

ON A CASE OF ANÆSTHESIA AND TROPHIC  
CHANGES CONSEQUENT ON A LESION  
LIMITED TO THE GYRUS FORNICATUS AND  
PART OF THE MARGINAL CONVOLUTION;  
WITH REMARKS ON THE SENSORY AND  
TROPHIC CENTRES OF THE CORTEX.

BY THOMAS SAVILL, M.D.LOND.

BEARING in mind the difficulties which often present themselves in observing aberrations of sensation, and the scarcity of uncomplicated clinical observations of this class, the following case seems worthy of being placed on record. Not that the sensory phenomena here were as unmixed and as clear as could be desired; but the symptoms are as uncomplicated as one is likely to find in such cases, which occur for the most part in the aged. The case, moreover, looked at in the light of recent laboratory investigations, assumes an importance which it would not otherwise have. The very unusual position of the hæmorrhage; and the existence of arterial thickening without renal change, are features of considerable interest though they do not call for further comment in this place.

The symptoms, so far as our present inquiry is concerned, observed during life were loss of sensation, and subsequently the formation of bedsores and œdema, on one side of the body. These were found, when death occurred two years later, to be associated with a lesion occupying the grey matter, and some of the white fibres beneath, of the gyrus fornicatus and part of the marginal convolution of the opposite hemisphere.

Richard Sharpe, eighty-two years of age, a locksmith, was admitted into the Paddington Infirmary on the first occasion in May, 1888. His family history presented nothing worthy of note; his parents had been healthy and long lived. His previous history also was unimportant. He had passed a healthy abstemious life, free from gout, rheumatism, or syphilis. He was at work and in his usual health, when, on *May 16th*, 1888, he had his first "stroke." His wife informed us that he was taken suddenly; that he fell down unconscious and remained so for three days; and that on coming round his left arm and leg were weaker than his right.

He was brought to the Infirmary seven days later (May 23), when I saw him for the first time, and made the following note:—"Patient is conscious, but his mind is rather confused and there is some degree of aphasia.<sup>1</sup> *No apparent loss of power in either arm or leg now.* Both patellar reflexes normal. *Common sensation seems to be absent everywhere on the left side and partially on the right, excepting the area around the right elbow joint.*<sup>2</sup> Tongue protruded straight. Incontinence of both urine and fæces. Back and hips very sore—more on the left side, on which side there are superficial sloughs in places. No signs in heart or lungs. Urine, acid, 1018, faint trace of albumen."

The mental condition rendered the examination somewhat difficult, requiring time and care, but I made the examination, and this note myself, and distinctly recollect that, whereas the absence of response to touching and pricking on the whole of the left side was absolute and beyond doubt, there were places here and there on the right side, besides the right elbow where he could feel. It may not be out of place to remark that testing sensation in very old, and in very young subjects is always a little difficult; in the former, partly owing to the slowness of mental faculty and partly owing to senile changes in the skin. It is not, of course, sufficient to trust to reflex

<sup>1</sup> "Forgetfulness of Words" would have been a better phrase, owing to the confusion of thought.

<sup>2</sup> Where sensation was perfect.

phenomena. Patience and sufficient time are above all things needed.

*May 31st, 1888 (one week later).*—"Much improvement, both in mind and body. Sensation has returned everywhere, he has no incontinence now."

*June 14th.*—"Has suffered from headache for the past two days; was sick yesterday. Gets up daily."

*June 21st.*—"Is still improving. Swallows quite well now. Pupils unequal, right larger than left. (He had dislocation of the left lens from an accident early in life.)" The temperature was 99·8 on admission, but afterwards it was about normal throughout his two months' stay in the Infirmary.

He made a complete recovery, and was able to leave the Infirmary and return to his work two months after admission (July 18th, 1888).

