FROM THE ARCHIVES


‘It seems at first strange, not to say unaccountable, that the centres of sensation and perception are themselves devoid of sensibility or sensitivity, and may be cut, lacerated, burnt, or otherwise injured, without the slightest indication of local suffering being manifested.’ But, as Ferrier goes on to explain, those organs that do give rise to pain when injured, have peripheral end-organs and nerve terminations that are capable of stimulation; and they are connected by nerve trunks to neural centres. Sensation depends on these three elements being in continuity. Thus, there are no sensory ‘nerves’, merely sensory ‘apparatus’ consisting of peripheral organ, nerve and centre. As George Henry Lewes (1817–1878) put it in Problems of Life and Mind (1874–1879) where, whilst allowing some separation, he sought to establish the dependence of mind on physiology and social experience (see pages 1741 and 1951): ‘the function depends on the collocation (sic)’.

Ferrier argues that since the brain substance has no end-organs, injury is not felt. Lesions of the nerve centre are experienced in the periphery where the end-organs are distributed. But, when the end-organs no longer exist, disease of the remaining parts of the sensory apparatus is referred to the periphery in consequence of the organic memory of the nerve centres. Against this background, Ferrier considers how it is that certain diseases of the brain are accompanied by the most excruciating pain felt locally:

‘The headache in many forms of cerebral disease is of such character, so all-absorbing in its intensity, differing much from all other forms of headache, sympathetic, neuralgic etc., and ... because it frequently corresponds with the seat of the disease and may be intensified by local irritation ... we may regard it as certain that the pain is intracranial, and therefore the brain may indicate its disease by local suffering’.

Ferrier has no time for Gabriel Andral’s hypothesis, set out in his treatises on medicine and pathology from the 1820s and 1830s, that the inflamed brain itself might hurt. Having no end-organs, it cannot generate pain. Rather, the origin of pain must be sought in structures other than the brain itself, and the best candidate is the meninges. The debate previously having been settled that the dura mater is indeed innervated by the fifth cranial nerve (Ferrier quotes but does not cite ‘Alexander’ and we have been unable to locate this reference), the French neurosurgeon Henri Duret had already suggested that irritation of these nerves is responsible for pain in traumatic cerebral lesions (On the role of the dura mater and its nerves in cerebral traumatism. Brain 1878; 1: 29–47), localization depending on the density of nerves within the section of stretched membrane. But Ferrier doubts whether meningeal irritation can explain all instances of headache in association with brain disease. Furthermore, the pia mater is generally regarded as insensitive. Its nerves are exclusively vasomotor sympathetic fibres, and electrical stimulation of the pia is painless. Could it be that the vessels have pain sensitive structures within their walls–headache, therefore, being vascular in origin? Certainly, tugging on a ligature applied to the pia, and the symptoms of arteritis and phlebitis seem to support that formulation. Ferrier stresses that headache is often throbbing and in synchrony with the pulse. From this analysis, it follows that headache is most probably associated with conditions in which there is a rapid increase in intracranial pressure or inflammation of the meninges. Each may occur in isolation, or they may co-exist. On the intensity of headache, Ferrier notes that mere engorgement of the pial vessels is an insufficient explanation, since effort and forced expiration rarely cause pain in the head. Explaining the headache of meningitis presents little difficulty when this affects the dura mater, but requires the vascular formulation when considering the symptoms of tuberculous meningitis (primarily affecting the pia) and syphilitic brain disease.

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Both in the context of inflammation and raised intracranial pressure, the tendency will be for pain to reflect the point of maximum distension of the membranes. This feature will more usually localize the seat of superficial than deep lesions. For Ferrier, these considerations seemed to resonate with case series of cerebral abscess and ‘effusions’ in which lesions of the ganglia are painful only when pressure is raised generally. Localization is most exact with lesions of the posterior fossa where, as a result of tentorial stretching, the pain is usually referred to the occiput. Why, in other cases, the ‘lieu d’élection’ is frontal remains unexplained. By comparison with lesions of the posterior fossa, the localizing value of headache in cerebral disease appears to be less good.

Writing at the dawn of surgical treatment for brain and spinal cord tumours (a meningioma was removed by Sir William MacEwen in 1879; the first brain tumour was localized and removed by Alexander Bennett and Sir Rickman Godlee in 1885; and Sir Victor Horsley and Sir William Gowers dealt with a spinal tumour in 1888), yet at a time when there were no investigations to assist the neurologist in identifying the seat of disease, Ferrier advocated the use of skull percussion in localizing brain lesions. In his experience, a tap on the head overlying the responsible lesion often intensified symptomatic headache. Thus,

’a patient under my care staggers when walking and... when the feet are placed together... falls backwards... there is complete deafness in the left ear, partial paralysis of motion and anaesthesia of the left side of the face and loss of tactile sensibility and taste on the anterior two thirds of the tongue. He has occasional pain in the head in the occipital region, with nausea and feeling of sickness... percussion over the left superior curved line of the occiput causes very marked intensification of the headache... the symptoms and the situation of the pain are thus mutually confirmatory of lesion, probably tumour, in the left cerebellar lobe’.

In none of the 11 anecdotes relating to skull percussion, discussed by Ferrier, was post mortem confirmation of the lesion or its location available. Nevertheless, he advocated the more general adoption of head percussion as a means of localizing brain disease ‘with a view to testing its value over a much wider range of clinical and pathological observation than can possibly occur to any one individual’. In his Thomas Vicary Lecture for 1922 (A glimpse into the history of the surgery of the brain), Sir Charles Ballance stressed the importance of Ferrier’s 1879 paper in Brain, and the companion article on vomiting in connection with cerebral disease (Brain 1879; 2: 223–233). More generally, Ferrier the experimentalist, neurophysiologist and neurologist brought a clinical science perspective to the commonest symptom in neurology, and advanced bedside methods to the stage where they could be used by the pioneers of neurosurgery to inform their surgical interventions.

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