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THE NEUROLOGY OF JOHN HUNTER'S LAST ILLNESS

BY

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John Hunter's angina has often been described, but less attention has been paid to the neurological symptoms of his last illness. These were unusual enough to be intrinsically interesting, and they derive an added interest from the fact that the account of them given by Home (1794), John Hunter's brother-in-law, was clearly derived from Hunter himself and includes his own attempt to interpret them.

The First Symptoms

Home, alluding to the year 1776, when Hunter was 48 years of age, writes as follows:

"Towards the end of the spring he was seized with a very severe and dangerous illness, in consequence of anxiety of mind from being obliged to pay a large sum of money for a friend, for whom he had been security, and which his circumstances made extremely inconvenient.

"At two o'clock in the forenoon he eat some cold chicken and ham, and drank a little weak punch; immediately after this he went eight miles in a post-chaise. While he was on the journey he had the feel of having drank too much, but passed the remainder of the day tolerably well; at twelve o'clock at night his stomach was a little disordered, for which he took some caraways, and went to bed; he had no sooner lain down than he felt as if suspended in the air, and soon after the room appeared to go round; the quickness of this motion seemed to increase, and at last was very rapid; it continued for some time, then became slower and slower till the whole was at rest; this was succeeded by vomiting, which was encouraged, and gave him a good night's rest; next day he was tolerably well, but fatigued; the morning after, thinking himself quite recovered, he went out before breakfast, drank some tea and eat some bread and butter, which he was not accustomed to do. At eleven o'clock, he felt his stomach much in the same state as before; in about half an hour, the sensation of the room appearing to turn, recommenced, and continued for some time, but not with such violence as in the last attack; he became sick and vomited; the sensation of himself and every thing else going round, went off, but that of being suspended in the air continued, with a giddiness; he now could hardly move his head from an horizontal position, and about two o'clock was brought home in his carriage, the motion of which was very disagreeable, giving the sensation of going down, or sinking.

"After he went to bed, the giddiness and the idea of being suspended in the air increased, and the least motion of the head upon the pillow appeared to be so great that he hardly durst attempt it; if he but moved his head half round, it appeared to be moving to some distance with great velocity; the idea he had of his own size was that of being only two feet long, and when he drew up his foot, or pushed it down, it appeared to him to be moving a vast way. His sensations became extremely acute or heightened; he could not bear the least light, so that although the

window-blinds were shut, a curtain and blanket were obliged to be hung up against it, the fire to have a skreen before it, and the bed-curtains to be drawn; he kept his eyelids closed, yet if a lighted candle came across the room, he could not bear it; his hearing was also painfully acute, but not so much increased as his sight; the smell and taste were also acute, every thing he put into his mouth being much higher flavoured than common, by which means he relished what he eat; his appetite at first was very indifferent, but soon became good; his pulse was generally about sixty, and weak, and a small degree of heat on the skin, especially on the hands and feet; he remained in this state for about ten days, and was obliged to be fed as he lay; by this time he was rather better; that is, he could move his head more freely.

"When he was first attacked, the pulse was full, and eight ounces of blood were taken away, but this did not appear to be of service; the day following he was cupped between the shoulders, and had a large blister applied upon the part; he took an emetic, and several times purging medicines, and bathed his feet in warm water, but nothing appeared to be of the least use. The purging and vomiting distressed him greatly, for both the stomach and intestines were so irritable, that less than half the usual quantity had the desired effect. He took some James's powder, and drank some white wine whey on account of the heat in the skin, especially in the feet and hands, which took it off, and gave him for the first time a comfortable feel. At the end of ten days all his ideas of his present state became more natural, the strange deception concerning his own size was in part corrected, and the idea of suspension in the air became less; but for some time after the fire appeared of a deep purple red. When he got so well as to be able to stand without being giddy, he was unable to walk without support, for his own feelings did not give him information respecting his centre of gravity, so that he was unable to balance his body, and prevent himself from falling."