It was upwards of a year before I saw him again, but I remember the case distinctly on account of its unusual features, viz., a combination of (1) Anæsthesia, (2) One-sided bed-sores, (3) Confusion of thought and (4) Double incontinence, without motor paralysis. During the ensuing year he kept in good health, except occasionally, to use his own words, his "senses would go away from him all of a sudden while at work."<sup>1</sup>

In the summer of 1889, these attacks became worse, and he was also subject to transient attacks of giddiness and syncope, in one of which he fell down in the street, and was taken into St. Thomas' Hospital, where he remained for a short time. I have been unable to find any record of his symptoms at that time; but the patient said that he was only in the hospital a little while, chiefly on account of his "fainting" attacks, and had no other symptoms. But after leaving he found that his memory was failing him, and his

<sup>1</sup> These momentary, transient confusions of thought are very frequent in the aged, and without entering into a discussion of the proofs, I may perhaps here state my adherence to the view that they are due to disturbance of the cerebral circulation. They occur most frequently in subjects with thickened systemic arterioles, a condition which existed in this case to a very marked degree. The case is further alluded to in this connection in a paper on Senile Cardio-vascular Changes, read before the British Medical Association Meeting, at Bournemouth, July, 1891.



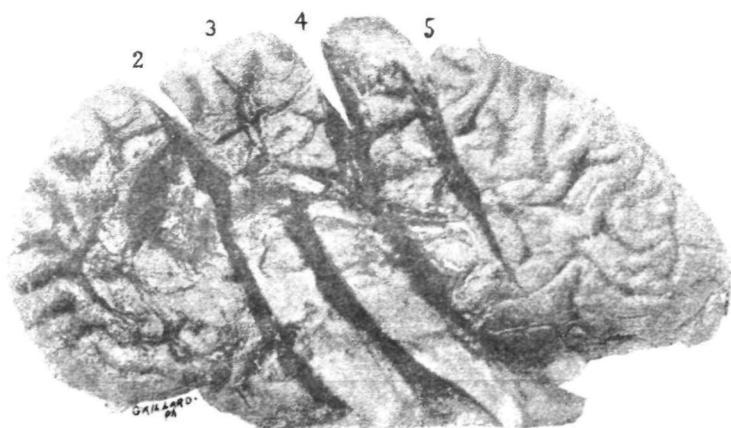


FIG. 1.

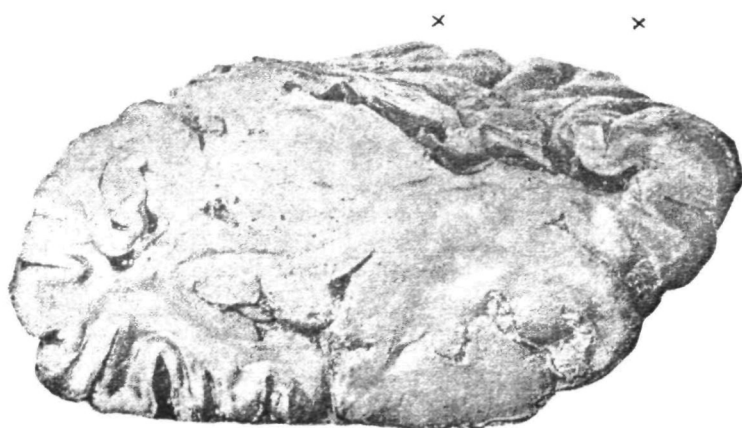


FIG. 2.

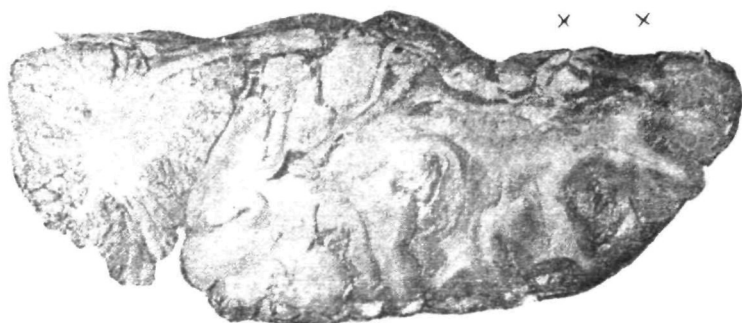


FIG. 3.



FIG. 4.



FIG. 5.

