

Case of General Paralysis complicated with severe Unilateral Epileptiform Attacks, Temporary Hemiplegia, Aphasia, &c. Autopsy. By W. JULIUS MICKLE, M.D., Medical Superintendent, Grove Hall Asylum, London.

George H—, a Quarter master-Sergeant in the 38th Regiment, was admitted into Grove Hall Asylum on the 12th of June, 1874. It was stated that the attack had then been of between one and two months' duration; that it was supposed to be aggravated, if not induced, by tropical climate;* and that he had become epileptic. Previously to his admission he had been in a state of active maniacal excitement, incoherent, noisy, destructive, violent, and the subject of delusions of a grandiose character.

On Admission.—Height, 5ft. 10½in.; weight, 161lbs.; married; æt. 34. Is moderately nourished, possesses a powerful muscular development, and fine soldierly carriage; skin fair, complexion rather pale and sallow, irides bluish-grey, pupils equal, pulse varying from 108 to 120. There are no signs of constitutional syphilis. State of tongue, speech, and gait characteristic of incipient general paralysis. Sometimes he will not answer questions, or only in an angry tone; at others he replies by a furious shout. The irritability is most intense, and he makes frequent violent attacks upon attendants or patients. He declares that he "is Colonel of the Regiment, has £23,000, and is about to marry the daughter of a peer."

Treatment had only the effect of moderating in some degree the excitement, the restlessness, the irritability, the insomnia, and the disposition to outbursts of furious threatening, and of overt violence. In fact, the maniacal symptoms were undoubtedly in part the outcome of the epileptic neurosis, which also manifested itself at this period in occasional nocturnal epileptiform attacks.

To pass rapidly over this portion of his history we need only add that subsequently he had hallucinations of hearing, declared he was in communication with the Deity, and received constant audible telegrams from heaven; that in August he became far more quiescent and tractable, and in September was able to occupy an associated dormitory. The fits were then almost in abeyance. On the morning of Oct. 14, 1874, a succession of severe epileptiform convulsions occurred. When visited he was in one of these attacks, which affected the right side, especially the right eyelids and face, the head and eyes being turned completely to the right

* This is doubtful. He was admitted into hospital on six occasions in India, between 1862 and 1870, for fever, diarrhæa, ague; and for cerebral symptoms in England, in March, 1874.

in clonic spasms; the occipito frontalis was implicated, especially on the right side, and also the right side of the neck, both sides of the abdomen (but more particularly the right); the right pectoral muscles also, but only to a slight degree. He was semi-conscious, and the pulse was small, feeble, compressible. Catheterisation and enemata relieved the loaded viscera, but were required during several days subsequently, especially the enemata. When he was again seen, an hour after the above attack, dextral hemiplegia was present; his replies were quite unintelligible, and he attempted in vain to protrude the tongue, or to raise himself up when requested to do so. The right side of the face was flattened, the right side of the mouth incompletely paralysed, as well as the right eyelids (orb. palp.), nostril, and side of tongue; the right palatine arch was the lower and narrower, and the uvula tip was curved towards the right side. He could swallow. The right arm and leg were much palsied; pupils sluggish, the right one the larger. Temperature—right axilla, 99·5°; left ditto, 99·6°. The convulsive attacks continued for several days (until the 22nd), there being several successions of severe fits every twenty-four hours, besides a number of single convulsions. Throughout he was examined frequently, and full records were made, but only the more salient points need be reproduced here. The convulsive attacks described were some of those which occurred under my own observation. On the 15th the hemiplegia continued; there was moderate conjugated deviation of the head and eyes towards the left side; the respiration was stertorous, but he could be roused from the state of semistupor; and the pupils were dilated, sluggish and equal. A fit was then witnessed the moment after it began. The right side of the mouth and face and the right eyelids were in violent convulsion, the right nostril and face drawn up, the head and eyes turned completely to the right, the occipito-frontalis was moderately affected; the right arm was strongly convulsed, the elbow bent at a right angle, and the sheet seized between the thumb and forefinger; the right leg participated to a less extent, and the abdomen and chest to a slight degree only. As the spasm died away the mouth, eyes, and fingers were still working slightly. Ten minutes later he was noticed in another fit, which was confined to the right side of the mouth and face, the eyes, right forearm and arm. Slight twitchings of these parts persisted for some time after this fit. On the fifth day there was another

