skin in early-stage syphilis) was—if discovered—typically attributed to cerebral syphilis, which was often recorded as a separate condition. In a sample of brains from 95 male general paralytic patients, Francis Simpson noted only two instances of gumma, for example.[29]

There were, then, several appearances consistently mentioned in discussions of general paralysis's pathology that promised to reveal a little more of the disease's character. These could map the disease's progress as well as explain its clinical manifestations: anomalous blood vessels, brain wasting and softening, false membranes (or arachnoid cysts—an accumulation of fluid in the arachnoid), and cortical adhesions (the adherence of the pia mater to the brain substance). These were well illustrated by Harvey Baird's results of 131 postmortems performed on male general paralytic patients at the West Riding who had been admitted between 1896 and 1902. His results demonstrated the most common lesions seen in the disease, finding false membranes in 30.5%, cortical erosion in 55.7%, and "diseased basal vessels" in 53.2%.[30] The postmortem examination of Thomas S., whose death was the subject of a coroner's inquiry (as we saw in Chapter "Bone"), revealed a classic example of the general paralytic brain:

> The brain is pale externally – the membranes opaque & thickened over all but the occipital region. There is a considerable effusion of serum beneath the arachnoid. … There is considerable adhesion of the pia mater the adhesions much most [*sic*] marked over the left hemisphere, especially along the marginal convolution & angular gyrus. There is some thickening & opacity of the arachnoid over the inferior aspect of cerebellum on either side of the middle line. The grey matter of the convolutions is shallow & pale. The white matter has a shiney [*sic*], greasy appearance & shews some pale yellow discolouration. There are very many coarse vessels. … About 4 ½ ozs. of fluid escaped upon removal of the brain.[31]

Upon removing the brain from the skull at postmortem, the most immediately evident of these changes was cerebral softening. One brain, when

placed on a flat surface, had "hemispheres [that] separate[d] widely."[32] Like the muscles and bones, the brain was susceptible to degenerative, softening influences. Causes of death in asylum annual reports and minutes regularly included "softening of the brain" and the term was still used by some doctors at the beginning of the twentieth century as a synonym for general paralysis. In asylum admission documents, it is clear that "softening of the brain" was a term that could be used by doctors outside the asylum to refer to what they saw as a specific variety of mental disease. One doctor related that the patient had "[m]arked symptoms of brain softening," and another noted of a criminal lunatic: "His disease is softening of the brain, known as general paralysis of the insane."[33]

Although terms like this may appear somewhat vague and even 'quaint' to modern eyes, nineteenth-century diagnoses like softening of the brain should not simply be dismissed as mistaken explanations to be corrected by more 'enlightened' terms, nor should we assume that they were used by all doctors in a uniform manner. Indeed, the term 'softening' appeared to cause some controversy in contemporary medical circles, used in a variety of ways as well as in an extra-professional context. Guy's Hospital physician Samuel Wilks was critical of the unthinking use of terms like softening, arguing that it was an extra-medical expression often used as though referring to a disease itself—and indeed a layman might jokingly explain a friend's eccentric behaviour as the result of "softening of the brain."[34] Whereas softening of the brain was a long-term process, Wilks suggested that softening could also occur as the result of acute inflammation.[35] It was not unusual for inflammation itself to be viewed as "a disease process"—in contrast to the idea of inflammation as a beneficial response to damage as articulated by chemist and physician Georg Stahl in the eighteenth century.[36] Crichton-Browne expressed his support for the inflammatory theory of general paralysis, citing the presence of headache as proof of the brain's congested nature.[37]

Softening of the brain might have been a key indicator of mental disease, but it was a process that was maddening to the pathological investigator, in some instances reducing the brain to nothing more than a boggy mess. Taming this softened tissue was a frequent challenge for asylum pathologists. Bevan Lewis wrote a series of articles for the neurological journal *Brain* between 1880–1882, titled 'Methods of Preparing, Demonstrating, and Examining Cerebral Structure in Health and Disease.' One part of this series, 'Physical Properties of Grey and White Matter,' emphasised the importance of the student's own senses in observation, as there was "no

more exact gauge of consistence of texture than the rough-and-ready methods afforded by the sense of sight and touch."[38] Here, he described the key differences between a normal brain and the softened, diseased brain: whilst the former was "plump, rounded and compact," the latter was "flattened," "squat," and, when removed from the skull, revealed "flabby" convolutions and little resistance to pressure.[39]

Despite an obvious interest in these softened brains, they proved difficult to preserve as pathological specimens, although they could sometimes be salvaged as teaching materials. The brain of Hannah Y., although wasted and "flabby," was retained to be "used for injection" and one hemisphere of Joshua T.'s brain was reserved for "anatomical purposes."[40] Brains were often photographed during preservation as both an additional record of the specimen and as testament to the skill of asylum staff—such as the brain of William H., added to the Asylum museum as an example of a general paralytic brain (Fig. 1).[41] Contemporary concern for general paralysis—the large number of incurable patients in asylums, and the disease's debilitating mental and physical effects—was reflected in the number of brains from general paralytic patients that were preserved for museum use. A key step in making these brains ready for preservation was stripping away the outer tissues with a fine pair of forceps or a needle. Crichton-Browne complained that "[t]he brain in general paralysis [was] so soft that stripping with the forceps [was] not very successful," and noted that stripping the brain while it was immersed in water merely exacerbated the problem.[42] This added further frustration to the task of determining the seat of the disease. Bevan Lewis urged caution when dealing with such specimens: "the brain-tissue clings to the blade with unusual tenacity … As a result the cerebral tissues … are lacerated and tear away in shreds, leaving an unmistakable softened rottened [*sic*] aspect of the surface."[43] Postmortem reports repeatedly testified to the challenges of handling and preserving the softened brain: one noted that "[t]he cerebral substance was too soft to permit of fresh sections being obtained," another that "[s]ections failed."[44]

To study mental disease successfully it was necessary to overcome such obstacles. Crichton-Browne described a method he used in which the brain was "preserved in nitric acid, which [had] the property of hardening and condensing the cerebral substance, and which at the same time blacken[ed] and [ate] away all animal membranes with which it [came] into contact."[45] By steeping brains in this acidic solution for a few weeks the pia mater was consumed, leaving the convolutions of the brain intact and ready for further study.