#### DESCRIPTION OF PLATES.

FIG. 1.—Right hemisphere of Richard Sharpe. The front end of cavity is propped open by a glass rod. The numbers are opposite the sections from which the subsequent figures are taken.

FIGS. 2, 3, 4 and 5.—Vertical transverse sections through the right hemisphere as indicated in Fig. 1, seen from behind; Fig. 2, obliquely across fissure of Rolando; Fig. 3, obliquely across ascending parietal convolution; Fig. 4, obliquely across mid-parietal region; Fig. 5, obliquely across anterior occipital region. Crosses are placed opposite the superior and inferior limits of the cavity.



hands were not so adept at his work as formerly ; so he was obliged to seek refuge in the Paddington Workhouse, where he again came under my care, and the case was classed as one of (so-called) arterio-capillary fibrosis, or arteriole hypermyotrophy, as I prefer to designate this condition.

Whilst in the Workhouse, on January 25th, 1890, he was seen to fall. He was not unconscious at that time, and explained that he was too giddy to rise. When examined an hour or so later he was found to be in a semi-conscious condition ; he could be roused to answer questions, though not very intelligently, and there was some stiffness of the left arm and leg.

*January 29th, 1890 (four days later).—* The stiffness had passed off, and there was “ complete flaccid paralysis of the left arm and leg. Can speak and understand a little now ; does not articulate well ; is not sufficiently collected to test sensation. Left side of mouth does not move, tongue protruded very much to left. Can shut both eyes. Swallows with difficulty ; fluids run out of mouth. Yawns very frequently, and when he yawns, the left (paralysed) arm goes up towards his head. Knee jerks normal on both sides ; complete double incontinence ; heart and viscera normal ; arteries very thick. Temperature last night 99°.”

*February 28th, (thirty-fourth day after the attack).—* “ Has a slough forming on left heel and left buttock (paralysed side), although he does not lie more on this side, and is most carefully nursed. Right side sound. Left leg becoming contracted.”

*March 7th (forty-first day).—* “ Has had marked œdema of left hand, leg and foot (paralysed side) for the past week—more of the hand than foot. No œdema of right extremities. Heart sounds healthy but feeble. A few râles at basis of lungs.”

*March 14th.—* “ Both legs are becoming more drawn up, and great tendency to bed-sores on bony prominences, chiefly on the left side.” The urine was examined from time to time, and always found normal till towards the end of life it became alkaline, ammoniacal, and contained pus. The temperature was normal until the occurrence of cystitis.



In the latter part of April he became drowsy, and wandered at times. The weakness increased, and he died very gradually, apparently of asthenia, without the appearance of any fresh symptoms, on May 1st, 1890, two years after the first seizure, and three months after the last. These are the main points in the clinical history so far as our present inquiry is concerned.

*Autopsy*, fourteen hours after death.—Serosus cavities healthy. *Heart*,  $10\frac{1}{2}$  ozs. (after removal of clots). Aortic, mitral, and other valves normal; marked hypertrophy of left ventricular wall (average  $\frac{7}{8}$  inch. thick); muscular substance firm and under the microscope normal.

*Lungs*—Emphysematous.

*Spleen*.—3 oz. ; substance rather firm and capsule thickened : substance pale, and the vessels stood out from the cut surface.

*Liver*— $43\frac{1}{2}$  oz. ; normal.

*Kidneys* (right 4 oz., left  $5\frac{1}{2}$  oz.) were about the normal size and consistence. The cut surface showed very slight atrophy of cortex ; vessels stood out and remained patent. Capsule slightly adherent ; microscopically, only very slight increase in the interstitial tissue (such as is normal in old age), and the epithelium was healthy, but the arterioles of the kidney, and elsewhere throughout the body, showed a very great increase in the thickness in their walls. This increase was chiefly in the tunica media, slightly in the tunica adventitia also. The increase was due to hypertrophy of the muscular tissue, for by leaving sections in staining fluid for 18-24 hours, the characteristic rod-shaped nuclei could be brought out very distinctly.