attack of hemispasm under observation. It affected the right angle of the mouth more severely than any other region, and died away last at that part. The head and eyes were turned to the right, and the right pupil was the wider of the two. This attack extended to the right arm and leg. A few minutes after it had completely subsided the mouth was again suddenly and violently jerked to the right, as well as the face generally, and the eyelids and eyeballs. But the spasm did not diffuse itself beyond these parts in that instance, although it was of several minutes' duration, as usual. Again, it died away last at the mouth. Temp. 100°. On the sixth day the paralysis was diminished; it was most marked in the arm, less in the leg, and only moderately in the face and tongue. Had had no fits for several hours; the left pupil slightly the larger. Temp., right axilla, 100·9°; left ditto, 100·8°. Next day the patient was drowsy and dull, but was free from convulsion, and the hemiplegia was lessening. Temp., right axilla, 101·7°; left ditto, 101·6°. On the ninth day he was brighter, and the palsy was still disappearing. On the twelfth day he could grasp with his right hand, could walk across the room, but swayed towards the right side, and now and then would have fallen if not supported. In walking the right foot was raised high, the heel passing just above, and close to the left inner malleolus, and this foot was planted in front of the left one, so that his course swerved constantly towards the left in progression; the gait also tended to become hurried, awkward, and confused. Slight tension of the facial muscles was now observed on the right side, and the mouth was drawn a little to the right. Temp., right axilla, 97·1°; left ditto, 97°. The hemiplegia gradually disappeared after this date, and the speechlessness was succeeded by aphasia, partly amnesic, and partly of the nature of heterophasia; the old defect of articulation, the broken, hesitating character of utterance, being also marked. Though he understood simple questions he gave no replies until he was pressed to speak, and the successive answers to the same reiterated query were very different. For example, when thrice pressed to tell his name his replies were as follows:—(1) "My name is right—John." (2) "Man." (3) "Mankin." When requested to mention the number of his regiment he replied at first in an unintelligible jumble of words, and subsequently said, "fifty-eight." He was shown a pencil, and was asked what it was. He said, "What's that for,—regiment,—regiment,—is the,—belongs to you,—to

you,—to you,—now, sir.” Sometimes longer portions of sentences relevant to some subjects were uttered, but when he had found his own name he simply repeated it in reply to every question. It was evident, however, that he understood plain questions. This form of aphasia continued during the rest of the patient’s life. For instance, a few weeks before death he returned three different and irrelevant replies to the thrice repeated question, “How old are you?”—(1) “Long” (2), “eight” (3), “about two nights.” He remained dull and somewhat obtuse, void of emotion, and would stand quietly for hours in one posture if not moved, gazing idly before him, or with his sallow, mask-like countenance bent upon the ground. He was tractable, and readily did what he was desired, so far as his failing power of comprehension permitted him to understand directions. Convulsive seizures broke in upon this condition in January of the present year, but subsided, leaving a transient slight dextral hemiplegia. At the end of February night fits occurred, and he became weaker and bedridden; the fits continued, and on March 11th and 13th he passed into a condition analogous to the status epilepticus, the convulsions affecting the right side only, just as upon former occasions; and he died on March 13th, from the combined effects of the progressive general paresis and the epileptiform attacks.

Autopsy, 80 hours after death.—Permission was obtained to examine the head only, and the description of the morbid appearances is greatly curtailed here. Calvarium not quite symmetrical, the right half appears to be the more capacious in front, but this is not the case at the posterior region, where the right occipital and the posterior half of the right parietal bones have lost their normal outward curve. Numerous vascular foramina give a worm-eaten appearance to the bones along each side of the median line passing through the sagittal suture. The posterior half of the cranium is the thicker; there are bluish areas on the inner surface of the bones from fulness of the diploe; the calvarium is dense, its upper portion removed at the usual level, weighs 16½oz., and is replaced with ease. Dura mater injected, and slightly adherent to the skull; sinuses full of dark clot and fluid blood; sphenoid angle small; no unnatural appearance in the arteries at the base; firm interlobar adhesions, and arachnoid thickened at the interpeduncular space. No thrombosis or embolism discovered in the vessels in or near the Sylvian fissure. The walls of the fourth ventricle have an