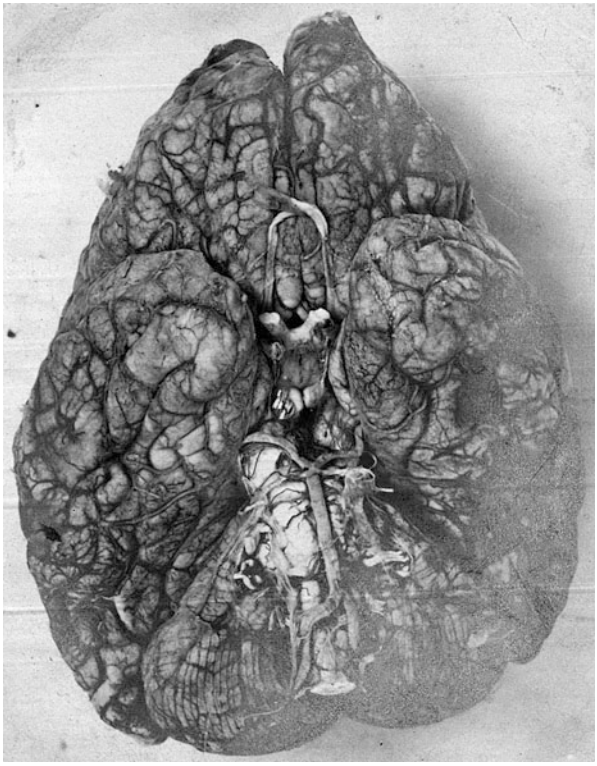

Fig. 1 The brain of general paralysis patient William H., added to the West Riding Asylum museum in 1896. Reproduced with permission of West Yorkshire Archive Service: Wakefield and the South West Yorkshire Partnership NHS Trust. WYAS C85/1111

Making the brain more easily readable was a central concern, then. Serial sectioning (cutting tissue into a series of thin sections) was crucial to a new view of the brain in the nineteenth century, as it exposed a number of the brain's features to the observer.[46] The West Riding's J.O. Wakelin Barratt, for example, cut whole sections of brain to trace the course of the ventricles through the organ, and Rainhill's Alfred W. Campbell pioneered the study of cytoarchitectonics (the cellular composition of bodily tissues), which relied heavily on serial sectioning.[47] The freehand sectioning or stripping employed in the 1870s was generally replaced by serial sectioning by the

1880s, but this too was a delicate task. H.R. Octavius Sankey advocated the use of an amputation knife, describing how to hold a brush in such a way as to keep the specimen constantly wet and thus easier to cut.[48] Cutting fine sections of tissue by hand—using a specially designed 'Valentin knife' or a razor blade for the purpose—was an exercise requiring significant skill and patience, but the introduction of the microtome made lighter work of a lengthy chore. Simple hand microtomes were "little more than a metal tube that [held] the embedded specimen and whose edges support[ed] the knife or the razor," yet they struggled to deal with large pieces of tissue like a whole brain.[49] Heini Hakosalo credits Bernhard von Gudden in Munich as the first to design a specialised microtome to deal with large specimens in 1875, though it seems likely that similar endeavours were occurring simultaneously elsewhere as innovative efforts tended to cluster together.[50] Bevan Lewis's freezing microtome was concomitant with similar innovations by Richard Hughes in Manchester and William Rutherford in London.[51] Freezing tissues prevented distortion under the scalpel, as well as removing the need for hardening a specimen—a process that could take months depending on the method used and which was, noted Bevan Lewis, "extremely tedious."[52] Freezing instruments typically used either ice and salt, or ether. The former had a tendency to break up tissue with ice crystals, so Bevan Lewis's microtome utilised the ether method. Using this apparatus, frozen sections could apparently be cut within 20 to 30 seconds.

The late nineteenth century saw an influx of equipment to aid the investigation and preservation of the brain substance at the West Riding, including a "Sliding Microtome (Rivett-Leiser Model)" and a "Cambridge Rocking Microtome" acquired in 1894–1895.[53] Besides technical apparatus, though, doctors evolved their own, simpler, methods of pathological investigation. We have already touched upon innovation in technology with Charles Mercier's breaking strain instrument and Theo Hyslop's use of a concrete-testing machine in the last chapter, and the same spirit was evident in studies of the brain. In 1872 Herbert Major described his tephrylometer, a hollow glass tube for measuring the depth of brain substance.[54] Similarly, Edwin Goodall related how a composition of glue and treacle was obtained from a local printer to make plaster casts of the brain in place of the usual plaster of Paris that—as Barratt had noted—tended to alter the shape of the tissues it surrounded due to its weight.[55] Such innovations and practices were a useful way of showcasing the practical talents of the researcher. Even the well-rehearsed methods of the skilful pathologist might not be sufficient to tame the extremely softened brain,

however. Sometimes the best that could be hoped for was obtaining "a little of the creamy pulp upon the scalpel, and transferring it to a slide" for microscopic examination.[56]

Softening of the brain was a phenomenon that invited a variety of readings as to its significance in mental disease, and it inspired the adoption and adaptation of existing sectioning instruments to deal with its problematic substance. Just as softened bone came to be seen as an important element of the disease profile of general paralysis, so too did the softened brain. And, as bone necessitated the evolution of new instruments and practices to study it, the brain highlighted the need for creativity in preservation and sectioning techniques. The softened brain was a material that had an impact on scientific practices in the asylum: new techniques and methods were evolved to make it readable and to render it suitable for preservation or further study. To the nineteenth-century alienist and physiologist, the brain was a highly charismatic object and, by the later years of the century, an increasingly rich substance with which to imagine the mechanics of mental disease, yielding as it did such clear physical evidence of degeneration. Brain softening, though, presented both practical and conceptual problems for the investigator: softening came to be viewed with some unease as an explanation for mental disease as nineteenth-century psychiatric research became increasingly physiologically oriented. Towards the end of the century, it was the specific lesion of the brain that began to arouse most interest, as postmortem investigations suggested the possibility of matching clinical symptoms to pathological findings.

