Fluid

This chapter considers the place of bodily fluids in the study of general paralysis, particularly cerebro spinal fluid (CSF) and urine. The study of the former was directly informed by postmortem evidence, which in turn fed into clinical interventions including trepanation. Though it did not lead to any breakthroughs in the systematic treatment or cure of general paralysis, trepanation was hotly debated by nineteenth-century practitioners both in terms of its efficacy and its ethical justifiability, and it is significant as an example of a physical operation that aimed to directly affect the brain. As the final chapter of the book, this chapter also brings us out of the nineteenth and into the twentieth century, when toxic theories of mental disease expanded upon existing work on the nature of general paralysis by suggesting that the condition was the result of toxins within the body. In studying the urine and other waste products of general paralytic patients, asylum researchers aimed to identify this toxin and thus the cause of general paralysis. During this investigative enterprise, the clinical picture of the condition as something often coincident with excessive drinking came to be reinforced by pathological findings that aligned the general paralytic body with that of the alcoholic. By the early twentieth century, laboratory-based studies had not eclipsed the social aspects of the disease, with significant attention still accorded to the role of heredity and the patient's personal history in the development of general paralysis.

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The Possibilities of the Postmortem

Chapter "Brain", described the time and effort expended by asylum researchers in examining the substance of the brain after death, much as—in "Bone"—we saw them dedicate significant attention to the study of the bones at postmortem. In the case of bones, despite initial enthusiasm for quantifying the bodily fabric using tools like Charles Mercier's breaking strain instrument, doctors increasingly recognised that such postmortem investigation did little to alter the lived experience of the patient or the asylum staff's responsibilities in caring for them. Was the asylum postmortem, then, a redundant exercise? Was it a practice that doctors undertook more for the sake of professional advancement and intellectual curiosity than for the good of the patient? Some historians have argued that postmortem pathology in the asylum was a practice that "seldom established new medical breakthroughs."[1] In assessing postmortem work in terms of its ability to produce medical breakthroughs, though, we are in danger of reverting to models of the history of medicine within which those productive effects of everyday practices that did not lead to a concrete discovery are lost. Michael Worboys, discussing the concept of a "bacteriological revolution," cautions against navigating through the history of medicine by means of specific discoveries such as the tubercle bacillus or the spirochete of syphilis. He argues that late nineteenth-century "Bacteriomania" was not defined by the rapid adoption of newly evolving practices and ideas, but rather "the uncertainties and possibilities of the new aetiological and pathological models."[2] What Worboys emphasises—and which is crucial here—is that ambiguous results did not necessarily mean unproductive or stagnant scientific investigation. In their examination of muscle tissue, bones, and lesions of the brain substance, asylum doctors were not stumbling blindly down an uncharted path, or carrying out pathological investigations without purpose. They were using these opportunities for observation and experiment to navigate complex issues of vulnerability, aetiology, and therapeutic possibilities. Though it might fail to solve an issue, the process of investigation could nevertheless lead to practical and theoretical developments that were important to contemporary observers (and continue to be so for historians of science and medicine). In the case of bones, although the investigations of asylum staff may have come to little in the sense of formulating an explicitly *medical* answer to the problem of fracture, they undoubtedly contributed towards a greater appreciation of the need to handle patients with extreme care, evidenced in

handbooks for attendants like that produced by the Medico-Psychological Association.

In examining the brain in general paralysis, as we saw in chapter "Brain", doctors found similar degenerative appearances to those seen in the bones. The importance of the physical brain to the study of general paralysis was agreed upon by almost all asylum investigators in the later years of the nineteenth century, but it was not necessarily a natural end point of investigation. Although death may be considered a source of authority, telling "the truth of disease"[3] (in, for example, the certain diagnosis of general paralysis that could replace a tentative diagnosis during life), the postmortem was often followed by a reorientation of the medical gaze towards the living body—sometimes due to a recognition of the limitations of pathological investigation. This tendency was identified in imperial German psychiatry by Eric Engstrom, who describes how alienists there "began going back to the patient's bedside in search of a viable system of disease classification" due to their "disappointment in the results of pathological anatomy."[4] This may also have been prompted by some practitioner's anxiety that, in locating the origins of disease in ever more discrete elements of the bodily fabric, the techniques of pathology and microscopy were overshadowing their own clinical skill.[5] The bedside and the bench could not be easily separated, however, and many published case studies included reference to both clinical and pathological findings in order to offer a complete narrative of disease. Although pathological results might be disappointing (the difficulty of definitively linking general paralysis and softened bones, for example), what was seen in the course of postmortems had the potential to influence what went on at the bedside as doctors linked pathological changes with clinical symptoms. There were two phenomena found at postmortem that suggested to doctors that the problem lay not in the substance of the brain itself, but in the fluid that surrounded it: false membranes and large amounts of CSF.

False membranes were said, like adhesions of the membranes to the brain substance, to be common in cases of general paralysis. The West Riding Asylum's J.O. Wakelin Barratt described them thus: "The fully formed membrane … consists of a very delicate, more or less grey or brownish-grey oedematous layer, lying upon, and adherent to, the inner surface of the dura mater, and exhibiting large thin-walled dilated vessels together with extravasations of blood of varying size."[6] In other words, these were a kind of blister or sac of blood found between the membranes surrounding the brain. In Joseph Wiglesworth's 1888 study of 400 postmortems, 42

(10.5%) of his sample had false membranes, 22 of these occurring in general paralytics.[7] At the West Riding, James Crichton-Browne found that general paralysis was aligned with 29 of 59 cases of false membranes, and William Bevan Lewis's later analysis of 73 cases found 34 occurring in general paralytic cases.[8] Postmortem reports frequently noted the presence and size of false membranes or blood clots in the brain, also depicting them in diagrammatic form like other lesions of the brain. False membranes were of sufficient interest to inspire a number of specialised instruments for dealing with them: a set of forceps for extracting false membranes were listed in a 1908 catalogue from London-based instrument makers Krohne & Sesemann.[9] That false membranes were connected with the profound changes taking place in the brain seemed apparent to many commentators: how could lesions of the brain substance occur without some morbid activity in the membranes and fluids surrounding it? The effusion of blood making up a false membrane, as well as suggesting underlying disturbance, implied some form of active production: upon patient John B.S.'s brain there was a "film of organised blood."[10] False membranes seemed to have their own network of blood vessels, aiding growth and providing stark visual evidence of the spread of disease. This was a disturbing sign, then, of unnatural changes taking place within the skull.

Charles Rosenberg, writing on early nineteenth-century American medicine, concludes that at that time "the body was seen as a system of intake and outgo—a system which had, necessarily, to remain in balance if the individual were to remain healthy … Equilibrium was synonymous with health, disequilibrium with illness."[11] Athena Vrettos, in *Somatic Fictions* (1995), argues that though Rosenberg's argument concentrates on the early nineteenth century, "his explanation of an internal paradigm of bodily economy" also informed medical thinking in later years.[12] The fixed energy model of the body was extended to the nervous system by a number of theorists in the second half of the century, and—from the 1860s—there was increasing reference to the "forces" of body and mind in works by prominent psychiatrists such as Henry Maudsley.[13] Balance was something that informed the body's workings at the most minute level, and the balance of fluids within the cerebrospinal system was something that caught the attention of a number of researchers working in asylum mortuaries and laboratories. Many postmortem examinations found the atrophied brains of general paralytic patients to be surrounded by an unusual amount of fluid. Joseph Workman found an astonishing one and a half pints in a skull

at postmortem, intimating that this explained why the brain had been compressed to half its normal size.[14] George Robertson, Superintendent of the Perth District Asylum, offered a neat explanation that harked back to ideas of humoral balance in the body: "As the cranium is a closed box, and its contents always completely fill it, when the active agent, the blood, increases or diminishes in amount, the passive agent, the CSF, must alter in amount inversely, the brain substance being regarded as neutral."[15] Thus, the fluid compensated for the loss of other bodily substances. This notion of fluid moving in to fill the space previously occupied by the brain and its blood supply could be seen in a number of articles, books, and postmortem reports: one West Riding postmortem examination found "wasting of the convolutions which [was] compensated for by 6oz. of fluid," another "Three ounces of compensatory fluid."[16] This fluid was used by some to make estimations of the brain's state prior to the patient's illness. T.W. McDowall's 1886 paper, 'An Unusually Heavy Brain in a General Paralytic,' clearly illustrated his belief that CSF was a compensatory product, arguing that the 11 oz. of escaped fluid "without doubt represented some ounces of brain tissue."[17] McDowall felt confident enough in this assertion to publish his results under a title that highlighted brain weight despite the brain being wasted; his "unusually heavy" brain was based on the brain substance present as well as the surrounding liquid that he believed represented several more ounces of degenerated brain tissue. At the West Riding the degree of brain atrophy or shrinkage was estimated in another way. Frederick St. John Bullen, writing in 1889, said that it was usual at every postmortem to preserve a ring of bone from the skull—and this is indeed noted in many postmortem reports. This segment of the skull —"taken through the occipital protuberance behind and through the frontal bone at about an inch above the root of the nose"—allowed the measurement of skull thickness. In conjunction with a wax cast of the cranial cavity, the pathologist could ascertain the volume of the skull more accurately than via external measurements, comparing the cast with the size of the brain to determine the extent of brain wasting.[18]