uneven, granular, sanded appearance. The vessels are deeply injected, and the meninges unusually thick and tough over the pons Varolii and medulla oblongata, and on section these parts are seen to be hyperæmic, of a lilac hue, and their vessels dilated. Cerebellum pinkish and hypervascular. The veins of the cerebral meninges are gorged with dark blood, except over the anterior frontal regions, where the venous engorgement is much less, especially on the left side. The left frontal gyri are somewhat wasted, and their anfractuositities widened, but only to a moderate degree, and the pia mater covering this part is slightly infiltrated with serum. The wasting involves all the left frontal and the ascending parietal gyri, and the lateral surface of the superior parietal lobule slightly. The membranes generally are thick, tough and highly vascular, and the first two of these changes are *much* more obvious over the superior and lateral surfaces of the *left* side of the cerebrum than elsewhere. The membranes do not strip off readily; they are adherent to the cortical grey matter over certain areas where a superficial layer of the cineritious neurine separates along with them from the prominences of the convolutions. On the *left* side this separation of the superficial grey matter along with the adherent membranes is found in the most extreme degree over the following gyri:—Second external annectant; angular; posterior part of supra-marginal, and of second and third temporo-sphenoidal; the posterior half of the first frontal, and the greater part of the second frontal. It is found in a less degree over the other annectant and the third frontal gyri; still less at the ascending frontal, and absent at the ascending parietal. On the inner surface of the left hemisphere these pathological alterations are well marked at the parieto-occipital fissure, decided, but less, near the anterior extremity, and still less in the frontal and parietal regions above the fissura calloso-marginalis. They are nearly universal on the inferior surface of the left cerebrum, including the orbital gyri where the adhesion is close. On the *right* side the meningeal adhesion and the decortication of the grey matter are absent over the occipital and annectant gyri; slight on some of the parietal prominences; very slight over the temporo-sphenoidal; moderately well marked over the frontal gyri, especially the superior surface of the first frontal, the inner aspect of which also presents a slight amount of the same change. The right orbital surface is not at all implicated.

The entire encephalon is softer than natural, and readily breaks down under moderate pressure, especially the left hemisphere. The layer of grey matter is rather thin on the left side, its strata not very obvious, its larger vessels quite visible to the naked eye, and its colour ordinary. White medullary substance hyperæmic, puncta numerous, vessels dragged from their channels with ease; the vascular injection and dilatation are most marked in the posterior regions, and also, but to a less extent, in and about the left corpus striatum. No special localised lesion of the left third frontal gyrus, or of the insula, or corpus striatum. Weight of encephalon, with the pia mater and arachnoid still investing it, $55\frac{1}{2}$ ozs. After separating the greater portion of these cerebral meninges, the right hemisphere weighs $24\frac{3}{8}$ ozs., and the left $22\frac{1}{8}$ ozs. Cerebellum $5\frac{3}{8}$ ozs. Pons and med. obl., barely 1oz.

Specific Gravity. On the *left* side. Averages. Cortical *grey* matter of tip of first frontal, 1039; of third frontal, $1041\frac{1}{4}$; of ascending frontal, $1041\frac{1}{4}$; of tip of occipital, $1041\frac{1}{4}$. *White* medullary substance of first frontal, $1040\frac{1}{2}$; of third frontal, 1042; of ascending frontal, 1042; near the tip of occipital lobe, $1041\frac{1}{2}$. On the *right* side the grey matter of the ascending parietal gyrus has a specific gravity of 1040, and the corresponding medullary tissue, $1041\frac{1}{4}$. Portions containing about equal quantities of grey and of white substance are also tested. Their sp. gr. when taken from the tip of the right first frontal is 1039; from upper part of right ascending frontal, 1041; from tip of right occipital, 1043.

Microscopical examination of prepared sections. The brain substance, particularly the left third frontal gyrus, does not take the carmine stain well. In the cortical layer of the left third frontal convolution some of the vessels are twisted, dilated and thickened, and deposits are here and there observed between the adventitia and the sheath. In parts there is some proliferation of the nuclei of the neuroglia and vessels. Some of the pyramidal nerve cells are granular, and their nuclei obscured; some are quite loaded, others more healthy. Fatty-like scattered *débris* of granular cells is also observed, and the same appearance is also seen in most of the sections from other gyri. The anterior portion of the left first frontal gyrus stains better. In it only a few of the cerebral vessels are dilated, or tortuous; the pyramidal nerve cells are small; a few are slightly granular or cloudy, with obscured

nuclei. The left ascending frontal gyrus presents much the same appearances as the convolution last mentioned, but a greater number of the vessels are thickened, and have hematoidin and granular deposits between the sheath and adventitia, a few also are shrunken and surrounded by a vacant perivascular space; and the neuroglia is well developed. At the posterior extremity of the left occipital lobe the morbid changes are slight; most of the pyramidal cells are healthy, a few are slightly granular, and there are many oval or rounded cells which exhibit the same slight alteration.

