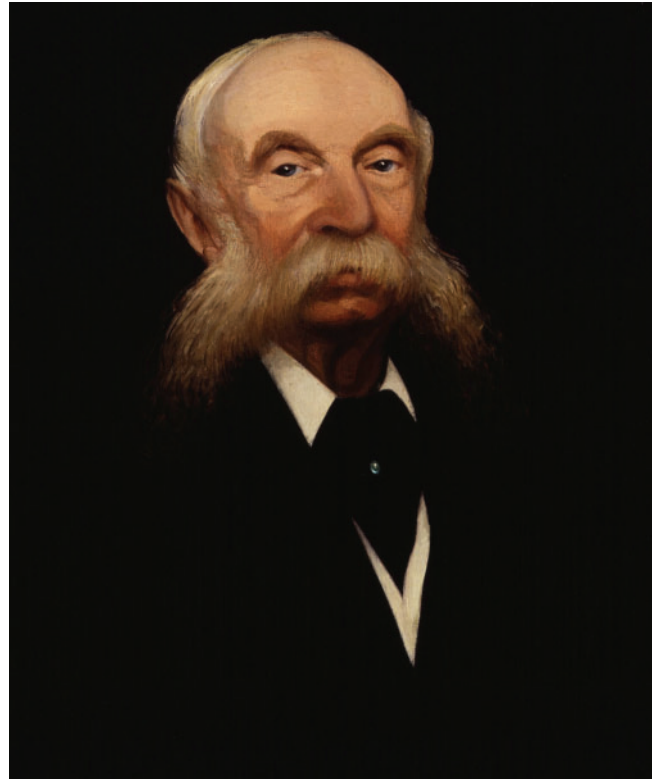


FROM THE ARCHIVES

On the weight of the brain and its component parts in the insane. By J. Crichton-Browne, MD, FRSE, Lord Chancellor's Visitor. *Brain* 1879: 1; 514–518 and 1879: 2; 42–67.

In many respects, the modern study of disordered brain function in Britain has its origins in Wakefield, Yorkshire. This was where James Crichton-Browne turned the West Riding Lunatic Asylum into a research institute that attracted, amongst others, Sir David Ferrier and John Hughlings Jackson. It was a school much influenced by the teachings of Thomas Laycock. The attitude to mental illness in Yorkshire was emancipated. Crichton-Browne promoted the study of neuropathology and the keeping of meticulous medical records. These provided material for the six volumes of *Medical Reports*—a relatively unknown but crucial source of material in the history of neurology. In 1876 Crichton-Browne left Wakefield to take up the position of Lord Chancellor's Visitor in Lunacy. Two years later, he co-founded *Brain* with Ferrier, Hughlings-Jackson and John Charles Bucknill. Born in 1840, Crichton-Browne lived to the age of 98 and even delivered a broadcast on the BBC, founded in 1922.

Writing in volume 1 of *Brain*, Dr Crichton-Browne reminds readers that, under his direction, Dr Crochley Clapham has already reported on the weights of the brains in 1200 insane patients coming to autopsy at the West Riding Asylum. Clapham's methods were primitive. Yet Dr Crichton-Browne has himself been frustrated in the attempt to collect a definitive series, for examination using a more sophisticated method that allows regional weights accurately to be derived, through having had his career in Wakefield cut short at a time when the sample had only reached 400: 'although...my original scheme was thus baffled, and my anticipated wealth of facts arrested prematurely...I had still...collected a store of observations worthy of sifting and arrangement'. The brains here described—from 244 men and 156 women—were collected between May 1, 1873 and May 1, 1876. In most, death was attributed to cerebral degeneration. Examples of tumour or haemorrhage are excluded. 'Removed from the skull by a pathologist...and never by a porter...a few rents were made in the pia mater, and a couple of incisions in the corpus callosum, so that the serous fluid which in chronic lunatics is so often found in large quantity, in the ventricles and sulci of the frontal and parietal lobes, might drain away'. The brain is sliced using landmarks that allow for no reasonable concern on the weight of the constituent parts, with the possible exception of the medulla oblongata. The lower weights recorded by comparison with Mr Clapham (around 22 g lighter) are explained by the greater drainage