Not all were convinced that excess CSF was the result of a compensatory process in which fluid represented lost brain tissue. John Batty Tuke, Fife and Kinross Asylum Superintendent and Assistant Physician to the Royal Edinburgh Asylum, argued that it represented "an effusion produced by morbid processes going on in the brain."[19] Others highlighted the effects such liquid had on the brain and the rest of the body. An 1892 piece in *The Lancet* graphically portrayed the notion of balance as applied to the brain,

theorising that cerebral pressure had the effect of hindering respiration. A patient, on the operating table for the removal of a brain tumour, stopped breathing, whereupon the surgeon "punctured the brain, and witnessed the gratifying return of respiration in proportion as the pus flowed out."[20] Pressure within the brain caused by an increased amount of fluid might also be estimated by the 'gravity' of the brain tissue, determined by suspending the tissue in fluid.[21] The exchange of brain mass for fluid, then, fit into a wider picture of dynamic exchange within the body, with the fixed-energy model capable of being extended, at least theoretically, to other bodily material. Bodily balance was subverted by the body of the general paralytic patient, in which the maintenance of equilibrium had descended into chaos; this imbalance was graphically illustrated by the production of large amounts of fluid in the skull. In the final decade of the nineteenth century, several doctors theorised that it was this fluid—seen so often in the postmortem room—that was responsible for many of the symptoms of general paralysis. If this theory was correct, then perhaps there was some hope for the patient, though it could only come via one route: draining the fluid from the skull.

Trepanation and General Paralysis

In 1890, writing in the *BMJ*, T. Claye Shaw and Harrison Cripps related the case of a male general paralytic patient at Banstead Asylum. W.F. was suffering from a build-up of fluid in the skull that seemed to be exerting considerable pressure on his brain and causing severe headaches. The suggestion that their patient was suffering from excessive fluid in the skull was based on the many postmortems that Shaw and Cripps had witnessed, which had revealed large amounts of fluid in general paralytic cases. Removed to the care of Cripps at St. Bartholomew's Hospital, trepanation was performed: W.F. was given chloroform before Cripps made two one-inch trephine holes, one of them revealing bulging of the dura mater, a portion of which he removed. On returning to Banstead a few days later the patient's delusions and headache seemed to have abated.[22]

Physical intervention in the brain during the nineteenth century is probably most associated with the work of Swiss alienist Gottlieb Burckhardt. Burckhardt, who performed surgery on at least six patients under his care in 1888, is often credited with the birth of modern psychosurgery, having removed small portions of these patients' brains.[23] Indeed, he was declared "the first to propose and undertake a destructive

operation upon the anatomically intact brain in the hope of relieving mental symptoms" by his almost contemporaries and pioneers of lobotomy, Walter Freeman and James Watts.[24] Burckhardt's method of accessing the brain by making holes in the skull was a procedure that had gained significant interest in the second half of the nineteenth century, partly inspired by anthropological findings. In the early 1870s Paul Broca had examined a Peruvian skull sent to him by anthropologist Ephraim George Squier that bore the marks of trepanation; Squier wished to ascertain whether the patient had lived following the operation and Broca said that, in his opinion, they had.[25] Such archaeological discoveries found a receptive audience among alienists and neurologists who were actively investigating the connections between body and mind. Victor Horsley, for example, studied prehistoric skulls held in Paris's Broca Museum and used these to inform his own theories of the surgical treatment of conditions including epilepsy.[26] At around the same time, others were performing craniectomies (removing bone from the skull to increase the space available for the brain), usually on children diagnosed with idiocy. The Royal Albert Asylum's T. Telford-Smith estimated that over 200 such operations had been performed in Britain, America, and France between 1890 and 1896.[27] Yet within histories of surgery on the brain in the late nineteenth century, the use of trepanation in cases of general paralysis is barely discussed, with the notable exception of the work of German Berrios.[28] Berrios argues for trepanation as a form of psychosurgery—though practitioners were not removing portions of the brain, they were aiming to change its physical status in order to change behaviour—and calls for more research on such nineteenth-century interventions as well as the responses to them. The different responses to the work of Burckhardt, Shaw, and Cripps highlight—as Berrios notes—that sustained courses of experimentation and operation on the brain depended on social as well as medical forces.

In part, the relative paucity of historical work on physical interventions in nineteenth-century psychiatry likely stems from our own unease with the topic. The popular view of the Victorian asylum is usually one of sinister doctors needlessly operating on their helpless charges, and in tackling the history of psychosurgery we are forced to confront instances that come close to confirming that stereotype. Within the history of psychiatry, somatic treatments such as insulin therapy are also often used to signify profound shifts in psychiatric thinking, which can lead us to assume that somatic treatments were universally adopted and practised.[29] In surfacing

the body and the practices that surrounded it in the nineteenth-century asylum, we are obliged to examine not only the medical or scientific reasons behind those practices (both theoretical and practical), but also their social and ethical position. It is important to recognise that surgical interventions such as trepanation were not carried out without explicit discussion of their merits, risks, and ethical implications. Berrios argues that the use of trepanation in cases of general paralysis was not presented unproblematically by alienists and that its proponents "were aware of the potentially serious consequences of their treatment, and of the fact that—as well as careful antiseptic precautions—they needed scientific, ethical and social warrants."[30]

Shaw and Cripps were enthusiastic in their advocacy of trepanation, appealing to data that apparently demonstrated "increased arterial tension" in the early stages of general paralysis and arguing that "[t]he only way of stretching the brain [was] by giving it more space in which to expand."[31] In justifying the procedure, Shaw looked to established methods of counter-irritation: if blistering or cutting the skin to allow the escape of fluids could alleviate mental symptoms, then why not the release of fluid from inside the skull? Though logical considering contemporary observations of the brain at postmortem, Shaw and Cripps' surgical solution to general paralysis was not without controversy. Between 1889 and 1892, a number of articles and letters in the *BMJ* debated the appropriateness of trepanation in general paralysis on both theoretical and ethical grounds. To some, the intractable nature of general paralysis merited such measures. "[I]n the words of Professor Ferrier," wrote John Macpherson and David Wallace of Stirling Asylum, "the disease [was] so fatal that any experimental attempt to relieve it [was] justifiable."[32] Others took the view that trepanation was at best a temporary palliation of suffering via its relief of immediate symptoms, noting their contempt for the practice. Prestwich Asylum's George Revington criticised Shaw as well as John Batty Tuke (who had also utilised trepanation in a case of general paralysis) .[33] "I may mention the practical point," wrote Revington, "that general paralytics are quite sufficiently apt to injure themselves [as we saw in "Bone"], and to be injured by others without the additional facilities which a trephine hole in the skull would afford."[34] For Revington, once signs of general paralysis were discernible, the brain substance was already too far degenerated to expect any improvement to result from surgical intervention. Tuke was confident, though, that the operation could be performed with benefit for the patient—even if it only offered temporary relief—especially considering

the "simplicity" of the procedure.[35] Indeed, the operation was viewed as a relatively minor one; Shaw advised, for example, that there was no harm in carrying out a "primary trephining" to inspect the region of the brain suspected to be at issue.[36]