A Later Illness

In 1785 fresh symptoms appeared. Hunter began to suffer from attacks which began with "a sensation of the muscles of the nose being in action . . . attended with an unpleasant sensation in the left side of the face, lower jaw, and throat, which seemed to extend into the head on that side, and down the left arm, as low as the ball of the thumb, where it terminated all at once; these sensations were not constant, but returned at irregular times; they became soon more violent, attacking the head, face, and both sides of the lower jaw, giving the idea that the face was swelled, particularly the cheeks, and sometimes slightly affected the right arm." Later they spread to the sternum and were accompanied by severe soreness of the throat and left arm, which he could not bear to be touched. The attacks present some unusual features, particularly their apparent precipitation by mental stress and physical exertion. Nevertheless, there were exceptions to this, and it is

possible that some confusion arose between these attacks and the angina from which Hunter had by then long suffered, since pain in the left arm was common to both.

"These spasms," writes Home, "although they did not increase in violence, were uniformly more frequent, and came on upon a greater variety of occasions; but as he became accustomed to their effects, less attention was paid to them. Nothing particular occurred from this period till about the beginning of December, 1789, in the evening, when at the house of a friend on a visit, he was attacked with a total loss of memory; he did not know in what part of the town he was, not even the name of the street when told it, nor where his own house was; he had not a conception of any place existing beyond the room he was in, and yet was perfectly conscious of the loss of memory. He was sensible of impressions of all kinds from the senses, and therefore looked out of the window, although rather dark, to see if he could be made sensible of the situation of the house; this loss of memory gradually went off, and in less than half an hour his memory was perfectly recovered. About a fortnight after, as he was visiting his patient one forenoon, he observed occasionally a little giddiness in his head, and by three o'clock it was attended with an inclination to vomit. He came home and drank some warm water, which made him vomit severely, but nothing came off his stomach except the water. The giddiness became severe, but went off again about seven or eight o'clock; about nine or ten it returned with more severity, and when going to bed, about eleven o'clock, he had entirely lost the centre of gravity, although he could move his limbs as the will directed; light became offensive, and every thing had a kind of yellow cast; sounds were more acute than natural, objects had lost their true direction; a perpendicular, for instance, seemed to lean to the left, making, as nearly as he could conjecture, an angle with the horizon of fifty or sixty degrees; objects were also smaller than the natural recollection of them; his idea of his own size was that of only being four feet high; also objects appeared to be at an unusual distance, as if seen through a concave glass; he had a slight sound in the right ear, at every stroke of the pulse; motion in his head was extremely disagreeable, he therefore moved with great caution; although coughing and sneezing did not affect it: during this illness Dr. Pitcairn and Dr. Baillie attended him. It is difficult to describe sensations, especially when they are not common; the sensation which he had in his head was not pain, but rather so unnatural as to give him the idea of having no head; with all this, neither the mind nor the reasoning faculty were affected, which is not the case when such effects are produced from liquor. Objects in the mind were very lively, and often disagreeably so; dreams had the strength of reality, so much so, as to awaken him; the remembrance of them was very perfect. The disposition to sleep was a good deal gone, an hour or two in the twenty-four being as much as could be obtained; these symptoms were much the same for about a week, and began gradually to diminish, so that in a fortnight he was able to sit up, and in three weeks went an airing in the carriage; cordial medicines were given, and the body kept open; he felt a pain in the joint of the great toe, which inflamed gently, but soon left it; his pulse was rather increased in frequency, the urine at first was high coloured, deposited a sediment, and was rather diminished in quantity; but the retention in the bladder was very great, as he was not able to make water from ten o'clock in the evening till the same time the next evening, the quantity being very considerable, although not so much as would have been made in the same time had he been in health. The urine became of a yellow colour, and afterwards pale; the stools were solid; the taste of victuals was not impaired, except tea, for which he had no relish. Although he had no particular inclination to eat, yet his appetite was not much diminished. To excite the action of the gout, sinapisms were applied to the feet, but had not the desired effect; in the fourth week the head not recovering so fast as was expected, a blister was applied between

the shoulders, but had no immediate effect, probably did harm, by producing irritation and want of sleep; one night, not having above an hour's sleep, he drank a tumbler full of hot water, which set him immediately to sleep, in which state he continued near four hours; he took a hint from this and drank a tumbler full of hot water every night, just before he went to bed, which did not fail of putting him soon to sleep, and giving him a good night's rest.

"The apparent obliquity of objects he accounted for by supposing that the two corresponding oblique muscles had an unnatural contraction which moved the two eyes round near thirty or forty degrees; we shall suppose that the obliquus superior of the left eye brought the eye-ball forwards towards the nose, while the obliquus inferior of the right eye contracted equal to the superior of the left; this turned the under part of the right eye inwards towards the nose, and the upper part outwards, which moved a lateral part of the eye upon the object, and gave it that obliquity.