Localising Lesions

A 'lesion' is generally defined in terms of harm, a change in structure as the result of injury or disease. For many historians of medicine, the lesion has particular significance as a sign of the increasing professionalisation of medicine in the nineteenth century. In *The Birth of the Clinic* (1963), Michel Foucault argues that the lesion became the focal point of disease in the nineteenth century—a reorientation of perspective that was assisted in large part by postmortems and hospital medicine.[57] It should be noted that although an interest in morbid anatomy can be discerned before the nineteenth century, certainly asylums and hospitals routinely allowed the exploration of lesions. Pathological investigation like that described by Foucault is often imagined to have reduced the importance of clinical

phenomena, as doctors directed their attention to the physical fabric of patients' bodies rather than their reported physical and mental symptoms. Andrew Hodgkiss has described the lesion as a powerful means of transferring decisions about bodily health and disease from patient to doctor: whereas the patient may decide that they are ill, the ability to identify disease as evidenced by a lesion rests only with the doctor.[58] The bodily lesion was not simply proof of the physician's or pathologist's specialist knowledge, however. To the asylum investigator it was the key to unearthing the cause of mental disease and the possibility of relief or even cure. As Edward Long Fox put it, disease was "nature's most delicate experiment," "her constant mode of teaching": by looking at the anomalous appearances of the insane brain, one could trace the sequence of events that produced them.[59] There had been attempts to systematically investigate brain lesions in the insane earlier in the century, such as James George Davey's survey of 100 brains in which he tabulated unusual appearances including softening and colour changes.[60] By the 1850s cerebral dysfunction was commonly "explained in terms of deformity in the tissues of the hemispheres," mirroring wider views of disease as the result of physical deformities.[61] Increasingly, medical journals printed accounts of postmortem work "in which researchers [tried] to link softening, discoloration, or erosion of areas of the brain … with loss or disturbance of functions."[62] At the West Riding, one staff member systematically recorded lesions found at postmortem by adding a short summary of brain lesions to the end of each postmortem record; later books contained an index of "abnormalities," with page references for records containing "Tumours & Cysts" and "Cortical Erosions."[63]

Lesions didn't always reveal a neat sequence of events, however. The work of late nineteenth-century alienists makes clear that the lesions they discovered at postmortem were often related to existing definitions of disease with some difficulty. W.J. Collins, of Claybury Asylum, commented on the disparity between "the proportional littleness of [brain] lesions with the magnitude of the malady": it seemed inconceivable that a tiny blemish on the surface of the brain could be responsible for the profound changes to patients' lives.[64] How could one rationalise the experience of William T., a 42-year-old tailor who, unable to continue his work, found himself in the workhouse and then the asylum?[65] Or that of William R. who, by the time of his admission in 1873, had lost his job and attempted to strangle his wife?[66] At the postmortem of John H., a criminal lunatic and "undoubted G.P." who had died just two months after admission, the doctor seemed to

express some surprise that no lesions had been found on the brain. Before death, John's doctors had speculated that—following a seizure that had ushered in almost constant twitching of the right side of his body—they would see lesions on the left side of the brain after his death. At postmortem it was noted: "Nothing whatever special is found on the left hemisphere at the site supposed to correspond with the movements of [the] right arm."[67] From a practical point of view, there was also the difficulty of charting a lesion's development: by the time postmortem was carried out, it could be impossible to identify a lesion's starting point, not to mention the problem of differentiating between lesions and natural processes of decay. Contemporary ideas of what constituted a lesion could vary between practitioners: Fox, in *The Pathological Anatomy of the Nervous Centres* (1874), suggested that alterations in blood vessels could be considered lesions, whilst Bevan Lewis referred to "the lesion termed yellow softening of the brain."[68]

In general paralysis, it was the occurrence of several lesions at once that merited attention. Bullen pointed out that the deciding factor was not necessarily the specific lesions themselves, but their appearance in the same brain: "It can hardly be said that appearances exist which by themselves are distinctive of *general paralysis*. Nevertheless, there are many common to it and other varieties of insanity which, by their grouping and aggregation in especial plenitude, denote certain characteristic features, having rightly an association with this disease."[69] The brain of the general paralytic patient seemed susceptible to a whole host of degenerative processes, but of these one in particular stood out, both on account of the perceptible change to the brain and the practical challenge that it posed to those undertaking pathological work. Morbid adhesions—in which the pia mater became stuck to the brain's surface—caused the brain substance to tear away from the membranes encasing it when it was removed at postmortem, leaving behind a distinctive worm-eaten appearance:

> On attempting to strip off a portion of adherent membrane, there are seen by the naked-eye numerous tough fibrous prolongations, which look like enlarged blood-vessels, connecting the under-surface of the pia with the cortex of the brain. When forcibly removed, the upper layers peel away to varying depths upon the pia, leaving an eroded surface which presents a highly characteristic aspect. The surface looks gnawed or worm-eaten along the length of the gyri with irregular sinuous margins, so that it somewhat

> resembles the aspect presented by a succulent leaf which has been attacked by a caterpillar.[70]