*The Brain*, after being hardened in Müller's fluid for several weeks has been submitted to careful examination. The right hemisphere, when fresh, weighed 24 oz., the left  $25\frac{1}{2}$  oz. There was some obvious flattening of the convolutions of the right hemisphere, especially on the superior aspect, along the margin, and in the frontal region. By making a series of vertical transverse sections as shown in the illustrations<sup>1</sup> an elongated, rough walled cavity was

<sup>1</sup> Executed from photographs which Professor Victor Horsley has kindly had made for the author.

discovered, measuring nearly four inches in length, stretching from before backwards, immediately beneath the median aspect of the hemisphere. (*See Plates.*) The cavity is such as follows a hæmorrhage of old date, when the patient survives two or three years. Its walls are very ragged, but not crossed by any trabeculæ or bands. The lesion is strictly localised, and is situated precisely beneath those parts of the cortex corresponding to the gyrus fornicatus and part of the marginal convolution, in their entire extent, and also beneath the anterior half of the quadrate lobule (præcuneus). The cavity stretches from before backwards over the roof of the right lateral ventricle, from which it is separated in places only by the ependyma, without nerve tissue. The cavity is largest at its anterior extremity, where its vertical diameter measures  $1\frac{1}{4}$  in., midway 1 in., and at the back it fades away to a point. In the fresh state, it measured nearly four inches in length, antero-posteriorly, and its sides, laterally, were almost in apposition, separated only by a little serum. The median wall of the cavity, formed by what is left of the convolutions just named, is very thin. Thus the grey layer of the Gyrus Fornicatus in its anterior half only measures  $\frac{1}{12}$  in. thick, and here there is no white matter at all; in the posterior half of the gyrus fornicatus grey matter  $\frac{1}{7}$  in., white layer  $\frac{1}{12}$  in. In the marginal convolution anteriorly, grey matter  $\frac{2}{3}$  in., white matter  $\frac{1}{10}$  in.; posteriorly, grey matter  $\frac{1}{8}$  in., beneath which is only a faint streak of white fibres. In that part of the quadrate lobule where the cavity is found, the grey matter is  $\frac{1}{8}$  in. thick (which is about normal) and white fibres  $\frac{1}{10}$  in. thick. *All direct communication between the gyrus fornicatus and the white matter beneath is entirely cut off.* There are no trabeculæ stretching across the cavity. The cavity in front extends to within  $\frac{1}{2}$  in. of the vertex of the brain, so that in this position also *all direct communication* between the marginal convolution and the centrum ovale are cut off. The tail of the cavity beneath the quadrate lobule is very small, and not sufficient to sever communication between that structure and the white fibres beneath.

The frontal, central, parietal, and occipital convolutions

and the uncinate and hippocampal gyri of this hemisphere, and all the convolutions of the other hemisphere, are quite healthy. The cerebral meninges are somewhat thick. The cerebral arteries are atheromatous, but do not present the hypertrophy of the middle coat found in the systemic arteries. By close inspection of sections through the brain in various directions, numerous minute cavities are to be seen in the white matter of both hemispheres about the size of a hemp-seed or less. These are apparently the results of miliary softenings. Situated in the right internal capsule is a localised softening of more recent date about the size of a small split pea, obviously the lesion producing the "stroke" three months before death. But this lesion is scarcely large enough to account for all the symptoms present during the last three months of life, which included amongst others transient rigid then complete flaccid left hemiplegia followed by contracture of both lower extremities; and it seems more than probable that inflammatory oedema around the old lesion produced some of the later symptoms, and contributed to a fatal issue. The greater extent of the symptoms on the second occasion would be in keeping with an inflammatory lesion, whose extent would be greater and less well defined than a hæmorrhagic lesion.

It is to be regretted that the spinal cord was not preserved.

#### REMARKS.

There can be no doubt from the foregoing account that the symptoms from which the patient suffered in May, 1888, were due to a hæmorrhage into the right hemisphere just beneath the median surface in the position of the cavity which was discovered after his death two years later. It is a matter for sincere regret that the boundaries of the anæsthesia at that time were not observed with greater exactitude. But those who have tested sensation in the aged will readily appreciate the difficulties encountered, and will share my satisfaction that certain facts were made out. It is, however, quite certain:—

(1) That this patient had completely lost the power of perceiving when the left side of his body was touched or pricked seven days after the hæmorrhage, which cut off all communication between the right gyrus fornicatus, marginal convolution, and anterior part of quadrate lobule with the parts beneath.