Remarks.—(1.)—*The weight of the brain.* When the brain invested in its two inner tunics was placed upon the scales at the moment of removal from the cranium the weight was found to be $55\frac{1}{2}$ ozs., which is larger than is usual to general paralytics, and falls within the higher limits often found in epileptics. But after the hypertrophied and thickened pia mater and arachnoid had been stripped off to a considerable extent the weight of the encephalon was reduced by $2\frac{1}{2}$ ozs. The exact weight of the cerebrum could not be obtained by the method of Tiedemann in consequence of the partial separation of the superficial layer of the grey cortex along with the meninges. Instances of large brain weight occur now and then in general paralysis, particularly when death takes place in the earlier stages, and it is to be remembered that this patient perished when mental derangement was scarcely of one year's duration. Mr. W. S. C. Clapham* has given the statistics of a large number of brain weights in different classes of the insane, and thus has supplied a void in the masterly paper on the weight of the brain by the late Dr. Thurnam.† The maximum brain weight amongst the 118 male general paralytics mentioned by the former was 58ozs., and the average about $46\frac{1}{2}$ ozs.; whilst the average in 31 male epileptics was about $49\frac{1}{2}$ ozs., and the maximum 56ozs. In the general paralytic, forming the subject of this paper, the brain weight, therefore, was far from rare or unprecedented, though it was unusual, and all the more so inasmuch as softening co-existed.

A more interesting feature was the decided inequality in the weight of the two cerebral hemispheres, that of the left side, the one opposite to the hemispasm and hemiplegia, being $22\frac{1}{2}$ oz., and that of the right $24\frac{3}{4}$ oz.—a difference of

* West Riding Rep., vol. iii., p. 294.

† "Journal of Mental Science," April, 1866.

2½ oz. When weighed separately, each hemisphere had lost a little grey matter by its detachment with the meninges, and the left more than the other; but allowing a few grammes for this, the difference was very considerable, as just stated.* Nor was it an instance of natural congenital difference between the weights of the respective sides of the brain. The lesser weight of the left cerebrum was evidently due to the morbid change, which was more extensive and advanced on that side, and which, amongst other effects, had induced moderate wasting of the left frontal and of some of the left parietal gyri.

2.—*The Specific Gravity.* As in the healthy brain, so in this, the specific gravity of the grey cortex was found to range higher as the examination proceeded from before backwards over the upper convex surface of the cerebrum, though the difference between the anterior and posterior gyri was less in this respect than usually obtains in the healthy cerebrum. The cortical substance in the anterior and middle regions on the left side was thinner, but of slightly higher average specific gravity than that of the corresponding regions on the right side, whilst as regards specific gravity, the reverse condition obtained in the occipital lobes. Dr. Bastian† finds that the average specific gravity of the cineritious neurine is slightly higher in the left than in the right cerebrum, and if this conclusion be accepted, we need seek no further explanation, in our case, of the facts relating to the specific gravity in this respect. This conclusion, however, is by no means universally applicable to the healthy brain.‡ Nor will a consideration of the absolute specific gravity avail us much in this investigation. It is difficult to obtain a reliable standard of comparison, inasmuch as the differences between the statements of various observers are very considerable. While Dr. Bastian gives the average specific gravity of the grey *cortex* in the insane as 1032½, Dr. Bucknill makes it 1037, and Dr. Skæ 1039 ⅓; and the same three writers give the specific gravity of the *medullary* substance,

* Dr. Boyd has stated that the average weight of the left side of the cerebrum is about ½ oz. greater than that of the right side, but Dr. Thurnam mentions that Wagner has denied the preponderance of the left cerebrum. The subject of these notes was right-handed.

† "Journal of Mental Science," January, 1866, p. 493.

‡ In a healthy brain examined by Dr. Crichton Browne it seems that when the grey matter in corresponding situations in the two hemispheres varied in this respect the higher specific gravity was more frequent in the right than in the left one in the proportion of eight to five. Vide "Lancet," Aug. 22, 1874, p. 269.

respectively, as 1040 $\frac{1}{2}$, 1039, and 1042 $\frac{3}{4}$. On examining the detailed records by Dr. Bucknill* the average specific gravity both of the grey and of the white † matter of the brain in his cases of epilepsy and of general paralysis is seen to be above the average specific gravity found by him in the insane generally; and that of the atrophied layer of cortical material in his illustrations of general paralysis more nearly approached the specific gravity of the corresponding medullary regions than is usual in epileptic insanity, or insanity generally.

3.—*The lesions which caused the convulsions.* The presence of the encephalic lesions of general paralysis, the time at which the convulsions made their appearance, namely, just before mental disorder was established; the diffused softening in a comparatively recent case, and the absence of that fibroid induration which is so frequently found in chronic cases of ordinary epilepsy‡ are sufficient to separate the fits in this case from the category of epilepsy proper, and to relegate them to the domain of epileptiform convulsions, such as are often due to some accidental or secondary change. Some slight degree of congestive (false) hypertrophy probably occurred, in the case we are considering, under the combined influence of long-continued cerebral hyperæmia, and of the congestions and vascular distensions engendered by the convulsive attacks, while softening was gradually taking place from the advance of disease on another line, and the tendency was towards atrophy in the more advanced stages, as seen on the left side. None of the induration of old epilepsy was present, nor were the post-mortem appearances in any respect those of "true" cerebral hypertrophy or general sclerosis of the neuroglia.