As we saw in Chapter "Skin", the West Riding staff were accustomed to carrying out operative procedures such as the excision of tumours. On at least one occasion, they also carried out trepanation. In January 1893, 40-year-old Elizabeth Ann A. was admitted to the Asylum after being found naked in an empty house. Though she expressed a number of delusions—one of which was that she was about to be married and required 15 carriages for the wedding day—she was described as "not at all excited," had experienced no fits, and was noted to have a facial expression that did not indicate any "special mental state," although she did have slightly slurred speech. It was likely this slurring, along with a slight anomaly of her right pupil and absence of knee jerks, that led the admitting doctor to record a tentative diagnosis of "? General Paralysis." Elizabeth Ann's casebook charts a startling development in her care shortly after her admission: an entry of 29 March 1893 noted that she was possibly "in remission," showing no mental symptoms of general paralysis, although her knee jerks were still absent. Less than a month later, however, on 17 April 1893, she "was trephined." The operation, undertaken by Edwin Goodall, made a hole in the right side of her skull. This did not reveal a large amount of fluid in the skull or any unusual appearances of the membranes covering the brain, although it was noted that at the moment of incision her right pupil "contracted distinctly." Over the two months following the operation, Elizabeth Ann experienced various ups and downs —primarily defects of speech and fits—but was soon sent out on trial before being fully discharged ("recovered") on 25 July.[37] A month later an article by Goodall in the *BMJ* described the operation as a general success, though he was chiefly interested in the aural hallucinations that Elizabeth Ann seemed to have experienced following the operation.[38]

Goodall would have been wise to wait a little longer before penning his article, as the following month—on 25 August 1893—Elizabeth Ann was readmitted to the West Riding. In contrast to her happy delusions of marriage upon her previous admission, this time she was described as "despondent," saying that she had died and come to life again and was "sure she will have something done to her."[39] It was a stark contrast to the spirit she had shown immediately following the operation, telling Goodall that he had "done his best to kill her" and that once her father heard of the

procedure he "would make him 'smart' for it."[40] It seems clear from the notes on Elizabeth Ann's second admission that the procedure had had a definite impact upon her mental state—she was said to be particularly distrustful of the medical staff—but no doctor attending her explicitly acknowledged this in the casebook. The trephine hole on her head was by now covered over in soft tissue (the piece of bone had not been replaced during the operation), her speech was slurred, her knee jerks were absent, and her pupils were of unequal size. This time diagnosed with "Melancholia [and] ? of Gen. Paralysis," she spent her time wandering the wards, not speaking, and had to be spoon-fed.[41] The notes for her second admission describe several years of fits and complaints of pain in the head until she died in January 1897, her death attributed to "Mania" and "Dementia of General Paralysis." Her postmortem found extensive erosion of the brain substance, and a microscopic examination by Francis Simpson seemed to suggest that trepanation had had some beneficial effect despite her condition in the years leading up to her death. He found "a much less degree of pathological change than [was] usual in G.P.s of such duration … [and] hardly any proliferation of spider cells."[42] Even when a patient died, then, it was possible to position trepanation as a positive and beneficial measure—emphasising that the operation *itself* was a success, but that the patient had (inevitably) died as the result of a fatal condition.

One thing that is notable in Elizabeth Ann's case is that trepanation was carried out when there seemed no extensive evidence for her disease being general paralysis. No explanation was offered as to why the operation was done only a month after she had been described as possibly in remission, except that she was suspected to be in the early stages of general paralysis and thus able to benefit from treatment. Remission was not unheard of in general paralysis, with some patients recovering sufficiently well to return home for a period. Others—like male general paralytic patient Samuel W.—were allowed home at the request of their families; Samuel's wife wrote to the Asylum after his death, thanking the superintendent for "granting is discharge so that we cud se the last of him at home [*sic*]."[43] Although some doctors undertook trepanation in cases where the signs of the disease were marked, Goodall seemed to have done it in a case with minimal indications. Berrios suggests that, in utilising trepanation, doctors such as Goodall were able "to target *individual* (troublesome) mental symptoms without committing themselves to having to treat the entire disease," influenced by a view of mental disease as the result of multiple lesions.[44] To Shaw and Cripps, the operation on W.F. still had its merits as the "painful urgent

symptoms" had been relieved. Underlying both W.F.'s case and that of Elizabeth Ann was the awareness that the general paralytic patient could not be expected to make a full recovery in any case. Surgical intervention was less about curing general paralysis than relieving its symptoms. Interventions such as these are a good example of why we should be cautious in dismissing surgical procedures like trepanation as misguided and 'extreme' attempts at bringing about complete cure; contemporary doctors did not necessarily view them in this way, but rather through the lens of palliative care or temporary relief. The case of Elizabeth Ann, though, raises the possibility of misdiagnosis: the operation was carried out without a great deal of evidence that she was suffering from general paralysis, and indeed justified as a measure that was to be taken early on in the disease, when only a limited number of symptoms might be present. And, crucially, in general paralysis—a condition with many varied physical and mental manifestations—it was not uncommon to confuse the condition with something else. Tuke warned that before undertaking any operation, the asylum doctor was to be absolutely sure that the case *was* one of general paralysis.[45]

Differential Diagnosis: General Paralysis and Alcohol

Surely, considering the many lesions left by the disease and its distinctive clinical character, there was little danger of confusion? As detailed in "Brain", many doctors expected general paralysis to leave noticeable lesions in its wake that testified to its distinctive and ferocious character. The picture though, was easily blurred, especially if one did not keep in mind that it was the appearance of these lesions *together* that confirmed the presence of general paralysis, as we saw in "Brain". Far from resolving the mysteries of general paralysis, pathological evidence in many cases had served to complicate the picture. Physician Edward Long Fox declared, when discussing arachnoid cysts (like the false membranes discussed in the last chapter, but filled with CSF instead of blood), that "Idiocy, dementia, and general paralysis [were] the forms of disease most usually associated with … cysts of the arachnoid; but, like many other cerebral lesions, they [were] found in persons who have not been insane."[46] Atrophy of the brain, too, was associated with all conditions of dementia. Thus many—if not all—of the essential lesions of general paralysis could be present in

other forms of insanity. Thomas Smith Clouston noted that false membranes were not only seen in general paralysis, but also "in some epileptics, a few maniacs, a few cases of senile insanity, some cases of ordinary dementia, and a small number of the cases of organic dementia with hemiplegia or local paralysis."[47] Even Crichton-Browne had to admit that he had seen adhesions in tuberculosis, meningitis, encephalitis, atrophy of the brain, and chronic alcoholism.[48] Later, Bevan Lewis would note that adhesion of the membrane to the cranium was common in all forms of chronic insanity—a logical result of long-standing inflammation.[49]

In relating a case of trepanation for the disease, Tuke noted that throughout his article he had placed 'general paralysis' in inverted commas in order to "indicate the opinion that it is really a generic term applied to very various and varied morbid conditions."[50] In an 1895 article, Reginald Farrar of Stamford and Rutland General Infirmary concluded that as the pathological signs of dementia and general paralysis were almost identical, they were the same disease. It was merely the demographic characteristics of the patient that tipped the balance, said Farrar: younger patients were said to be general paralytics whilst the elderly were diagnosed with dementia. Farrar labelled general paralysis nothing more than "a convenient clinical term for a *symptomen-complex*" with clinical and pathological signs that could simulate almost any other form of insanity.[51] Others expressed similar concerns. Alfred Fournier called general paralysis "a theory that [had] been allowed to run riot," and Samuel Wilks cynically surmised that alienists divided their cases into two: "functional and curable diseases such as mania and melancholia … [and] the organic and fatal … which in an asylum seem to be called general paralysis of the insane."[52]

The varied symptoms that could be seen in the disease made it all too easy to diagnose a case as general paralysis based on the findings of reflex tests or observations made at admission. A number of conditions existed that were said to be commonly confused with general paralysis, not only by the student but by experienced observers. Several clinical cases were reported throughout the period that noted the difficulty in differentiating general paralysis from tumour of the brain, for example. Some tumours presented no clinical symptoms (such as the Cane Hill Asylum patient who "never complained of headache, and had no vomiting," yet at postmortem was found to have a tumour as large as an orange in his frontal lobe), but more often they presented the kind of symptoms normally seen in general paralysis.[53] German neurologist Moritz Jastrowitz noted the peculiar cheerfulness seen in many tumour cases, and his contemporary Hermann