The condition of the sensation on the right side is not quite so clear. There was an area on the right arm where sensation was intact, but over the rest of that arm, the right leg and the trunk on that side, the sensation is noted as being imperfect. The tumefaction of the right hemisphere at the time of the hæmorrhage must have been very considerable, and there would undoubtedly be considerable pressure on the hemisphere of the opposite side. Is it possible that this pressure, which would take effect mainly on the corresponding convolutions on the median aspect of the left hemisphere, could give rise to impaired sensation on the right side of the body, if it be conceded that the gyrus fornicatus is the centre for sensation of the opposite side of the body? The anæsthesia after the initial attack passed off in the course of fourteen days.

The mental condition after the second attack did not permit of an investigation of the sensation.

(2) That when he came under observation on the first occasion, seven days after the first stroke, and the anæsthesia was noted, there was no paralysis.

(3) It is also quite certain that although there were abrasions on both buttocks, nevertheless sloughs formed only on the left buttock, none on the right, almost immediately after the initial seizure. The same phenomenon occurred after his second seizure (eighteen months later) when sloughs not only formed on the left buttock, but also on the left heel, and in spite of the most skilful nursing; the right side not being affected until a much later stage of the case.

(4) Soon after the second stroke another, kindred, phenomenon presented itself in the shape of very marked œdema of the left hand and foot, in the absence of heart, renal<sup>1</sup> or

<sup>1</sup> It will be remembered that the examination of the kidneys after death confirmed this, a fact of great importance.

liver disease; the right side again remaining free. This condition remained till death.

The history of a transient left hemiplegia, when he recovered from three days' "unconsciousness," was obtained from his wife. Although we were unable to verify this ourselves, it is quite likely that it should be so for a small part of the motor area of the cortex, which extends on to the median aspect, must have been involved in the lesion, as well as that part occupying the vertex.

In a case of hemi-anæsthesia with other symptoms, published by Dr. Sharkey,<sup>1</sup> the extent of the lesion, judging by the illustration, was probably deep enough to involve the fibres coming from the grey matter on the median aspect of the hemisphere. Dr. Allen Starr<sup>2</sup> has collected a series of forty-one cases showing cortical lesions with sensory symptoms; and Dr. C. L. Dana<sup>3</sup> has collected 142 cases; which are admirably criticised by Dr. C. K. Mills.<sup>4</sup>

Munk, Luciani, and others hold that the area for common sensation lies in the central region of the cortex, that part beneath the parietal bone.

In some valuable laboratory researches made by Horsley and Schäfer<sup>5</sup> the conclusion arrived at is that the centre for painful and tactile sensations is in the gyrus fornicatus. There is a very striking similitude between many of the experimental lesions made by these observers, and the pathological lesion in the case under consideration. Taken in connection with these experiments the facts of this case become of much greater interest.

Dr. Ferrier<sup>6</sup> says, "I have maintained—and a similar opinion has been expressed by Charcot, Nothnagel, &c.—that there is no necessary connection between cortical lesions of the motor zone and affections of sensibility; and I am further of opinion that the motor and sensory centres are anatomically distinct from each other, though func-

<sup>1</sup> *Med. Chir. Trans.*, 1884, p. 265.

<sup>2</sup> *Am. Jour. Med. Sci.*, 1884, vol. 87.

<sup>3</sup> *Am. Jour. of Nerv. and Ment. Dis.*, Oct., 1888.

<sup>4</sup> *BRAIN*, part 47, p. 385.

<sup>5</sup> *Transactions of the Royal Society*, vol. 179 (1888), B., pp. 1-45.