The lesion which gave rise to the unilateral convulsions was, I take it, that meningo-encephalic morbid change which was much more marked on the left side, was most particularly developed there in the regions of the middle and anterior cerebral arteries, and was a source of "irritation," "over stimulation," or "discharge," of some of the convulsions there situated. So marked a difference between the two sides of the cerebrum, in the thickening and opacity of

* "Psychol. Med.," 3rd Ed., p. 588 et seq.

† This seems contradictory to his own statement on page 602, that the specific gravity of the medullary matter is always diminished in general paralysis.

‡ "Psychol. Med.," 3rd Ed., pp. 581, 582, 625. (The last by Dr. J. Batty Tuke.)

the membranes and in the degree of their adhesion to the cortex, is very rare in general paralysis. Bayle accounted for the epileptiform attacks in general paralysis as arising from "inflammation of the cortical substance, softening of it, and adhesion to the pia mater;" but Westphal* insists "that most violent attacks of convulsions constantly occur (in general paralysis) without any such involvement of the cortical substance—that is, without softening of it and adhesion to the pia mater; and again, this change in the cortex is frequently found where no such attacks have existed." It is to be noted, also, that extensive adhesion of the pia mater and decortication of the grey matter, of the characters present in the above case, though common enough in ordinary general paresis, are extremely rare in ordinary chronic epilepsy. In looking over the recorded necropsies of a considerable number of epileptics (proper) I can only find two or three which presented the compound pathological lesion in question, and in them the change was only slight. But we are now dealing with a totally different group of convulsions, and, reasoning from analogy, it seems that this particular morbid alteration may reasonably be deemed a probable cause of the epileptiform seizures in some, at least, of the cases, where they supervene in the course of general paralysis.

Dr. Ferrier† has recently discussed two instances of epileptic insanity in which the convulsions began unilaterally, and the post-mortem examination revealed softening, wasting, and depression of certain areas of the grey cortex, and, in one instance, adhesion of the pia mater limited to the belt of depression. To account for "convulsions he states that in both these cases it is assumed that the existing lesion gave rise to some degree of irritation of the brain while gradually progressing to destructive action." In short, it is difficult to see why an extreme meningeal alteration of the nature above described should not produce epileptiform convulsions by its direct effect; or, again, by its influence on the vasomotor system, similarly as many a source of peripheric irritation may cause convulsion—a doctrine enforced by Brown-Sequard in particular.‡ What more likely to induce convulsions proceeding from the cortex than the constant irri-

* Translated by Rutherford, "Journ. Mental Science."

† West R. Rept., Vol. iv., p. 51.

‡ "The Physiology and Pathology of the Central Nervous System," Am. Ed., p. 179, et seq.

tation of thickened, hypervascular, adherent membranes disturbing the balance of the cerebral circulation, causing nutrition imperfect in quality and abnormal in rhythm, and making discord, generally, of the harmony existing in health between brain circulation and brain nutrition?

The patient died in the status epilepticus, and so far as the mere condition of the vessels with respect to repletion was concerned, the turgid venosity which was present simply resembled that detailed by Dr. Crichton Browne* as usual to death in the "status:"—the sinuses and vessels engorged, the tissues injected, the punctæ numerous, the vessels covering the pons and medulla oblongata dilated and distended with blood, &c.

4.—*Localisation.* Assuming that the above hypothesis as to the source of the epileptic seizures is correct, it does not appear that the motor phenomena in this case have direct bearing upon the mooted question of the localisation of function in the brain. The convulsions, indeed, were unilateral, and of the side opposite to the most obvious lesion, but beyond this it would scarcely be safe to venture an opinion. The parts leading in the convulsive motor phenomena were—Firstly, the right angle of the mouth and the right side of the face and the eyes; and, secondly, the right arm. In *kind* the spasm was mainly clonic. The *range* of the spasm or convulsion included the right side of the mouth, and the face, eyes, head, neck, arm, hand, and lower extremity, all on the right side; and the bilateral ocular, thoracic, and abdominal muscles were slightly implicated, though not always on both sides. That is to say, it barely reached the second degree† of convulsion beginning unilaterally, in which the fit passes from the unilateral muscles of the side where it begins to the bilateral muscles of both sides. The *order*‡ in which the parts were involved in a fully developed seizure was as follows:—Right side of mouth drawn up in spasm, face and nostril convulsively jerked towards the right side, head and eyes turned to the right; then quickly, and almost simultaneously, the right hand and arm, and the orbicularis-palpebrarum, and the occipito-frontalis came into action; the right

* "Journal of Mental Science," April, 1873, p. 33.