Oppenheim identified a tendency for such patients to "joke" and "make sarcastic or trivial answers."[54] Lead poisoning, or plumbism, was another condition that had some clinical similarities with general paralysis, inducing hallucinations, paralysis of the limbs, and tissue degeneration resembling muscular atrophy. In some poisoning cases death might also be preceded by fits or coma.[55] Henry Rayner, Alexander Robertson, George H. Savage, and Ringrose Atkins contributed their observations on the subject to an 1880 article, 'Insanity from Lead Poisoning,' which highlighted how easily the two conditions might be confused. F.S., a painter, described "in a confidential manner and with great evidence of self-satisfaction, the way in which he destroy[ed] [the] figures [he saw at night] by some power which he possesse[d]," whilst J.P. (also a painter), "In the early stages of his disorder … might well have been mistaken for a general paralytic" due to his maniacal attacks.[56] The use of the term "lead encephalopathy" by Rayner and his colleagues made clear that lead could affect the brain in a most direct manner, having serious neurological effects even after recovery (and also mirroring the symptom-free periods of some general paralytics). Less widely discussed but also bearing some resemblance to general paralysis was pellagrous paralysis. Caused by the consumption of diseased maize, pellagra was extensively studied by Franz von Tuczek, who performed eight autopsies on cases of pellagrous paralysis and claimed to find pathological changes similar to those seen in general paralysis.[57]

These were relatively uncommon conditions, however. Lead poisoning could be ruled out among those patients whose lives had not brought them into close or sustained contact with the substance, and pellagra was generally found only in areas where maize was the dominant crop (the condition was most often seen in rural areas of Europe, but also increasingly in the American South from the late nineteenth century).

The most difficult problem facing the British asylum doctor was how to differentiate the general paralytic from the alcoholic. Tuke's warning about being sure the disease was indeed general paralysis before performing trepanation singled out alcoholic insanity as the key complicating factor due to its demographic and clinical similarities.[58] Like general paralysis, alcoholic insanity often claimed men as its victims. Examining the histories of 464 patients at the West Riding whose insanity was attributed to excessive drinking, Bevan Lewis reported that 344 of these were male.[59] The initial clinical symptoms were remarkably similar to general paralysis: the staggering gait and boastful plans of the general paralytic often meant that his friends "very likely [thought] he [had] been drinking."[60] This

confusion worked both ways. Tuke was one of several practitioners who could relate cases that presented all the usual signs of general paralysis (slurred speech, disordered gait, and so on), but that had completely recovered after a short asylum stay; that their attacks had coincided with heavy drinking led him to warn other doctors about the possible simulation of general paralysis by alcohol and not to be too hasty in their diagnosis.[61] Twenty-five-year-old West Riding patient F.S. exhibited a range of delusions that on paper looked remarkably similar to the characteristic expansiveness of general paralysis, having "exalted notions respecting his muscular powers" and proclaiming he could "easily lift half-a-ton, and [had] often raised many hundred tons aloft." Within a fortnight he was considerably calmer, and after a short stay in the Asylum was discharged recovered.[62] As Samuel Wilks pronounced, "a drunken man might be said to have an acute general paralysis of the insane."[63]

In the late nineteenth century, medical concern for the effect of alcohol was growing, both in Britain and elsewhere. Alcoholism claimed the attention of medical professionals outside and inside asylums, from the man who arrived at work rather worse for wear and caused an accident in the factory, to the sufferer of 'alcohol-induced delirium' who took up valuable space in the country's asylums (and who was a prime candidate for the new inebriate asylums provided for under the 1898 Inebriates Act). A reinvigorated temperance movement, Habitual Drunkards legislation, and a growing appreciation of the unhealthy effects of excessive alcohol consumption meant that alcoholism occupied an increasing amount of doctor's thoughts and day-to-day work. In 1882, 19% of the West Riding's total admissions were attributed to alcohol use/abuse (in the same year, beer was discontinued as an item of the Asylum dietary).[64] Such statistics should be read with caution, however; high rates of admissions for alcoholism could be a result of strong local temperance movements.[65] Although the West Riding Asylum did not diagnose a disproportionately large amount of patients with alcoholic insanity (in 1885 just four of 202 men admitted were diagnosed with alcoholism or 'mania *a potu*'), it remained a point of interest in terms of its connection with general paralysis.[66] By the late nineteenth century, the basic tenets of Benedict Augustin Morel's theory of degeneration were bound up with medical findings that highlighted the coincidence of alcoholism and more serious conditions (including insanity).[67] Prominent alienist Henry Maudsley viewed the insane as the "waste-matter" of evolutionary struggle, whilst groups such as the British Society for the Study of Inebriety began to

explicitly link new models of alcohol addiction with traditional denouncements of deficient moral sense.[68] "[C]hronic soaking" not only "rubbed off … the finer points of moral character and feeling," but could also mark the beginning of the path to serious brain disease.[69]

Many were beginning to ask how far alcohol was implicated in the aetiology of general paralysis, whether as cause, catalyst, or simple coincidence. Forty-eight-year-old grocer's drayman Joseph B., admitted to the West Riding in 1885, was diagnosed with general paralysis with the exciting cause identified as "alcoholic excess."[70] As Farrar noted: "Alcoholic insanity frequently merge[d] into general paralysis" as many general paralytic cases were "the direct result of alcoholic excess."[71] Similarly, Norman Kerr, President of the Society for the Study of Inebriety, said that in his own experience alcohol had been the leading factor in 22% of general paralytic cases.[72] Historian Patricia Prestwich, writing on perspectives on alcohol in nineteenth-century France, has emphasised how alcoholism could be integrated into existing definitions of mental illness, but could also expand that definition:

> … alcoholism provided a powerful example for those who argued for the biological or organic origins of mental diseases and their degenerative nature. In alcohol they found a physical substance whose toxic qualities could be tested in the laboratory, whose destructive impact on tissue could be revealed by pathology, and whose extreme effects, such as *delirium tremens*, bore a strong resemblance to accepted symptoms of mental illness.[73]

That alcohol loomed large in late nineteenth-century debates about health and mental disease is clear, but alcoholism was also—as Prestwich suggests here—aligned with general paralysis as a consequence of laboratory or postmortem examination. Similarities between the two were evident pathologically as well as clinically: Crichton-Browne confessed that there was a tendency among less experienced observers to confuse the general paralytic brain with that of the chronic alcoholic.[74] The effect of alcohol on the human brain had been investigated by a number of researchers throughout the nineteenth century, many of them aligned with temperance reform. Early in the century, John Percy's thesis on the presence of alcohol in the ventricles of the brain made clear the dangers of ingesting excessive amounts of alcohol. Percy related the case of a man brought to Westminster Hospital, already dead, having "drunk a quart of gin for a wager" and whose brain yielded "a considerable quantity of a limpid fluid,

distinctly impregnated with gin, both to the sense of smell and taste, and even to the test of inflammability."[75] Percy's experiments, in which he injected dogs with alcohol to determine how quickly and in what form alcohol reached the brain, led him to suppose that there was "a kind of affinity … between alcohol and the cerebral matter."[76] At the West Riding, the far-reaching effects of alcohol abuse were similarly investigated by observing the effect of the substance on dogs. Bevan Lewis related the case of:

> a dog, to which alcohol had been administered for a lengthened period, [that] not only succumbed to all the symptoms described in alcoholism in animals by [French psychiatrist Valentin] Magnan [such as a savage temper] … but the nutrition of the skeleton also became effected, so that a *notable* degree of mollities and attendant deformity ensued.[77]

Here, Bevan Lewis emphasised the deleterious effects of alcohol on the whole body, not just the brain (note too the reference to mollities ossium, discussed in Chapter "Bone"). He wasted no time in extrapolating these results to the local, human population, attributing the "dwarfed stature of [the] mining community" to drink and noting that excessive drinking was all too common amongst this occupational group.[78]

As a poison that affected the whole body, the expectation that alcohol would leave its mark on the delicate brain matter was clear. Bevan Lewis listed those appearances in the brain of the chronic alcoholic that resembled those of the general paralytic: a cloudy pia mater, atrophy of the brain substance, and anomalous blood vessels.[79] These features can be seen throughout the West Riding's postmortem reports. An 1898 postmortem examination of the brain of a patient who had suffered from alcoholic dementia revealed the dura mater to be "considerably thickened … & intimately adherent universally to [the skull] cap."[80] The spider cell, initially considered an anomaly specific to the general paralytic brain, was also found in the brains of alcoholic patients. Microscopic examination of alcoholic brains had uncovered spider cells "pervad[ing] the upper or outermost region of the peripheral zone of the cortex lying immediately beneath the pia."[81] Like the spider cells in general paralysis, they took on deep staining that was easily discernible with the naked eye. If microscopic appearances really could reveal underlying pathological processes, the presence of these cells in both general paralysis and alcoholism suggested that the two conditions were pathologically aligned.