"His recovery from this indisposition was less perfect than from any of the others; he never lost entirely the oblique vision; his memory was in some respects evidently impaired, and the spasms became more constant; he never went to bed without their being brought on by the act of undressing himself; they came on in the middle of the night; the least exertion in conversation after dinner was attended by them; he felt therefore obliged to confine himself within a certain sphere of action, and to avoid dining in large companies. Even operations in surgery, if attended with any nicety, now produced the same effects." From Home's account it would seem that the spasms to which he alludes in this passage were anginal, but he does not appear to distinguish the sensory attacks from these.

Hunter died in 1793, and the following is Home's account of the post-mortem examination of the brain.

"On inspecting the head, the cranium and dura mater were found in a natural state. The pia mater had the vessels upon the surface of the two hemispheres of the brain turgid with blood, which is commonly found to be the case after sudden death.

"The internal structure of the brain was very carefully examined, and the different parts both of the cerebrum and cerebellum were found in the most natural and healthy state; but the internal carotid arteries as they pass by the sides of the cella tursica [*sic*] were ossified, and several of the ramifications which go off from them had become opaque and unhealthy in their appearance. The vertebral arteries lying upon the medulla oblongata had also become bony, and the basillary artery, which is formed by them, had opaque white spots very generally along its coats."

Comment

Thus, John Hunter's first neurological illness occurred in 1776, when he was 48 years of age, and was characterized by vertigo and vomiting, disordered sensations of the size and position of his body, general sensory hyperaesthesia, and disturbance of colour vision, the fire appearing purplish. The next did not occur until 1785, when he began to suffer from sensory epilepsy. In 1789 he experienced a transitory loss of topographical memory. This was followed a fortnight later by an illness which in some respects resembled the first one. He again lost his sense of gravity, his appreciation of the size of his body was disordered, and he felt as though he had no head. Once more he was excessively sensitive to sound and light, and disorders of visual perception occurred: everything appeared yellow and objects seemed to be at an unusual distance, smaller than they really were, and rotated to the left. There was also a disturbance of sleep, since he could sleep for only an hour or two in the twenty-four, and sleep, when it occurred, was accompanied by extremely vivid dreams.

The severe vertigo accompanied by vomiting must have been due to a lesion involving the vestibular system, but the associated disorders of perception and of the body-

schema indicate that this lesion cannot have been in the internal ear, but must have involved the vestibular pathways at the cortical or subcortical level. The disorders of the body-schema, which impaired awareness of the size and shape of the body as a whole, the relationship between its parts, and of the whole body to gravity, are of an unusual kind, but are those particularly likely to result from a disorder of the vestibular contribution to the body-schema (Schilder, 1935). There is evidence for cortical representation of the vestibular system in the temporo-parietal region, and this lies near the parietal area, which, in the right cerebral hemisphere at least, is concerned with the representation of the body-schema. The visual symptoms, none of which is very common, are all known to occur after a lesion in the occipital lobe (Brain, 1947; Critchley, 1949). The apparent tilting of objects, a form of metamorphopsia, is not due to an abnormal position of the eyes, as Hunter thought, but to a disorganization of the cortical integration of awareness of visual space, a process in which both vestibular and ocular factors play a part, the latter undoubtedly including unconscious afferent impulses from the proprioceptors of the eye muscles. The appearance of objects as either abnormally small or abnormally distant can also be the result of disordered awareness of visual space, since judgments of the size of objects and their distance are reciprocally related.

Hyperaesthesia to sensory stimuli, while not uncommon in respect of painful stimuli—for example, in the thalamic syndrome—rarely occurs in the case of sight and hearing as a result of a cerebral lesion. One of my patients (Brain, 1947), who had a vascular lesion in the right parieto-occipital region, in addition to suffering from visual hallucinations, micropsia and macropsia, and disorders of the body-schema, complained that sounds appeared much louder than normal. Moreover, she experienced synaesthetic hallucinations, the sound of a car backfiring causing her to see brilliant colours in the left (hemianopic) half of the visual fields. Such symptoms, produced by organic lesions of the parieto-occipital region, resemble some of the symptoms of mescaline intoxication. We do not know how sensory activity can thus irradiate from one sense to another, nor how a general hyperaesthesia of hearing, sight, smell, and taste is caused. Loss of topographical memory is commonly due to a lesion in the occipital lobe, usually in the left cerebral hemisphere, but sometimes in the right.