† "St. Andrew's Rep.," Vol. iv., p. 166.

‡ The order of these phenomena was somewhat similar to that in a case by Dr. Hughlings Jackson, where the doubtful inference was drawn that the symptoms depended upon pressure on the healthy left corpus striatum.—Vide "Med. Times and Gaz.," Aug. 15, 1868.

leg, the chest, and abdomen were affected later, and not at all in the slighter seizures. In the most severe attacks moderate spasm remained at the right side of the mouth, the eyes, and the right fingers after the other parts had become quiescent, and finally died away last at the mouth.

Judging by the analogy of Ferrier's experiments, the centres leading in the excitation of convulsions in the case we have described should be:—

For mouth and tongue.—Left ascending frontal g. and posterior portion of third frontal g. (near their conjunction).

For Face.—Left ascending frontal g. near the posterior portion of second frontal g.

For arm.—Upper division of left ascending frontal g. and ascending parietal (and first frontal?).

For side movements of head and eyes and dilation of pupils.—Parts of first and second frontal.

The topography of the extreme degree of lesion scarcely corresponded to this. The parts of the left hemisphere in which there was found at the autopsy the most marked degree of thickening, toughness, and adhesion of the membranes, and decortication of the grey matter, were (enumerated from before backwards) as follows:—Posterior half of first frontal g., greater portion of second frontal; posterior parts of g. supra-marginalis and of second and third tempero-sphenoidal; second external annectant. To a less extent on the inner surface; orbital surface; third and ascending frontal gyri. Add to this a universal slight softening, more advanced on the left side, and slight wasting of some of its gyri. Though the adhesion and other changes just described were less marked over the left third frontal convolution than over some other parts, yet it must be noted that this gyrus was amongst the parts most softened—that it stained less with carmine than the other parts examined, and under the microscope presented pathological changes, as well, or more, marked than were observed in the sections from several other regions. The morbid lesions obvious to the naked eye were, however, spread over a considerable area, and although degeneration had proceeded as far in the third frontal as in any other gyrus, there is no proof that the focus of irritation was mainly seated in it.

Dr. Hughlings Jackson has asserted* that when convul-

* "St. Andrew's Rep.," Vol. iv., p. 166. "Lancet," Feb. 1, 1873.

sions begin unilaterally there are three parts where the fits principally commence : most frequently (1) in the hand ; next (2) in the face, or tongue, or both ; and lastly (3) in the foot. He states that in the simple cases of convulsion " there is well exemplified an important principle which, I presume, applies to all symptoms of the *cerebral series*. . . . The principle is that those parts are wont to suffer first and most which serve in the more voluntary (special) operations, and those last and least which serve in the more automatic (general) operations. . . . The spasm ' prefers, ' so to speak, to begin in those parts which have the more voluntary uses."* Dr. Ferrier describes the convulsions artificially produced by electricity in the lower animals as first invading " the muscles most in voluntary use,"† and also states that " when the irritation starts primarily from any one particular centre, it is the first to be thrown into action, and then the others are discharged, usually in a certain order. The order most commonly observed is that the centres seem discharged from before backwards." And again, " a general irritation of the whole hemisphere manifests itself primarily in the most excitable parts, and these coincide with the most voluntary centres."‡

In the case of G. H— the unilateral fits began at the mouth and face, and quickly involved the arm ; and it would seem as if this was due merely to a picking out of parts high (highest) in the voluntary series ; as if a widely-diffused irritation, or some morbid influence from a wide area, concentrated itself upon the parts where the actions represented were of the most varied, separate, and highly educated character ; as if this morbid influence merely converged to the particular channels into which the normal impulses of the widespread convolitional cineritious area had been most wont to flow in health. I would, therefore, explain the local character of the motor phenomena in this case, not as being due to any (hypothetical) lesion or cause of a strictly circumscribed nature, but upon the same grounds as one would explain the above-mentioned results obtained by Ferrier and others from prolonged *diffused* irritation of the cerebral cortex in the lower animals ; in fact, the analysis of a number of instances of epileptic or epileptiform convulsions beginning unilaterally would incline one to the belief that such attacks may begin

* " West R. Rep.," Vol. iii., p. 316, et seq.

† " British Medical Journal," April 26, 1878, and " Op. Cit."