The similarities between the two conditions were also evident when comparing one organ and another, as Mickle demonstrated: "By the increase and proliferation of interstitial tissue, leading to destruction of … nervous elements, the lesion of general paralysis was held to be brought into line, and to rank, with a cirrhosis of liver or of kidney."[82] This alignment of the general paralytic brain with the cirrhotic liver or the inflamed kidney of Bright's disease was a common one, inspired by "the adherent meninges, the atrophied cortex, the indurated medulla, and the great shrinking of the brain" which in their general appearance were reminiscent of the diseased abdominal organs.[83] Wiglesworth called general paralysis "a true cirrhosis of the brain" like that of the liver.[84] Savage observed that the "bright capillary congestions of the malar bones [cheekbones] … resemble[d] the patches seen in cirrhosis of the liver" and that alcoholics tended to have "the aspect [during life] of persons suffering from cirrhosis of the liver," such as visible blood capillaries on the skin.[85] All of this suggested that the liver and the brain were similarly affected by changes occurring in the body—raising the question of whether general paralysis was dependent on a toxic agent akin to alcohol.

Toxins in General Paralysis

In the late nineteenth century the possibility that toxins entering the body could be responsible for a whole host of diseases was something being discussed by medical professionals both in Britain and further afield. The idea that disease could be caused by toxins made sense in a climate where micro-organisms were generating significant interest. Working from the assumption that most diseases could be explained by the action of bacteria, auto-intoxication theory posited that toxins produced by these bacteria entered the bloodstream and affected internal organs, including the brain. Richard Noll describes two strains of auto-intoxication theory. The classic theory—with its roots in German medical literature—identified the intestines as the centre of the infective process, but evolved in the early twentieth century to focus on the teeth and tonsils as sites of focal sepsis (memorably analysed by Andrew Scull in his 2005 book, *Madhouse*).[86] The other strain of auto-intoxication theory arose with the endocrinology of the 1890s, focusing on the glands as source of infection and disturbance.[87] Here, chemicals secreted internally were the key concern, with over- or under-secretion implicated in disease. Thus, Emil Kraepelin linked dementia praecox—a condition characterised by headaches, delusions, and

hallucinations—with myxedematous insanity (caused by thyroid disease), pointing to enlarged thyroid glands and alterations of the skin as evidence.[88] Indeed, beside their interest in the surface appearance of the patient's skin as discussed in "Skin", many researchers in psychiatry were also investigating the chemical constituents of the skin's secretions. In Daniel Hack Tuke's *Dictionary of Psychological Medicine* (1892), A. Wynter Blyth described experiments in which ear wax and the contents of cysts (both considered by him to be "skin secretions") were analysed.[89] This concern for the chemical make-up of parts or secretions of the body was also evident in regard to other bodily fabric: that the explanation for bone disease could lie in individual strains of bacteria rather than as part of a generally disordered system had been suggested by some bone specialists, for example. Charles Macnamara had pondered the role of impure air in infecting open wounds, thus causing or aggravating bone disease, and some years later William Ford Robertson appealed to the state of the bone marrow as evidence for the bacterial origin of general paralysis.[90] Alfred Mantle, in the 1880s, had implicated bacteria in rheumatism based on his observation that rheumatism and scarlatina tended to be present in the same households, and in 1883 W.D. Miller put forward his "chemico-parasitic" theory of tooth decay.[91] It should also be noted that infection continues to be implicated in delirium today, particularly amongst elderly patients. Though it remains difficult to identify infection as a *primary* cause, a 1997 study of the causes of delirium in 171 elderly patients attributed 73 cases (34%) to infection, a large proportion of which were due to urinary infection.[92]

One of the most commonly investigated of bodily products in the nineteenth century was blood. Studies of the blood of the insane were carried out as early as 1854, when William Lauder Lindsay at Crichton Royal Institution examined "the relative numbers or proportions of the structural elements of blood as counted through microscopic observation."[93] Later research would focus on the specific elements of haemoglobin content and red and white cell counts across a number of mental diseases. Garlands Asylum's S. Rutherford Macphail found a steady decrease in blood quality as general paralysis progressed, so that in advanced cases "the individual corpuscles were … irregular in outline and deformed," but also identified an increase in white blood cells (leucocytosis), suggesting a response to an outside infective agent.[94] Like the investigation of blood flow, the content analysis of blood depended on the use of specialist instruments such as the haemoglobinometer and

haemocytometer, which aided assessment of the colour and quantity of blood cells. The altered state of the blood might also be evident without microscopic analysis, however, flowing sluggishly and making collection of it difficult.[95] The content of the blood, then, was a topic of interest some years before the early twentieth century, when the spirochete causing syphilis was identified and general paralysis was firmly linked with syphilitic infection.

For the late nineteenth- and early twentieth-century doctor, toxins of various kinds were thought to be responsible for mental symptoms such as delirium or acute excitement but were also suspected to have longer-term effects. Chronic forms of insanity depended on a toxin's effect on internal structures—changes taking place over an extended period of time that would eventually become evident to the asylum pathologist. The steadily advancing physical symptoms of the disease, and the varied body parts found to be affected at postmortem, illustrated the movement of the toxin around the body. The 'toxic theory' of general paralysis thus seemed to provide answers to questions that had dogged alienist researchers throughout the last quarter of the nineteenth century—particularly, what had caused such striking lesions in different parts of the body? Making these connections between general paralysis and some form of toxin, researchers found themselves leaning more towards the idea—voiced by several doctors in the final quarter of the nineteenth century—that general paralysis was dependent upon earlier syphilitic infection. As the link between general paralysis and syphilis became more certain, Ford Robertson wrote:

> I would summarise what appears to me the most probable hypothesis regarding the pathogenesis of general paralysis as follows. The disease depends upon the occurrence of a general toxic condition, the exact nature of which is still obscure, but which is certainly in many cases the result of antecedent syphilitic infection. The first important effect produced by the toxins is a proliferative and degenerative change in the walls of the vessels of the central nervous system, including those of the capillaries of the cerebral cortex. This alteration in the capillary walls interferes in various ways with the nutritive exchanges between the blood and cerebral tissues. Consequently the adjacent cortical neurons undergo primary degeneration, and the neuroglia [supporting structure of nervous tissue] also tends to suffer certain morbid alterations. At the same time these tissues are to some extent affected directly by the toxic agents circulating with the blood.[96]