<sup>6</sup> *BRAIN*, part 45, p. 38, July, 1889.

tionally and probably organically connected together. Others (Exner, Luciani, &c.) hold that the sensory and motor centres coincide, and believe that cortical motor lesions affect common sensibility as well as motion." The case under consideration goes a long way to settle this question in favour of the first-mentioned observers.

But Ferrier in his earlier experiments<sup>1</sup> found that destructive lesions of the cornu ammonis and gyrus hippocampi caused impairment or abolition of tactile sensibility on the opposite side of the body, and located the cortical centres for this form of sensibility in the hippocampal region. In experiments with Professor Yeo in 1884, he confirmed these results, but did not produce permanent anæsthesia.

Professors Horsley and Schäfer remark in conclusion:<sup>2</sup>—"The results of our experiments upon the limbic lobe<sup>3</sup> seem to point to the conclusion that this portion of the cerebral cortex is largely, if not exclusively, concerned in the appreciation of sensations, painful and tactile. This is an extension of the view put forward by Ferrier, who was inclined, as the result of his own experiments, to limit that function to the hippocampal region. Dr. Ferrier, who was good enough to assist at some of our experiments upon this part, has fully accepted the conclusions to which they point.<sup>4</sup> These conclusions appear, however, completely incompatible with the views of most of our predecessors in the field of experimental cerebral research."

The question is undoubtedly beset with considerable difficulty. It is in the belief that every contribution tending to throw light upon the subject, however small, is of value, that I have been induced to publish this case.

Bearing in mind the more permanent character of the trophic and vaso-motor lesions (sloughing and œdema),<sup>5</sup> the

<sup>1</sup> Quoted by Dr. Charles K. Mills, *BRAIN*, part 47, p. 380.

<sup>2</sup> *Loc. cit.*, p. 23.

<sup>3</sup> The limbic lobe in the monkey corresponds morphologically with the gyrus fornicatus and gyrus hippocampi in man (Broca).

<sup>4</sup> "Functions of the Brain," 2nd edition, pp. 341 *et seq.*

<sup>5</sup> The sloughing of the buttock after the first "stroke" (May, '88), passed off in a week or two, soon after the anæsthesia had cleared up. But the sloughing which occurred on the left buttock and heel shortly after the second "stroke" remained till death, two months later. The œdema of the left arm and leg came on about March 1st, and remained until death on May 1st.

case would seem to throw even more light on the centres for these influences than on those for common sensation. I am informed that both Professor Schäfer and Dr. Bradford have observed vaso-motor phenomena to follow experimental excitation of the limbic lobe in monkeys, but I do not know where these results are published. It will be remembered that the trophic symptoms after the first, or apoplectic attack, were only transient, just as the irritative effects of the hæmorrhage were transient; but after the second (inflammatory) lesion the trophic phenomena were both more severe and more permanent, just as the lesion itself was more irritative, more lasting, and fatal.

In conclusion, I submit:

(1) That this case supports the experimental evidence that the gyrus fornicatus, is the centre for common or tactile sensation on the opposite side of the body.

(2) That this loss of sensation, in man, may be quickly recovered from, *e.g.*, in the course of fourteen days, presumably by a process of substitution of centres, the other hemisphere taking on the functions of the parts destroyed; as is known to occur in the motor centres; such substitution in the case of sensation taking place more quickly than in the case of motion.

(3) That a destructive lesion in this position (gyrus fornicatus) may produce loss of sensation without involving loss of voluntary motion, at any rate, to any serious extent.

(4) That a destructive lesion in that position is attended by vaso-motor or trophic changes, of a more permanent character than the sensory changes, in the skin and subcutaneous tissues on the opposite side of the body; and therefore these convolutions may possibly be the centre not only for sensation but also for trophic influences transmitted to the opposite side of the body.

The trophic changes affecting the skin in my case were the sloughs which occurred during the first attack in May, 1888, from which he recovered, and the sloughs and œdema of limbs which occurred after the second attack in January, 1890, from which he did not recover. The latter may

have been due either (1) to a permanent interruption of the trophic paths by the apoplectic cavity at the time of the first attack, or (2) to the irritation set up by the inflammatory œdema around the cavity at the time of the second seizure.