‡ " Op. Cit.," Vol. iv., p. 60 ; and Vol. iii., p. 88.

(say) in the hand, and be strictly confined to a limited range, and yet the lesion apparently causing them be found at the autopsy to involve very different localities in different cases. It is true that we are told not to state that there is convulsion of a certain group of muscles, but that there is "the sudden development of certain co-ordinations of these muscles,"* and this is a line of inquiry which, while more difficult, promises more exactitude of result—the line of inquiry as to the localities for the leading representation of the most special movements in which each part engages.† But in the case of G. H.— this physiological localisation could not be effected; many centres being implicated, one could not learn the particular order in which the movements evolved were represented in specific portions of the nervous tissue.‡

A minor point is, that in some, at least, of the fits when the pupils widened the right one was the more dilated, and several times it was noticed that the right side of the occipito-frontalis was mainly in action; and that, of the chest and abdomen, the muscles of the right side were those mainly or solely convulsed. Dr. Russell has recorded a case of "hemiplegic epilepsy" in which, with right unilateral convulsions, the only muscles of the trunk implicated were the right rectus abdominis, and right pectorales.§

Now Dr. Jackson describes the spasm in this group of cases as affecting primarily the unilateral muscles of the side in which it begins, and in the next degree as passing on thence to involve also the bilateral muscles of *both* sides.|| The case I have described is rather opposed to this view, so far as it goes. Indeed, in a later paper,¶ he writes with some doubt on this point in referring to cases of unilateral convulsions originating in like manner as occurred in G. H.'s case—convulsions depending on "discharge" of convolutions in the region of the middle cerebral artery, and near to the motor tract.**

5.—*Hemiplegia*.—In range the paralysis was dextral hemiplegic. The distribution of the varying degrees of palsy in the different parts was similar to that of hemiplegia

* Dr. H. Jackson, "Med. Times and Gaz.," Dec. 23, 1871.

† "Med. Times and Gaz.," Aug. 15, 1868, p. 178.

‡ "Lancet," Feb. 1, 1873, p. 164.

§ "British Med. Journal," June 22, 1867, p. 732, et seq.

|| "St. Andrew's Rep.," Vol. iv., p. 166.

¶ "West R. Rep.," Vol. iii., p. 337.

** "Lancet," Feb. 1873, p. 163, and "West Rid. Repts.," Vol. iii., p. 336.

of the ordinary kind. The facial and lingual muscles were, however, considerably involved. The order of recovery from the hemiplegia was that the leg regained power rapidly, the arm slowly, the face more quickly than the arm, but the incomplete right facial palsy was replaced, during the course of recovery, by a state of slight continuous spasm, which excludes the face from further consideration in this particular relation. A fortnight elapsed before the hemiplegia had quite disappeared.

Of what character, then, was this temporary paralysis? Its history was that of epileptic hemiplegia—it was found for the first time after a succession of severe fits affecting the same side; there was slight amelioration whenever any remission occurred in their severity and frequency, and improvement set in steadily when these convulsive attacks ceased. Though the face was more involved than is usual in hemiplegia due to a cause whose locus is the cerebral cortex, yet the general distribution and history of the palsy, in this case, seemed to point to exhaustion of an area of the cortical cineritious neurine, following upon that excessive excitation which produced the convulsive phenomena. Thus, as Dr. Todd says, the undue exaltation of polar force which causes the fits, “induces subsequently a state of depression or exhaustion . . . which will be most upon that side upon which there has been the greatest previous excitement.”* In the case of G. H. the epileptic hemiplegia, although temporary, exceeded the more usual degree and duration. “The presumption is that the degree of palsy depends on the severity of the convulsion, *i.e.*, on the *quantity* of discharge. When the fit is severe, there may be hemiplegia complete in range, except perhaps for conjugated deviation of the head and eyes.”† In the above case conjugated deviation of the head and eyes occurred to some extent, and was further evidence that an extreme degree of epileptic hemiplegia had been reached. The posterior divisions of the (left) superior and middle frontal gyri were amongst the parts deeply implicated, and an abeyance of their function on the left side might be an explanation of this sign, based on Ferrier’s experiments.‡ As Dr. Bastian, however, has recently stated, “conjugated deviation” arises from lesions of various parts of the cerebrum. “Met with

* “Clinical Lectures on Paralysis,” &c., 2nd Ed., pp. 299 and 300.

† Hughlings Jackson, “St. Andrew’s Rep.” Vol. iv., p. 169.