Ford Robertson's comments echoed those of writers such as Clouston, who had surmised that subjecting the nervous system to "imperfectly purified blood" could cause permanent and serious damage to the brain.[97] The toxic theory of mental disease also fit with an existing discourse that emphasised the peculiar bodily characteristics of the general paralytic patient. In 1900, eminent physician Sir Dyce Duckworth noted that general paralysis was by that point "regarded as coming into the category of auto-intoxications" (he did not think that syphilis provided the toxin, rather that it set in motion a degenerative process that released other toxins from the tissues), and in John Macpherson's *Mental Affections* (1899), general paralysis appeared in the section devoted to "toxic insanities."[98] That the bodies of general paralytics harboured a number of corrupting elements seemed evident in their bodily fabric: the sores on the skin, the wasted musculature, the fractured bone, and the softened brain. Toxins explained this range of bodily transformations, as their spread around the body via the bloodstream allowed them to alter almost any organ or structure. Indeed, Ford Robertson argued that the wide-ranging changes to the body seen in general paralysis could only be explained by toxins: "The urine almost constantly displays various important abnormalities. In advanced cases the bones and cartilages always manifest certain gross structural changes which can only be adequately accounted for by toxic action."[99] Ford Robertson was enthusiastic about the potential of the toxic theory in explaining insanity, and led a great deal of work on the subject at the Royal Edinburgh Asylum where he was Pathologist as well as Director of the Scottish Asylums' Pathological Scheme, which had been set up by Clouston. In order to test his toxic theory, Ford Robertson attempted to induce general paralysis in rabbits by adding cultures taken from patients to the animal's food but, as he himself recognised, it was difficult to generalise the reactions and behaviour of rabbits to that of humans.[100]

Similar experiments using animals were undertaken later in the twentieth century by Edwin Goodall. He was particularly interested in questions of bacteriology and toxic theories of insanity, a line of work that he was strongly supported in during his time at the West Riding. In 1890 he was granted a month away from his duties to attend "a course of Lectures & Practical Work in Bacteriology" at Guy's Hospital.[101] By the following year he had apparently "fitted-up a complete Bacteriological Laboratory" at the West Riding.[102] Later leaving the West Riding to take up a position at Carmarthen, then the Superintendency of Cardiff Asylum, Goodall continued to pursue an active research career. Together with Laboratory

Assistant A. Dignam at Cardiff, Goodall experimented with transferring bodily substances from general paralytic and dementia praecox patients into animals, injecting human CSF into rabbits, for example.[103] These tests resembled earlier experiments Goodall had undertaken on human subjects at the West Riding, investigating the efficacy of blistering in general paralysis, and transferring pus between patients. This latter procedure—and the precautions to be taken when undertaking it—was described in an 1893 article, 'The Effect upon Mental Disorder of Localized Inflammatory Conditions.' In this article he recounted injecting patients with pus obtained from boils, hoping to discover whether a bodily reaction might "bring about or accelerate cure in recent and acute forms of insanity."[104] The rationale was to precipitate a bodily crisis, as mercurial treatment had done for many years, as Julius Wagner-Jauregg's fever treatment had attempted to do in the 1880s, and as malarial therapy would do in the early twentieth century. The results of Goodall's tests with the rabbits at Cardiff were inconclusive: 33% of the animals died during the course of intravenous injection, 25% of unknown causes.[105] Goodall's method of causing a somatic crisis was aimed at acute cases of insanity, and thus was not especially well suited to the chronic condition of general paralysis, but there was still room to suspect that—like trepanation—it could provide temporary relief from painful mental symptoms that were dependent on the action of some toxic agent inside the body.

In exploring the possibilities of the toxic theory and locating the toxins that were suspected to be the cause of mental symptoms, many bodily products came under closer scrutiny. This included CSF, which, it was surmised, might carry toxins that were responsible for both the symptoms of general paralysis and the material changes to the brain substance that we saw in "Brain". In the postmortem record of 34-year-old Annie L., the large amount of fluid found in the ventricles of the brain was noted in the section dedicated to the ventricles, where the writer was also instructed to make notes about the choroid plexus (the network of cells that produce CSF). In this case, the choroid plexus was examined under the microscope and meticulously drawn by a staff member, who also carefully noted which objective lens and eye piece ("Leitz No 3 obj. No 4 Eye piece") he had used during his observation (Fig. 1).[106] It is a striking example of the "scientific self" cultivated by nineteenth-century researchers, with the image produced by careful subjective observation in conjunction with the appropriate technical equipment.[107]

The study of CSF during life was aided by the lumbar puncture, a procedure which had been developed in the 1890s. (At the West Riding,

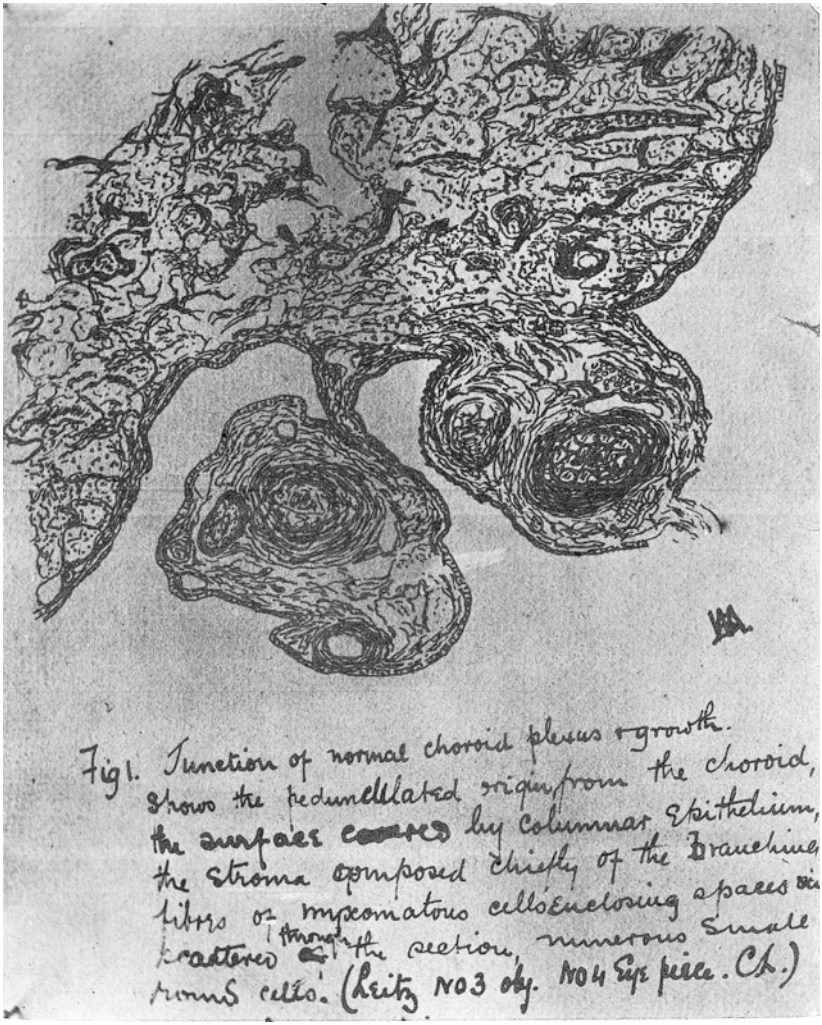

Fig. 1 Sketch of the choroid plexus by one of the West Riding's staff in 1901. Reproduced with permission of West Yorkshire Archive Service: Wakefield and the South West Yorkshire Partnership NHS Trust. WYAS C85/1133

lumbar punctures—by no means a straightforward procedure—were being regularly performed in the 1910s.[108]) A 1901 report from the West Riding also noted that sputum, pus, semen, and urine had been "specially examined" by staff in the laboratory.[109] Urine had been investigated by asylum doctors and others for some time. London physician Samuel Wilks related a case in his 1874 article 'Mania as a Symptom of Bright's Disease,' in which he suggested that the build-up of waste products in the blood due to failure of the kidneys (uræmia) explained the maniacal symptoms seen in some cases of Bright's disease. He described a trial that had taken place a few years previously, when a man on his death bed made a will in favour of his wife:

> … a person beneath him in station, and to whom it was not known at the time that he was married. In an attempt to set aside the will, a *post-mortem* was made, in the description of which a hesitating opinion was given as to the healthiness of the kidneys; whereat the solicitor in the action gathered information relative to the existence of cerebral disturbance in connection with the diseases of these organs, and on which he tried to found an argument as to the soundness of the testator's mind.[110]

Called in to give their opinions, Wilks and a colleague were inclined to agree with the general theory, but believed the symptoms of the man were not in line with such poisoning (whereas he was suffering mere mental 'disturbance' in his apparently irrational wish to leave his possessions to his wife, the more usual signs of uræmia were unconsciousness and coma). Looking back on the case, Wilks said he still could not bring himself to entirely agree with the solicitor, but noted that "in uræmia certain aberrations of mind are occasionally found."[111] If an excess of urea and other products in the blood could cause temporary mental derangement in otherwise sane patients, could it also be at the root of more chronic mental diseases?