This condition was well illustrated by the frontispiece to Thomas Smith Clouston's *Clinical Lectures on Mental Diseases* (1883), which clearly showed the small pieces of brain substance adherent to the membrane that had been pulled back from the organ (Fig. 2).[71] Both Clouston's illustration and Bevan Lewis's observation that these changes could be "seen by the naked-eye" backed up the assertion of Crichton-Browne that the importance of adhesion in general paralysis had been overlooked due to an over-reliance on the microscope. Crichton-Browne emphasised the importance of naked-eye examination at postmortem, though he was clear that this was to be far from a "cursory glance."[72] Whilst he had seen adhesions in 80% of the general paralytic cases coming under his gaze, Crichton-Browne noted that adhesions were not always immediately obvious and required careful detective work as they were sometimes hidden within the folds on the brain's surface. In this endeavour, Crichton-Browne brings to mind Christopher Lawrence's "gentleman-physician," whose special skill was evidenced by his disembodied judgement as well as his manual activity.[73] The asylum doctor, though he might carry out postmortems as well as make psychological diagnoses from the comfort of his office, could perhaps still distinguish himself from the dissector or surgeon by emphasising the "incommunicable" knowledge that he possessed, such as how to find or infer adhesions of the brain in general paralysis.[74] Those cases of the disease where adhesions were *not* found at postmortem aroused Crichton-Browne's suspicions, suggesting to him that a "laborious and time-consuming examination of the brain" had not been carried out.[75] Even when not present, then, adhesion could be inferred, and the expectation that the brains of general paralytic patients would present adhesions is evident in the West Riding's postmortem books. One noted that "[t]he extent of the adhesion was not marked, for General Paralysis," another that "adhesion of the pia mater [was] ascertained to be present in every lobe and region of the brain," suggesting a purposeful search for this feature.[76] The importance of adhesion to contemporary observers is evident in the notations added in red ink to many postmortem records, denoting whether the case was one of "GP with adhesions" or "GP without adhesions."[77]

Most interesting to Crichton-Browne was the location of these adhesions. The degenerative character of general paralysis, evidenced in both

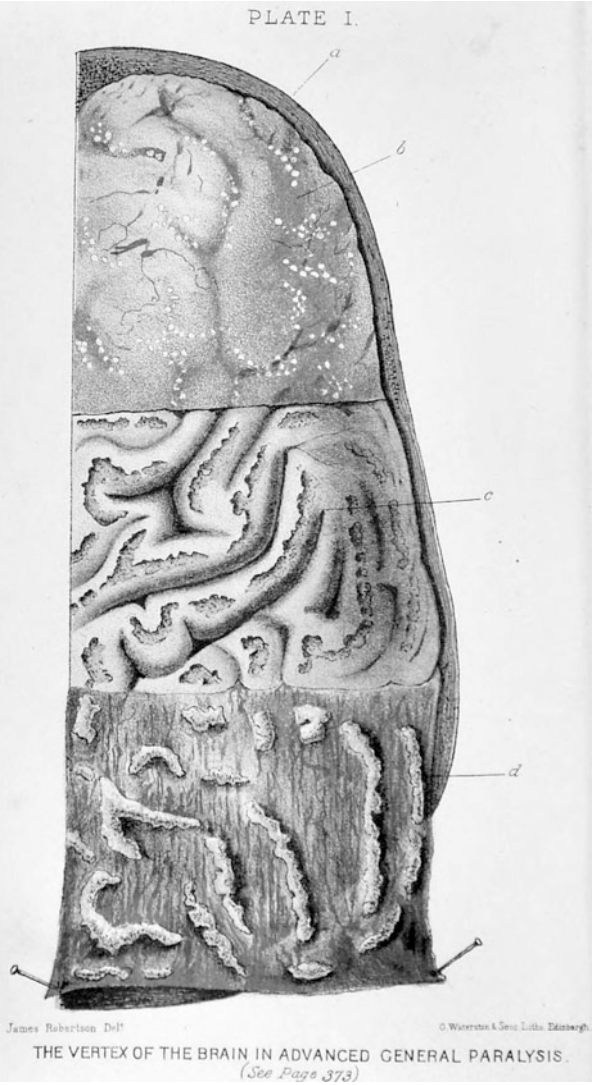

Fig. 2 Adhesions of the brain in general paralysis. The frontispiece to Clouston's *Clinical Lectures on Mental Diseases* (1883). Reproduced with permission of the Wellcome Library, London

the patient's mental state and physical ability, had led several alienists to suggest that the condition was a perfect example of Jackson's theory of dissolution. It was unsurprising that doctors would link visceral changes to the brain substance with the 'dissolution' of mental and physical functions. General paralysis had clear relevance for localisation theory, "[imitating] the experiments of the physiologist upon the brain" by producing lesions on the brain's surface.[78] The brain of the general paralytic patient offered a kind of natural experiment, with the physical damage the organ underwent simulating the ablation studies of the physiologist. For Crichton-Browne, pathological findings could inform and reflect upon wider theories of general paralysis as a kind of de-evolution. He had become more confident about the potential of pathological studies of the brain by the time of the final volume of the *West Riding Lunatic Asylum Medical Reports* in 1876. His paper on cranial injuries and mental disease in the first volume five years earlier was tentative about the links between the state of the brain and mental health, concluding that "derangement of the mental powers" could depend on a range of factors "which even aided by scientific instruments we are unequal to discover."[79] By 1876, however, he felt able to advise that the brain of a general paralytic patient should be obvious to "the practised eye."[80]

In charting the site of adhesions on the brain substance, the West Riding staff were following in a long tradition of producing morphological and functional maps of the brain, from medieval maps of the senses to phrenological drawings of character traits.[81] Such images were a means of proving and circulating one's own knowledge and experiences, as well as guiding other researchers in their experimentation. Brain lesions were systematically recorded at the West Riding from 1875 on small printed diagrams of the brain stuck into the postmortem book, upon which the doctor could shade in areas of adhesion or softening as well as mark the site of specific lesions.[82] In some cases the elaborate and careful detail of such diagrams and drawings sits in stark contrast to the content of the rest of the record. A haemorrhage in the brain of a melancholic patient who died in the first years of the twentieth century was meticulously illustrated by hand; the facing page—usually dedicated to the rest of the body—was blank and crossed through, however, where it was noted that postmortem examination of the rest of the body was "not allowed."[83] In the late nineteenth century, it is likely that the Asylum's pathologist would also have used 'brain slates' to record the location of lesions, an innovation introduced by James R. Whitwell at nearby Menston Asylum:

> MESSRS. DANIELSSEN & Co. have recently, at the suggestion of Dr. Whitwell, of Menston Asylum, made a set of engraved diagrams of the brain on slates for use in *post-mortem* and dissecting rooms. The diagrams are life size, twenty-five in number, and arranged on ten slates. The engraved outlines are filled in with white enamel, the Sylvian, Rolandic, and parieto-occipital fissures being, however, coloured red.[84]

Such slates were ideal for the postmortem room, marked in chalk, able to be used by the pathologist while his hands were wet, and reusable. The technical detail included in these slates demonstrates a clear concern for recording the site of lesions as accurately as possible. Slate six, for example, depicted a "Vertical section through the corpus callosum, anterior pillars of the fornix and optic chiasma; vertical section through the corpus callosum, optic thalamus and crura cerebri."[85]

Looking at the location of these lesions, whether on slates or printed diagrams, Crichton-Browne said he was reluctant to link them with clinical symptoms.[86] Here he agreed with his successor Herbert Major who, despite finding noticeable lesions in all general paralytic brains at post-mortem, could not "see his way to connect them with the abnormal symptoms present during life."[87] In practice, though, it proved difficult to discuss these lesions without appealing to symptomatology. The lesions of general paralysis made the dismissal of clinical symptoms particularly problematic due to their location, as they were typically found within the same area of the brain—one that was of special interest to asylum researchers. Crichton-Browne's 1876 paper noted that the frontal lobe of the brain was the "favourite site" of adhesions in general paralysis and this was a feature also noted by researchers elsewhere.[88] The West Riding's postmortem reports frequently referred to frontal lobe adhesion. In cases of "Dementia with GP" like James F.'s, adhesion was often noted: "adhesions exist at one or two points on the 2nd and 3rd left frontal convolutions"; "Strong adhesion between pia mater and the convolutions of the frontal and parietal regions on both sides; more especially over posterior part of 1st and 2nd of ascending frontal convolutions."[89] This apparent preference for the frontal lobe was too perfect to ignore as it epitomised the idea of general paralysis as a de-evolution, a disease that was little more than a reversion to baser instincts as it attacked the part of the brain believed to be the seat of the higher intellectual functions. Cases such as Broca's 'Tan' or that of American railway worker Phineas Gage around mid-century had illustrated the complexity of the frontal lobe, with injuries to discrete

sections of it leading to striking functional changes and even—in the case of Gage's injury to the prefrontal cortex—an apparent change in personality.

This notion of the frontal lobe—specifically the prefrontal—as an intricate entity that represented the pinnacle of human development was one explicitly appealed to in late nineteenth-century physiological writings. This was particularly evident in the work of Spencer and Jackson, both of whom drew strongly upon evolutionary theory, but also in works by other researchers. Mickle, in an 1895 article 'Atypical and Unusual Brain Forms,' noted the "enormous importance of the evolutionary advance of the lower part of the frontal lobe in man as compared with other animals, in the human adult as compared with the foetus."[90] His explanations of deviations from normal "brain-surface morphology" clearly aligned the atypical human brain with that of the animal or "savage." Of six possible reasons that he offered for deviation, two noted the concept of 'reversion'—either to the brain types of "lower animals" or to those of "lower mankind."[91] Bevan Lewis, referring to Ferrier's experiments in which he had removed the prefrontal region of monkey's brains, was "struck by the remarkable similarity presented, in the mental deterioration of [insane patients], to the animals in whom the prefrontal lobes had been removed": the animals had become apathetic, sleepy, and appeared to lose "the faculty of attentive and intelligent observation."[92]

By the last quarter of the nineteenth century, the importance of the frontal lobes in higher functions was increasingly accepted by the medical community, bolstered by animal experimentation and postmortem findings. It made sense that the symptoms of general paralysis—inarticulate speech and loss of control over motor functions—might be due to a lesion of the brain substance in the frontal lobes. Crichton-Browne also appealed to localisation theory when explaining the motor symptoms of general paralysis: the order in which these occurred, he said, corresponded "pretty closely with the order in which, according to Ferrier, the motor centres are arranged in the cerebrum from before backwards."[93] Crichton-Browne saw a footprint of the disease where the site of adhesions could not only explain its clinical manifestations, but also account for the various stages of general paralysis, with mental and motor symptoms appearing in a more or less predictable fashion as the disease spread through the substance of the brain. Initial minor problems with speech progressed, as in James F.'s case, to slurring and incoherent shouting, and motor ability diminished until the patient could do nothing but lie helplessly in bed.

To Mickle, the lesions visible at postmortem did not indicate the heart of the problem; the worst affected centres were "not necessarily those most obviously diseased," but those that felt the effects of disturbed circulation and nutrition—a phenomenon that was much less visible.[94] Like Crichton-Browne, Mickle's theories resulted from carefully mapping lesions at postmortem—in 1873 he began to record the location of adhesions of the pia mater to the cortex[95]—and like Crichton-Browne he could not avoid linking lesions with symptoms seen in life. In an 1881–1882 set of papers, 'Hallucinations in General Paralysis of the Insane,' Mickle examined what he saw as an overlooked clinical phenomenon. He had directly observed hallucinations in over half of his own general paralytic cases, he said, and combed the case histories of 100 patients—the majority of them soldiers—to conclude that hallucinations were present in 55%.[96] Visual hallucinations were especially common, and notable for their consistency: seeing people in rooms at night, huge birds flying through the air, angels, and heaps of corpses piled up on the wards. For Mickle the links between a patient's mental degeneration and the physical degeneration of the brain substance could not have been more explicit, as he argued that the symptoms of the disease flowed from structural lesions of the brain substance. Thus, the "crude, coarse, confused nature" of early hallucinations in general paralysis was directly related to the "coarse, gross, material character of the morbid process."[97] Reading Mickle's article, one is struck by his conclusions: whilst clearly keen to match adhesions with patient's hallucinations, this was not always possible. Observing that adhesions "sometimes [did], and sometimes [did] not, affect the supposed cortical sensory centres described by Ferrier," Mickle used this discrepancy to question localisation theory.[98] In discussing the lack of evidence for a link between auditory hallucinations and lesions of the temporo-sphenoidal region, he deduced that "the morbid anatomy of general paralysis fails to support the exclusive view that these gyri are, or contain, respectively the sole cortical centres of sight and hearing."[99] In 1883, during the discussion following a paper on cerebral localisation delivered by Bevan Lewis, Mickle's continued doubt about localisation theory was evident: he "did not agree with the rigid localisation attempted by some observers who made diagrams of the brain, and on it placed circles, within which they definitely locate[d] the movement of the arm or leg."[100]