It is highly probable that the sensory attacks accompanied by pain and hyperalgesia or hyperpathia in the left upper limb were a variety of sensory epilepsy, for Home noted that they always began in the same way, and records that on one occasion Hunter lost consciousness at the height of the attack and had no recollection of it afterwards. They suggest a lesion of the right parietal lobe extending rather deeply into the neighbourhood of the optic thalamus. Similar attacks were experienced by a patient of mine who had a glioma in this region (Henson, 1949).

D'Arcy Power (1931) has attributed John Hunter's symptoms to cardio-aortic and cerebral syphilis resulting from his self-inoculation with syphilis in 1767. The dilatation of the aorta, amounting to an "incipient aneurysm," certainly suggests syphilis, and the description of the intimal surface is consistent with a syphilitic aortitis which had gone on to severe atheroma. The patches of pericarditis might have been due to underlying infarction of the myocardium. But the naked-eye appearances of both his coronary and his cerebral vessels suggest advanced atheroma. It is surprising that no naked-eye changes were detected in the brain. D'Arcy Power points out that the anatomists who carried out the post-mortem examination would have been unlikely to overlook a gross lesion such as a gumma, and infers that the lesions must have been due to endarteritis of the smaller vessels. Whether this or atheroma was the cause, the resulting areas of ischaemic softening must have passed undetected. When we reflect that for the last 17 years of his life John Hunter suffered from progressive disease of the brain, and for the last eight years from

epilepsy, in addition to angina, we may agree with D'Arcy Power that it is "marvellous that he could accomplish so much, crippled as he was mentally and physically."

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REFERENCES

- Brain, W. R. (1947). *Acta psychiat. neurol., scand.*, Suppl. 46, 28.
 Critchley, M. (1949). *Trans. Ophthal. Soc. U.K.*, 69, 111.
 Henson, R. A. (1949). *Brain*, 72, 576.
 Home, E. (1794). In *A Treatise on the Blood, Inflammation, and Gun-shot Wounds* by the late John Hunter. London.
 Power, D'A. (1931). *Selected Writings*. Oxford.
 Schilder, P. (1935). *The Image and Appearance of the Human Body*, p. 94. London.

COMPRESSIVE LESIONS OF THE EXTERNAL POPLITEAL (COMMON PERONEAL) NERVE

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Most adults are familiar with the transient condition of "the foot going to sleep." If a hurried examination is made when this occurs it will be found that the temporary foot-drop results from weakness of those muscles supplied by the external popliteal nerve only. It is a commonplace observation that this event results from such things as prolonged kneeling or, more frequently, crossing one leg over the other while sitting. It would seem certain that it follows temporary ischaemia of the nerve, resulting from compression, and complete recovery in a matter of seconds is the general rule. The student or practitioner is not, however, so familiar with more prolonged states of external popliteal paralysis, and he is perhaps more likely to be puzzled by this fairly common condition than he would be by such disorders as tabes dorsalis, syringomyelia, or myasthenia gravis, all of which are rare.

Should the student wish to amplify his knowledge of the subject he will find little help from British medical writings; there have been remarkably few papers devoted to this subject, and references in current English works appear to be either inaccurate or inadequate. For example, according to Walshe (1952) the aetiology is usually unknown and the paralysis is often preceded by pain; recovery is slow and frequently incomplete, and the condition may result from a "neuritis" in association with diabetes or typhoid fever. According to Brain (1951) the external popliteal nerve may suffer from "interstitial neuritis," and the peronei are usually more gravely affected than the anterior tibial group of muscles. Purdon Martin and Elkington (1950), however, state that paralysis is usually severe, all the muscles being equally affected, subjective sensory changes being usually absent. Purves-Stewart and Worster-Drought (1952) state that the earliest symptom is pain in the relevant area of distribution. Most of these authors make some reference to compression by garters or tight bandages, though Worster-Drought and Sargent (1952) make no mention of pressure as a cause of external popliteal palsy. Gowers (1899) regarded most palsies of single peripheral nerves as being the result of "interstitial neuritis," though he thought that isolated