At the West Riding in the 1870s, coincident with Wilks' article, doctors were investigating patient's urine both on the wards and in the laboratory. Thirty-nine-year-old John E., diagnosed with general paralysis, was noted to occasionally suffer from retention of urine, which needed to be manually drawn off. In doing this, and in observing John's normal unassisted evacuations, it was observed that his urine contained a "thick deposit," and that he often passed strings of mucus and pus. Subjected to microscopic examination, "granular matter," "crystals," and "pus or mucus corpuscles" were found.[112] Brentwood Asylum's John Turner surmised that the larger

amount of phosphoric acid found in the urine after seizures and periods of excitement was a result of cerebral activity and the flushing out of waste products from the brain.[113] By the turn of the century, examination of the urine was a routine part of admission procedures at the West Riding. James W., admitted in 1901 and diagnosed with general paralysis, had his urine examined in terms of the amount passed, colour, specific gravity, and albumen and sugar content.[114] Goodall, in his post at Cardiff City, investigated the urine of patients for micro-organisms and claimed that, in his own experience, 48% of general paralytic patients had bacteria of various kinds present in their urine.[115] Similarly, Harvey Baird (previously of Cardiff City) examined the urethras of general paralytic and non-general paralytic patients for bacteria, finding "Organisms [to be] … much more abundant in the paralytics."[116] Urine could also be assessed in terms of the amount passed, though this could sometimes prove tricky in the asylum environment: West Riding Medical Officer Ernest Birt noted that even with the use of catheters many patients would accidentally urinate in bed, frustrating his attempts to monitor the quantity of urine.[117]

Like excess CSF as a rationale for trephining, the presence of bacteria in the waste products of the general paralytic body also went on to inform some treatment options, although—like trepanation—their impact was limited. Goodall, working with E. Barton White at Cardiff, examined the faeces of general paralysis patients, noting that "the number of total organisms per gramme of faeces … was above the average normal figure in a large proportion of [the] cases."[118] Goodall and White did not, though, frame this above-average bacterial count as the *cause* of mental disorder. In attempting to get rid of the organisms using orally administered disinfectants, they noted that the treatment had no effect on the patient's mental state. It was almost as if the body of the general paralytic patient was particularly suited to the growth of bacteria. Indeed, in an earlier article, Goodall and Bullen had suggested that "micro-organisms [found] a favourable soil in the degenerate tissues of the paralytic."[119]

Predisposition and Mental Disease

This idea of a favourable, fertile soil suggested that mental disease (of any form) was never entirely unexpected, and that all that was needed was a catalyst (like alcohol or bacterial infection) to start the process. Discussing alcoholic insanity, Bevan Lewis identified a predisposition to insanity in 37% of his male cases, with alcohol acting as the catalyst or 'exciting cause'

in the development of mental disease.[120] Clouston, referring to a patient suffering from the "insanity of Bright's disease," surmised: "No doubt the mental portions of his brain were the weak points of his central nervous system from his hereditary predisposition to insanity and the uræmic poison took effect there."[121] In the case of general paralysis, Sharon Mathews remarks that many late nineteenth- and early-twentieth century commentators on the disease accorded limited importance to heredity; Gayle Davis also notes a relative lack of concern for heredity in the discourse surrounding the condition, and astutely observes that the linkage between general paralysis and late nineteenth-century degeneration theory perhaps looks stronger to the historian of medicine than it did to the contemporary alienist.[122] Heredity did play a part in explanations of general paralysis; though this was more discernible as the turn of the century approached, it was an explanation that built on established ideas about disease aetiology. Charles Rosenberg notes of the early nineteenth century that "both learned physicians and the common man saw disease as the sum of one's transactions with the environment … Hereditary constitutional endowment was one given, the peculiar pattern of life through which that original endowment passed, another."[123] The brain predisposed to insanity had by no means disappeared by the end of the nineteenth century in light of advancing bacteriological knowledge or toxic theories of insanity (indeed, Rosenberg observes that the "most enthusiastic hereditarianism" of c.1885–1920 "coincided with the most enthusiastic and uncritical acceptance of the germ theory").[124] Compare, for example, these sentences written by Adam Addison in 1862, and Edwin Goodall in 1923:

> Addison: "It is quite consonant with our present physiological notions and our knowledge of insanity to believe that some ultimate "irritability", constitutional or acquired, resides in the nervous tissue of the insane, which, on the addition of another link, rapidly developes [*sic*] into disease of the mind."[125]
>
> Goodall: "[There are] several types of neuro-toxic bacterial infection to which the cortical nerve-cells of patients with predisposition to mental disorders show extreme sensitiveness."[126]

Developing bacteriological and neurological knowledge did not push the idea of a 'natural' susceptibility to mental disease from alienist minds, but rather led to its recasting in a new language. Germ theory could not explain why some people became ill but not others; rather than replacing existing

theories, it was drafted into existing accounts in order to provide a detailed, multicausal, explanation of mental disease. That a disease such as general paralysis could have so many varied manifestations suggested a slightly different reaction in each individual affected, according to their heredity, temperament, or predisposition.[127] Though we may be inclined to draw a clear line between the research climate of the 1890s and that of the early twentieth century, "a continuous structure" existed "of what might be called the predisposed body, the body between danger and disease."[128] The constitutional, predisposed, body was vulnerable, not only in its innate excitability, but because it predisposed the patient to even more ailments.

The body of the general paralytic patient around the turn of the century is a fine example of this, and demonstrates how seemingly disparate explanations of mental disease were able to co-exist within one body, and within alienist discourse. The notion of predisposition could complement laboratory findings too, such as Barratt's investigation into the production of false membranes in 1902. His research, funded by a British Medical Association grant, consisted of:

> Portions of subdural membranes varying in size from a pin's head to that of a hemp seed … placed in the subdural space of cats and dogs, being introduced through a small slit in the dura mater covering a cerebral hemisphere which had been previously exposed by trephining.[129]

Examining the animals several weeks later, no progressive changes could be seen—sometimes the implanted membrane had disappeared altogether—leading Barratt to conclude that it was likely impossible that the process of membrane formation could be set up in healthy brains. A similar conclusion was reached by Goodall in his experiments on inflammation: "In cases of general paralysis we may suppose that the vitality of the tissues is so much reduced as to permit the entry (by surface-wound or by the normal passages) and the development of organisms incapable of flourishing in the healthy body."[130] Bacteriological and pathological investigation, then, could prompt researchers to investigate pre-existing differences between people rather than viewing them all as essentially the same and their conditions as an inevitable consequence of bacteria or unknown toxins.[131] Here was another parallel with alcoholism, which some temperance advocates blamed on what they called an "inebriety-centre," a portion of the brain "in which the capacity to crave for, and to be involuntarily impelled to, intoxication reside[d]."[132] The toxic effects of alcohol on the

system did not come about entirely independently, but depended on the supplementary actions of the drinker themselves.