Hallucinations in general paralysis were, for Mickle, one way of considering the much broader issue under discussion at the time: whether mental functions could be localised. General paralysis appeared to be the

ultimate test of cerebral localisation, with striking clinical symptoms matched by equally startling pathological lesions. Yet, as the work of Crichton-Browne and Mickle showed, these seemingly definite signs of disease could be used in very different ways. A review of volume six of the *West Riding Lunatic Asylum Medical Reports* containing Crichton-Browne's piece on the pathology of general paralysis was sceptical about the possibility of a characteristic general paralytic lesion. "The fact is that these adhesions merely represent the points where the disease has been most marked," said the anonymous reviewer, noting that their own investigations had not always uncovered adhesions.[101] Crichton-Browne reasoned that in protracted cases of the disease the adhesions may naturally, with time, break down.[102] This was a common means of explaining cases that lacked adhesion, and echoed Fox's claim that lack of postmortem lesions didn't mean there had been none during life.[103]

The fact that one could infer previous lesions when none were visible at postmortem suggests a strong professional attachment to the lesion in the later nineteenth century. For example, in an 1874 article, Hampstead Asylum Superintendent W.G. Balfour suggested that the failure to find lesions in the brains of asylum patients "must be regarded as due entirely to the absence of sufficient power on the part of observers to discover them."[104] In this he was not simply positing the lesion on the brain as symbolic of more advanced medical knowledge, but also had in mind the practical obstacles that doctors faced in undertaking such investigations. Lesions such as adhesion frequently posed problems for the asylum investigator. Barratt vividly reported a case in which he experienced "difficulty … in removing the skull-cap, which was thin but dense, owing to the existence of adhesions on the part of the dura mater."[105] Adhesions might also alter how postmortem technologies were employed. Major's tephrylometer (for determining cortex depth) first required the pia mater to be stripped from the brain, but this proved difficult in cases where adhesion existed.[106] As the utility of the lesion as a means of revealing the true nature of general paralysis was questioned, and the fabric of the brain was exposed to increasing attention, asylum investigators began to shift their view to even smaller elements within the skull. This was a reorientation of gaze strongly bound up with emerging medical technologies and techniques, and the last section of this chapter examines how microscopy's revealing of a particular kind of cell added a new layer to late nineteenth-century understandings of general paralysis.

Putting the Brain Under the Microscope

If the location of a lesion wasn't the answer to the mystery of general paralysis, perhaps the key lay deeper within the substance of the brain. Bevan Lewis, investigating the softened brain, had suggested that one possible method of analysis was the microscope, and indeed this was an avenue pursued on a regular basis in order to investigate all types of brain material, not just that of the general paralytic patient. As interest in bacteriology and microscopy grew in the later years of the century, there was a sense that the truth of any disease lay deep within the fabric of the body. In 1896 Bevan Lewis suggested that the microscopic appearances of all brains should be recorded by the pathologist and published at the end of each year.[107] Like the records of investigations into bone strength discussed in the last chapter, different schemes used by different researchers could be a barrier to such collection, and suggestions for universal methods were frequent. Francis Simpson, for example, proposed a chart—supplementary to the postmortem book—upon which the pathologist could simply tick the relevant boxes regarding the brain's appearance (adhesions, clots, and so on), then cut the chart into strips at the end of each year to count up the numbers in each column.[108] By the early twentieth century, a systematic record of microscopic observations was being kept at the West Riding, with reference in postmortem reports to various volumes of "Microscopical records" (unfortunately these have since been lost).[109] The importance of microscopy within the nineteenth-century asylum is often overlooked, but Eric Engstrom has noted how the craft could "to a degree even [prefigure] the concepts of madness and normality" by making new objects of investigation—such as nerve fibres—visible.[110] Microscopy also offered the opportunity to resolve the issue of why some general paralytic brains showed limited change by arguing that pathological lesions *did* exist if only one looked carefully enough. As with the cutting of sections, however, different brains and their constituent parts required tailored methods of investigation to properly comprehend their structure.

Once sectioned, slices of brain tissue were often preserved. Like photomicrographs of muscle tissue, they were important material teaching aids, allowing the staff of the Asylum to compare and contrast specimens in its on-site museum. Specimens could also be exchanged; one whole brain from the West Riding was preserved and sent to John Batty Tuke.[111] Edwin Goodall, Pathologist in the early 1890s, dedicated a good deal of attention to specimen preservation in his *Microscopical Examination of the*