Sir James Crichton-Browne by 'Gluck' (Hannah Gluckstein), 1928. National Portrait Gallery, London [NPG 579].

of cerebrospinal fluid, and match those reported by Dr Boyd for the Somerset County Asylum and Dr Thurnam who has already published on 470 patients from Wilts County Asylum. The similarity in the weights of Dr Crichton-Browne and Dr Boyd's specimens is surprising, given marked differences in handling and drainage of fluid: could the explanation lie in the relatively high frequency of organic diseases of the brain in the busy West Riding compared with the agricultural populace of Somerset? Dr Boyd has previously weighed the brains of 1424 adults dying from all causes in the Marylebone Parochial Infirmary in London. Surprisingly, there are no differences in brain weight between these samples. But are they matched? 'The brains of sane paupers... (are) actually... lighter than the brains of pauper lunatics... no doubt the social failures who take refuge in the workhouse are light of brain as well as pocket... either as the cause or the consequence of their misfortunes... many are of

defective cerebral development... but Dr Boyd's tables... have been quoted... as affording a standard of British brain-weights... and have... become the parents... of injustice to the inhabitants of these islands... on the scale of cerebral development'. A trustworthy standard of British brain weights is not to be had from the assessment of inmates of workhouses, prisons, lunatic asylums or general hospitals. Rather, it requires a sufficient sample of individuals who have died suddenly from accidents.

Drs Crichton-Browne, Clapham, Boyd, Thurnam and Skae (newly introduced) agree that the female brain is less weighty than the male, even after adjustment for general bulk and notwithstanding the fact that these are observations made in chronic lunatics in whom wasting diseases of the brain are more prevalent in males than females: 'as to the weight of the brain... in perfectly healthy persons... it will show, I am satisfied, a balance in favour of the male sex of more than 136.2 grammes'. This assessment is consistent with the observation that sex differences in brain weight are least marked at ages when wasting brain diseases, especially general paralysis, predominantly affect males, and greatest at the extremes of life when 'men and women meet on more equal terms as regards their cerebral weight'. And Dr Crichton-Browne ventures the opinion that the relatively small female brain depends 'as Broca has argued, as much on her intellectual as on her physical inferiority'. Generally, the right hemisphere of lunatics is heavier than the left; but wasting of the gyri that accompanies chronic insanity may proceed more rapidly on the right, thereby, for a while levelling up the differences until accelerated atrophy restores the differential. This observation sits uncomfortably with the opinions of Dr Brown-Séguard (not previously mentioned) and Dr Boyd, whose method of removing and weighing the brain leaves him not entirely free from criticism with respect to accuracy, as previously pointed out by Professor Wagner (newly introduced) and the afore-mentioned Dr Thurnam.

Crichton-Browne opens part 2 of his paper by stating the hypothesis that, since it results in 'reduction from a higher voluntary to a lower automatic sphere', insanity may preferentially target those parts 'supposed to be located in the left side of the brain which are most highly evolved'. First, he expects that cases of acute and recent (with respect to death) insanity will approximate most closely to normal regional brain weight. Indeed, the brains of 32 such cases show a slightly heavier right than left hemisphere, as in normal individuals. Again, the acutely manic male brain is heavier than the female—the extremes being J.C., a male youth aged 15 years who died of exhaustion during acute mania having been remarkable for his precocious intellect and powers of acquisition (1605 g), compared with a female, aged 22, who 'sank under pneumonia and erysipelas while labouring under puerperal mania' (1133 g).

Turning to differences in the weight of individual brain structures, Crichton-Browne admits that his dissections are