A 1902 address to the Medico-Psychological Association by Clouston also suggests that we should exercise caution in viewing toxic explanations of insanity as signifying a profound shift in contemporary understandings of mental disease. Clouston suggested that there was something amounting to a fashion for toxaemia among younger members of the Association who seemed to exist in an "all-pervading pathological atmosphere."[133] Though he did not dismiss the toxic theory of insanity entirely, he urged caution in embracing it wholesale at the expense of other long-established explanations. Many researchers were keenly aware of the need to focus on hereditary as well as pathological factors. Goodall, who had carried out trepanation for general paralysis as well as numerous other experiments investigating the bacterial basis of mental disease, was a vocal advocate for thorough history-taking in the early twentieth century. This was not simply a personal preoccupation of Goodall's: at the West Riding in the early 1900s, preprinted case files that replaced the more cumbersome casebooks of the previous century demonstrate that family history was still viewed as important. These family histories were much more detailed than they had been previously—especially notable considering the reduced space given to the rest of the patient record in the files—with preprinted sections provided to record information about paternal and maternal grandparents, uncles, aunts, and cousins, as well as parents and siblings.[134] The impulse to collect such information extended beyond the asylum walls: the drive to gather information about heredity and illness grew with the success of the British Eugenics Society in the 1930s. Beginning in 1936, Goodall wrote and talked frequently about the necessity for families to keep pedigrees. Influenced by the Eugenics Society's pedigree-schedules, he called for "discipline" among the population, which entailed the compulsory recording of "maladies, defects, and disabilities" in order to check the spread of mental diseases such as general paralysis.[135]

That Goodall cast his call for keeping detailed family histories in light of the Eugenics Society's practices is a timely reminder—as this book comes to a close—that the social aspects of diseases were not erased by post-mortem or laboratory investigation, and that asylum doctors were not operating in a vacuum without reference to wider social or political ideas. General paralysis was in many ways a prime example of how "the language of disease continually aligns pathological processes with social forces."[136] The anomalous appearances of the general paralytic body—softening of

tissue, atrophy, large amounts of CSF, sprawling spider cells—were stark symbols of waste and decay, and epitomised the nineteenth-century economic conception of health in which the internal balance of the body was paramount. The excessive fluid found in the skull, for example, was a graphic illustration of a breakdown in this balancing act. Pamela Gilbert, discussing Herbert Sussman's work on Victorian masculinity, notes how notions of liquidity or "pulpiness" were used to describe the "unformed masculine self" that required constant and careful vigilance.[137] She suggests that divisions between health and illness were constantly threatened by a liquidity that threatened to "burst out" and "sink [those around] the individual": "wetness and liquidity often ground[ed] descriptions of the body disintegrating as a threat to the larger social body."[138] Certainly from the mid-nineteenth century more attention was accorded to waste—not only the waste produced by the body, but the refuse of the growing city and the classical ruins abandoned by previous generations—and its scientific and cultural "worth" was re-evaluated.[139] In the late nineteenth and early twentieth centuries, the waste products of the general paralytic patient were accorded greater significance as urine and faeces were examined for anomalous bacteria. If waste products are "fundamental to the ordering of the self"[140]—maintaining balance and a system of intake and outgoings—then the general paralytic was a profoundly disordered soul. Urine filled with strings of pus or bacteria-laden CSF suggested that not only was the general paralytic patient suffering at the hands of a cruel degenerative disease, but that they also posed a potential infective threat to those around them.

In considering the 'immorality' of the general paralytic patient who—even before the connection of general paralysis with syphilis—was considered an over-indulger in alcohol and sex who had put excessive strain upon his or her system, this notion of social threat is particularly relevant. The nervous patient of eighteenth- and nineteenth-century Britain, as studied by W.F. Bynum, highlights the "mixture of moral and medical language" that could surround psychiatric conditions at a time when alienists and neurologists were discussing functional explanations of disease.[141] Though functional diseases were real, they could affect different patients in different ways and depended upon a combination of moral and physical factors. In general paralysis, too, there was still room to consider questions of morality as well as physiological and pathological facts. Davis notes that those working on general paralysis drew heavily upon ideas about responsibility and respectability when considering the causes of the

disease. Although the causative factors suggested for general paralysis were numerous, "most alienists … combined shared notions of debauchery and strain in their aetiological explanations of this disease."[142] The general paralytic patient possessed a body that quite literally liquefied and softened as a consequence of failed sexual vigilance or over-indulgence in alcohol. By the final years of the nineteenth century, the link between sexual behaviour and general paralysis was strongly suspected, but not definitively confirmed. In this sense the general paralytic patient bears some resemblance to the transgressive member of the Nuer tribe as studied by Mary Douglas in her seminal work, *Purity and Danger* (1970).[143] The prohibition of incest among the Nuer was bolstered by the belief that any breaking of this rule would be punished in the form of visible skin disease that 'advertised' their crime. Like the potentially infectious general paralytic, the Nuer "polluter" is "a doubly wicked object of reprobation, first because he crossed the line and second because he endangered others."[144] The visible degeneration of the bodily fabric—from the muscles to the brain substance in general paralysis—encapsulated the "fear of collapse, the sense of dissolution, which contaminates the Western image of all diseases," in which the patient becomes the disease anthropomorphised.[145] At the same time the disfiguring transformations of the body and subjective experiences of the sufferer highlighted the patient as victim as well as vector.[146] The reduced physical state and diminished mental acuity of general paralytic patients called for special attention within the asylum—the careful physical handling detailed in "Bone", for example—and also marked them out as interesting from a clinical and pathological point of view.

Bacteriological and pathological investigation could draw upon and confirm social and personal factors in disease, then, but what of such investigation's scientific merit? Berrios is sceptical of the significance of scientific research at the West Riding after the Crichton-Browne era, arguing that work there "came to a premature end … because the scientific methods available then were incapable of producing significant new knowledge."[147] A sense of contemporary frustration is also articulated by Juliet Hurn in regard to the study of general paralysis more broadly: she says that despite the condition being described as "key to the potential of mental science, [alienists] remained painfully aware that their rhetoric was not being translated into theoretical developments or practical benefits."[148] Certainly, discussion of a *cure* for the condition was rare, but the volume of work and discussion on the toxic nature of the disease do not suggest that the discourse surrounding general paralysis was a stagnant one

in the final years of the nineteenth century, least of all at the West Riding. At the same time, we should be cautious in extrapolating findings from the West Riding to other institutions. More so than the other investigations and interventions described in previous chapters, sustained and detailed bacteriological research was possible only in those places with adequate laboratory facilities. The investigation of the toxic theory of general paralysis was to a degree place-specific—dependent on the facilities of institutions like Ford Robertson's Royal Edinburgh Asylum, Frederick Walter Mott's Claybury Asylum, and the West Riding. Similarly, the ability to carry out surgical interventions such as trepanation depended upon staff and operating facilities. Nevertheless, the lively debates about these topics at meetings of the Medico-Psychological Association, or in exchanges within the pages of *The Lancet*, *BMJ*, and *JMS* suggest that this was not a conversation entirely confined within asylum walls.

That general paralysis was not redefined in terms of its aetiology with each new avenue of work did not militate against treatment options, as this chapter has shown, though we may debate their utility. Therapeutic interventions such as trepanation were crucial as general paralysis remained an asylum-based condition into the twentieth century, with patients suffering from the condition taking up a large number of beds and requiring constant care. By this time, outpatient departments and acute hospitals were being built as adjuncts to many of the larger asylums. At the West Riding, an outpatient department was in operation from 1889 and an acute hospital opened in 1900.[149] Like many of the West Riding's activities, these were not entirely separated from the scientific work of its staff. Goodall and Bullen, discussing the West Riding and Whittingham asylums, commented on "the desirability of concentrating attention upon the acute block, and of associated work there by the medical officers and the pathologist" (in the twentieth century, Goodall anticipated that the 1930 Mental Treatment Act would provide a rich source of "material for research").[150] The number of patients utilising outpatient departments in their early years was limited, however, with the initiative largely viewed as "*a stepping stone to the asylum*" for those patients who would otherwise be cared for at home.[151] Cases of general paralysis generally remained cases for asylum treatment proper, and any outpatients diagnosed with the condition tended to find themselves swiftly transformed into inpatients.[152] Despite moves towards intervention beyond the asylum environment, the general paralytic patient remained—along with other chronic cases—a long-term problem for asylum staff well into the twentieth century.

The theories and practices described in this chapter—like those of previous chapters—are significant in highlighting the centrality of scientific work in the asylum, as well as the evolution of general paralysis as a disease entity. They highlight the gradual move away from the fabric of the body towards ideas of external, disease-causing agents. This conception of the disease would be given further credence with the identification of the spirochete as the causative organism in syphilis in 1905 and the discovery of these spirochetes in general paralytic cases in the early 1910s by Hideyo Noguchi and J.W. Moore at the Rockefeller Institute and Central Islip State Hospital of New York. The work of researchers such as Noguchi and Moore, though they may take the credit for definitively demonstrating the link between syphilis and general paralysis, was dependent on several decades of prior clinical and pathological research into the disease. The physiological tests, personal histories, and postmortems that have been described in this book all contributed to the disease's profile, bringing researchers closer to the truth behind the mysterious and fatal condition known as general paralysis.

NOTES

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