Human Brain (1894) and in regular articles for the *JMS*, explaining how to take plaster casts of the brain (pulling the body into a sitting position using a rope and pulley)[112] and which preservatives were best suited to different tissues. Particular attention was paid to the quality of the specimens, with Goodall using a novel range of substances to make them as aesthetically pleasing and useful as possible: when placing brain specimens in glass vessels, he advised painting the back and sides of the jar with black bicycle varnish in order to give a clean black background.[113] He also listed the equipment needed to furnish an autopsy room, including Whitwell's brain slates and an ether-freezing microtome. If an asylum had a photographic studio—as the West Riding did—then this added the possibility of photographing specimens as well as preserving them. Goodall offered detailed instructions for photographing brains: the camera was to be fixed vertically and the organ "placed in water in a basin painted black; the basin stands on the floor. By immersing the brain in water the surface glistening is obviated."[114] A similar technique was in use at the Salpêtrière, with a camera fixed above the postmortem table to photograph organs in situ.[115] In the photograph album kept by the West Riding laboratory, it is clear that Goodall's technique was in use: one photograph shows a brain sitting in a vat of water, photographed from above. Taking photographs of specimens in this way also allowed them to be disseminated beyond the asylum to doctors elsewhere. In an article in the *JMS*, an image of West Riding patient John R.'s brain was reproduced, matching a photograph in the pathology lab album. Here, pathological knowledge went alongside clinical information to provide a complete picture of John's stay in the Asylum from his admission to his death.[116] The article described John's symptoms during life and the appearance of his brain after death, with the inclusion of the image allowing doctors elsewhere to engage in a kind of "virtual witnessing."[117] The study of the brain also extended beyond human specimens. In 1878 Bevan Lewis was awarded a government grant for research into the histology of the brain in animals, spending £6 9s. buying mammalian brains, ether for frozen sections, and paying the necessary postage costs.[118] Similarly, in 1879 Major detailed his observations of the brain of a white whale, which he had obtained from the Westminster Aquarium. A photograph of this brain was sent to Professor William Turner, Professor of Anatomy at the University of Edinburgh, who produced a detailed sketch of the specimen to accompany Major's article.[119]

In order to perform any didactic function, pathological specimens—whether as images in journals, photographs and specimens mailed to

colleagues, or preparations in a museum for medical students—needed to be rendered readable. The substance of the brain was a precious learning material for asylum doctors, as Goodall suggested in his advice for saving "old, over-hardened brains" by simmering them in almost-boiling water for several days.[120] After this somewhat basic method came the more meticulous business of sectioning and staining. In his history of microtechnique, Brian Bracegirdle describes how, in the 1870s–1880s, a host of coloured substances were investigated for their use in microscopy.[121] At the time of his 1882 article, Bevan Lewis listed six key stains in general use: hæmatoxylin, carmine, picro-carmine, aniline blue-black, aniline blue, and osmic acid. In choosing a stain he advised that the student should consider whether he was aiming for a uniform stain or a differentiation of the various tissue elements, and the particular tissue under study. Blue-black aniline, for example, was "by far the most valuable of the aniline series of dyes for the brain and spinal cord," giving "the clearest and sharpest definition of elements in a tissue without modifying their structure by shrinking or other change."[122] Postmortem reports demonstrate how the staining of a tissue could guide the observer in his identification of the elements before him, and how evolving microscopic techniques were being incorporated into the day-to-day work of the asylum. Following one postmortem, examination of the grey matter revealed:

> round or oval bodies … like swollen corpuscles … [that] took a faint lilac or pink hue with logwood [but] gave no reaction to the iodine & sulphuric acid test nor were they affected by ether, chloroform, or osmic acid. They were undoubtedly genuine colloid bodies [gelatinous material that appears in diseased tissue].[123]

Evolving microscopic techniques, then, affected what was seen by the researcher, with the cells of the brain coming under closer scrutiny beneath microscopes of increasing power and coloured by newly developed stains. Microscopy was not without its problems, however, and by the second half of the nineteenth century many practitioners would have consulted handbooks that set out the precautions to be taken in accounting for both the technical shortcomings of the microscope and the limited visual abilities of the observer.[124] Staining merited particular attention. Hæmatoxylin could easily overstain, requiring correction with other solutions and thus raising questions about the naturalness and ultimate usefulness of the result. Stains were taken up more easily in fresh (rather than preserved)

tissue, so that 'better' stains were dependent on the medium used. Ernest C. Carter, like Bevan Lewis, complained that the softened brain was particularly difficult to study microscopically: its easier take-up of stains caused the observer some difficulty in ascertaining "how far the condition [was] actually morbid and how far due to faulty sectioning."[125] The method of hardening used and whether the staining was carried out on a bright or dull day, were also highlighted as important factors that could alter the final result.[126] An awareness of these problems—and a note of caution about the rush towards microscopy—could be glimpsed in Brentwood Medical Officer John Turner's analysis of motor cortex cells, where he expressed his reservations about "these days of elaborate technique."[127] His own solution, however—"colouring small pieces of the fresh cortex with methylene blue, and pressing the fragment out under a cover-glass"—did not necessarily reduce the amount of labour required to obtain a satisfactory sample.[128]

Despite the difficulties accompanying microscopic observation, several researchers attempted to link minute cell-level changes to the clinical characteristics of general paralysis. Both Mickle and Bevan Lewis had attempted to type general paralysis according to the stage of the disease and relate these to changes in the fabric of the brain. Bevan Lewis divided the disease into three: the first stage was one of general inflammation, the second saw the growth of what he called "spider cells," large cells found in abundance in areas of connective tissue overgrowth, and the third saw the spread of these cells throughout the brain.[129] In one postmortem of a general paralytic patient: "Sections … cut by Bevan Lewis's fresh method and stained with Aniline Blue Black shew[ed] [an] immense number of spider cells in the superficial layer of the cortex and considerable increase in number of nuclei in vascular walls."[130] Also referred to as "phagocytes," spider cells provided physical and nutritive support for the brain's neurons, as well as ingesting foreign bodies or waste material.[131] It seems likely that the term spider cells as used at this time referred to what we now call astrocytes: these are specific to the brain and spinal cord and perform a number of functions, including repairing the brain tissue after injury. Indeed, astrocyte was used "as a synonym for 'spider-cell'" by Whittingham Hospital Pathologist F.W. Eurich in 1897.[132] The prolific presence of these cells in general paralysis thus appeared to be evidence of the body's response to invasion or injury. However, Bevan Lewis attributed the spider (or "scavenger") cell a novel double role, as both healer and destroyer:

> The function of a scavenger is to remove refuse, effete and useless material. But Bevan Lewis enlarges the function of these cells still further by attributing to them an active and aggressive part in the production of the diseased condition. He figures them as attacking and disintegrating the nerve cells with their processes which he regards as suckers.[133]