based on somewhat arbitrary boundaries between lobes. He finds much variation; but 'the cerebellum does not participate to anything like the same extent as the cerebral hemispheres in the loss of substance that is commonly associated with mental diseases'. Indeed, Dr Skae and Dr Thurnam consider it to be heavier in the insane than in normal individuals; and Crichton-Browne shares that view in the context of acute mania. The pons Varolii and the medulla oblongata are also 'apparently invulnerable to senile decay', each attaining their full size at 20 years and rarely dwindling until the 60s or 70s. A detailed table summarizes regional brain weights in the whole series and by gender in the 23 categories of insanity that Crichton-Browne considers. But he senses that the inclusion of cases with 'mental exaltation' has introduced a potential confound, and a better separation would provide weights of 1263, 1344 and 1331 g for the cases with mental weakness (322 cases), exaltation and depression (together, 78 cases), respectively. However, although brain weight is high in pyrexial, simple and acute mania, 'a primary attack of acute madness with subsequent mental excitement of long continuance' is associated with brain atrophy. Many of these cases have died from causes other than insanity, especially phthisis, and these underlying conditions may have contributed to brain wasting. In classifying his cases, Crichton-Browne does not 'trust anything so treacherous as entries in Registers or returns to Commissioners' but has reviewed all the evidence himself: he has not followed Wilhelm Griesinger (1817–1868) in grouping chronic mania with dementia since these cases 'retain a certain limited persistence of spontaneity and mental and bodily activity and the absence of the progressive degeneration... characteristic of dementia.. Table VI must convince us on the most cursory glance that insanity, taken as a whole, and when fatal, is associated with a diminished brain-weight... we have... no evidence to justify the theory that those who are attacked by insanity are of superior cerebral development to the rest of the community who escape mental disease'. Loss of brain weight is most marked in those with developmental disorders resulting in amentia, and then alcoholic disease and general paralysis—especially since the natural history of the latter is readily observed through enhanced survival resulting from excellent nursing care in the West Riding Asylum. The hierarchy of decreasing brain weight, thereafter, is 'consecutive and chronic dementia (subsiding) into the depths of fatuous degeneration'; senile dementia; simple and acute dementia—'cases of sudden and sharp but (sometimes) short-lived mental prostration'; recurrent mania, 'monomania of pride' and 'mania à potu'; and, least atrophied of all, cases of epileptic dementia and melancholia.

The sample size is too small to comment on age-related trends in atrophy within the 23 diagnostic categories of insanity. Across the spectrum of these disorders, insanity in the male has a more striking effect on brain weight, thus levelling out the sex-difference seen normally when brains

are compared between the sexes—‘indicating the much greater severity of the pathological vicissitudes to which the male organ is exposed’. As for regional or focal brain atrophy, it is Crichton-Browne’s impression that, in the insane but presumably also reflecting normal arrangements, the motor areas delineated by Ferrier on the left are larger in men engaged in handicrafts requiring skill and dexterity; and Broca’s gyri are visibly fuller on the left than right. As for regional features of brain weight in the insane, Dr Crichton-Browne’s labours were interrupted after he had examined only 60 brains. The frontal lobes are disproportionately bulky in both sexes by comparison with other brain regions and—together with the occipital but unlike the parietal and temporal lobes—heavier on the right than left: ‘preconceived ideas might have led us to anticipate that these lobes would be found to be lighter in proportion to the rest of the hemispheres in women than in men; but this is not so... it may be that [women] are in some respects less highly developed... less richly convoluted... their grey matter shallower... their ganglionic cells less freely branched... [and] their blood supply less copious... but these lobes are not inferior... in women, to what they are in men’. Crichton-Browne does not describe lobar atrophy correlating with chronic insanity, or with any one category of the conditions he considers separately.

128 years on, what should we make of these observations on brain weight in the context of insanity, made by the founding editor of *Brain*? Ideas are expressed freely on inferiority of the female brain by comparison with the male: Crichton-Browne acknowledges that confounds have been introduced by variations in the amount of cerebrospinal fluid and the degrees of oedema and engorgement in brain samples; he accepts that arbitrary separation of different brain regions makes his regional observations open to error; nevertheless, he is impressed by relatively small inter-hemispheric asymmetries and the modest differences in brain weight observed between cases; above all, he cannot lean on the classical descriptions and classification of disorders resulting in insanity—acute and chronic—since most of these lay ahead. But as a first attempt at attributing mental disorders to abnormal brain structure, Crichton-Browne sets standards for a discipline that now dominates thinking in psychiatric disease and behavioural neurology, and to which papers in the current issue contribute by showing the macroscopic and microscopic features of schizophrenia—best described by Emil Kraepelin as ‘dementia praecox’ in 1898.

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