‡ Abstract in “Journal of Mental Science.”

occasionally with lesions in and upon the surface of either hemisphere, this sign occurs with greater proportional frequency as the lesion approaches the cerebral peduncle."* Dr. Bastian† regards the temporary hemiplegias in cases of the epileptic group as due to spasm of the cerebral vessels, plus certain mere molecular changes of a recoverable kind in the nerve elements occurring in, or produced by, the fits. And this damage would be less easily recovered from where, as in G. H.'s case, there were wide-spread degenerative alterations in the minute vessels, and in the nerve elements themselves. Here was found no central lesion, and none of the basal ganglia, to which one might refer the paralysis.

A comparison of the temperatures observed on the two sides of the body would also incline one to the belief that the lesion which produced hemiplegia was of the cerebral cortex. During the period of recurring convulsions the temperature ranged between 99·5°, and 101·7°, but on the twelfth day, when the fits had ceased, the temperature was only 97·1. On comparing the heat at the two axillæ on the first day the paralysed side was found to have a temperature ·1° lower than the other; by the sixth day its temperature was ·6° higher, and on the seventh and twelfth only ·1° higher. These notes accord with Bastian's statement that in hemiplegia from lesion of the cerebral cortex the difference in temperature between the paralysed and the sound side seldom exceeds ·1° Fah., and tends to disappear soon.‡

Baillarger,§ indeed, explained the transient hemiplegias occurring in general paralysis after epileptiform or apoplectiform attacks on the theory of sudden congestions which mainly implicated one of the hemispheres. The persistent hemiplegias sometimes found were, he thought, due to atrophy of the brain on the opposite side, the result either of these repeated unilateral cerebral congestions occurring during the fits, or of a confirmed unilateral congestion of gradual origin. In the case of G. H. the hemisphere opposite to the side temporarily paralysed was undergoing atrophy; its weight was 64 grammes less than that of the other side, whereas Baillarger only speaks of an inequality of from 20 to 62 grammes;|| the cerebral hyperæmia and the

* "Lancet," June 20, 1874, p. 863.

† "Lancet," April 25, and June 20, 1874, pp. 577 and 861.

‡ "Lancet," Nov. 7, p. 1874, 650.

§ "Annales Médico-Psychologiques," 1858, p. 168.

|| Baume cites cases of greater disequilibrium, "Ann. Med. Psych.," 1862, p. 541.

pathological changes in the membranes were more developed on that side, and were associated with a greater degree of cortical degeneration. Had the hemiplegia been permanent in this case it would have seemed to corroborate (*quantum valeat*) the views of Baillarger;* but as has been seen, the motor defect was comparatively transient, and, apparently, was due to a condition of the nervous tissue, the same as, or analagous to, that present in epileptic hemiplegia.

6.—*Speech.* The disorder or absence of speech may have been at first partly of the nature of epileptic aphasia, which is said to occur most frequently in those cases of unilateral convulsions in which the spasm begins at the right side of the mouth or tongue, or both.† When the speechlessness cleared away a variety of aphasia‡ still remained, and as this latter persisted throughout the rest of the patient's life, although for a considerable period he was free from fits, and without a trace of hemiplegia, there is every probability that it was due to those pathological changes in the cerebral cortex found over a wide area, not only in, but also on every side of, the left third frontal convolution.

CLINICAL NOTES AND CASES.

Lesion of Coordination in the Secondarily Automatic or Acquired Functions of Reading and Writing, with Hints of Hemiplegia. Question of Tabes Dorsalis, or Incipient Locomotor Ataxy: Clot in Right Optic Thalamus? By GEORGE SHEARER, M.D. Liverpool.

R. T., æt. 42. Government Ship Surveyor, of active habits, and invariable good health, educated, intelligent, humorous, and possessing considerable mental activity. He is a heavy smoker and accustomed to his beer, but strictly temperate.

While in London, some five years since, he was one of a small band

* Baillarger makes a clear distinction between the two varieties. He does not, as one of his critics seems to imply, attribute all the hemiplegias in General Paralytics with these modes of onset to unilateral cerebral atrophy. "L'atrophie prédominante trouve donc son explication dans ces congestions plus ou moins répétées sur l'un des hémisphères Les auteurs en effet ont admis deux sortes de congestions dans la paralysie générale, les unes lentes et permanentes, les autres brusques et instantanées Les congestions qui précèdent la paralysie générale ou qui surviennent dans son cours sont souvent accompagnées d'hémiplegies passagères En se répétant sur un seul hémisphère ces congestions finissent par amener des hémiplegies persistantes le plus souvent incomplètes."—Op. Cit., pp. 172 and 173.

† "Med. Times and Gazette," Dec. 23, 1871.

‡ "Bateman on Aphasia," p. 104, et seq.