In diseased states, these cells "enlarge[d] very considerably into great amœboid-like masses" from which "radiate[d] on all sides numerous branching fibrils, forming an intricate and delicate network around it as a centre"[134]—hence the name spider cells. Though they were sometimes observed in healthy brains, spider cells were most easily spotted in diseased brains where they accumulated in vast numbers, shown up by deeper staining. Their sprawling growth had a destructive effect on outlying tissue and vessels:

> They become the "phagocytes" or *scavengers of the tissue*; live, thrive, and multiply upon the degenerating protoplasmic masses of nerve-cells and their extensions, and all effete material lying in their neighbourhood is ultimately appropriated to their use. These active scavengers are also destructive of the living tissues; they affix their sucker-like processes to any portion of their structure.[135]

These spidery outgrowths acted like ivy, strangling everything around them. Under such conditions, it was no surprise that an individual's motor or sensory powers might be altered. Prolific growth of these elements could "[grow] out in the form of brush-like processes into the infiltrated pia and thus [form] an adhesion between this and the cortex."[136] These were cells, then, that could also explain how adhesions appeared in the brain. Like adhesions, the presence of the spider cell suggested a form of reversion or atavism:

> In man [the spider cells] appear in scanty numbers; in the Barbary ape, they become more frequent; in the cat and ocelot, they are still more abundant; in the pig and sheep so profusely scattered are they that they form a most characteristic stratum immediately below the pia mater … We find these corpuscles in human brain which has undergone senile degeneration – in other diseases attended by reduction in functional activity, and in vascular affections resulting in retrogressive changes and a reversion to a low type of structure.[137]

It is difficult to gauge just how much support existed in wider medical circles for Bevan Lewis's theories about the spider cell, but his research into them was contemporaneous with that of Elie Metchnikov in Russia, who in 1882–1883 described white blood cells engulfing bacteria. Metchnikov, like Bevan Lewis, regarded these cells (which he later termed 'phagocytes') as primitive elements in the body, simultaneously protective and "wild" in their action.[138] He also associated them with the degeneration of tissue, including the brain. Despite support for his theory from the prominent German pathologist Rudolf Virchow, elsewhere Metchnikov's work caused significant and critical comment—particularly his notion of phagocytes as purposeful protective agents in the body. Back in Britain, Goodall claimed he had heard no criticism of the spider cell theory five years after Bevan Lewis published on it in 1889, but Tuke's *Dictionary of Psychological Medicine* (1892) pondered whether "too much importance [had been] attached to the function of these so-called scavenger cells."[139] Like the doubts expressed about pathological investigation in the previous chapter, some remained cautious of a comprehensive turn towards microscopy at the expense of other methods. George Thompson, Superintendent of Bristol City Asylum and previously Medical Officer at the West Riding, asked how useful it was to view phenomena under the microscope when one could see the manifestations of disease during life by utilising technologies such as the stethoscope.[140] Nevertheless, spider cells were a finding of some significance to those interested in the pathological 'footprint' of general paralysis. Goodall, keen to investigate the topic in more detail, carried out experiments using rabbits to discover how spider cells developed "in inflamed states of the cerebral cortex."[141]

Using animals for experiments was not unusual at this time, but research like Goodall's had come under attack more frequently since the foundation of Frances Power Cobbe's anti-vivisection organisation, the Victoria Street Society, in 1875. Following the 1876 Cruelty to Animals Act, vivisection could only be undertaken with a licence granted by the Home Secretary and the Society kept a watchful eye on scientists who they suspected to be operating illegally (they took Ferrier to court on this basis in the 1880s, but lost the case when it was argued that his assistant had carried out those parts of experiment requiring a licence). Susan Leigh Star suggests that such scrutiny succeeded in bringing together those researchers who were working on matters such as localisation of function, ironically aiding their research.[142] For Goodall and researchers elsewhere, animal experimentation played an important role in investigating the functioning of the brain,

but this rarely makes the accounts of Goodall's experiments less unsettling. Goodall seemed to be more involved than other members of the West Riding staff in animal experimentation and surgical intervention—including some operations carried out on patients during life, as we will see in the next chapter. Pursuing his research on spider cells, Goodall first trepanned a hole in a rabbit's skull, before injuring the brain mechanically (using a piece of wire) and chemically (introducing turpentine, carbolic acid, and other substances). He observed changes in the brain for a period of five weeks, though he noted that even "Within 28 *hours* … there was found great enlargement of the spider cells *beneath* the cortex."[143] Summing up his findings, Goodall concluded that it was the process of inflammation that brought these cells into view, confirming earlier observations of the general paralytic brain that had suggested inflammatory processes as an explanation for anomalous appearances such as softening. The spider cell's sprawling growth, then, was suggested to be the cause of other phenomena such as softening and adhesions.

In a short space of time the characteristic signs of general paralysis had become increasingly specific—from the rather vaguely defined 'softened' brain identified by Crichton-Browne to the tiny spider cell of Bevan Lewis. Softening and adhesions were brought into focus by the systematic, large-scale practice of postmortems and the tabulation of their results, whilst the spider cell was dependent on the microscope for its existence. Techniques such as serial sectioning and microscopy fed into evolving theories about the workings of the brain: sectioning techniques, for example, could undermine the significance of surface lesions by revealing the inner depths of the brain substance to be relatively intact. At the same time, these techniques threw up obstacles that asylum researchers had to negotiate, such as differentiating between unusually degenerated brain substance and over-staining. Although technologies like microscopy "inevitably framed the terms of psychiatric research," then, they did not necessarily "guarantee scientific consensus" or "produce any information [that merited] the hours spent collecting it."[144] With techniques still evolving and the results of detailed examination dependent on a whole array of variables, simple visual and tactile assessment remained crucial, as demonstrated by Bevan Lewis's advice to make use of "the rough-and-ready methods afforded by the sense of sight and touch." Despite these challenges, doctors at the West Riding continued to expend significant time and effort on the study of the general paralytic brain,