

## THE REPRESENTATION OF THE FUNCTION OF VISION IN THE CEREBRAL CORTEX OF MAN.<sup>1</sup>

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ON May 8th, 1883, I read a paper before the Royal Medical and Chirurgical Society on "A Case of Asymmetry of the Brain presenting peculiarities which bear upon the Question of the Connexion between the Optic Nerves and certain Definite Areas of the Cerebral Cortex."<sup>2</sup> The case was one of congenital spastic hemiplegia, and the most striking peculiarities of the malformed half of the brain were as follows:—(1) The general arrest of development of the left hemisphere; (2) the small size of the corresponding crus cerebri and anterior pyramid; (3) the absence of the angular gyrus and superior temporo-sphenoidal convolution, together with fusion of some of the other convolutions of the left temporo-sphenoidal lobe; and (4) the small size of the optic tract, optic thalamus, and corpora geniculata on the same side. I might have added, I now think, that the angular gyrus in the healthy hemisphere appeared to be unusually large. In commenting on this case I wrote as follows: "Some physiologists have concluded from their experiments that the occipital lobe contains visual centres, and of late Ferrier himself has inclined to this view. The present specimen tells neither for nor against such a theory, for the slight reduction in the size of this lobe cannot be made responsible for the extreme atrophy of the optic tract, though it is possible that the remnant of the latter which still exists may owe its survival to the presence of the occipital lobe. The case is different, however, with the angular gyrus and temporo-sphenoidal lobe. Ferrier's experiments have raised a very strong presumption in favour of the theory that the angular gyrus has some special function in relation with sight, some intimate connexion with the optic tract. And we hold that the specimen under consideration affords anatomical confirmation of this view. Setting aside the central convolutions and the degenerate motor strands in relation with them, the rest of the left hemisphere, with the striking exception already mentioned, presents only such general slight diminution in size as will explain any inequality which may exist between the nerves proceeding from the two sides. But the angular gyrus and temporo-sphenoidal lobe on the one hand, and the optic tract, optic thalamus, and corpora geniculata on the other, are so exceptionally malformed and deficient that some interdependence between them is at once suggested. And although from a consideration of this single anatomical specimen we are not justified in making any more precise assertion, still, we can scarcely avoid looking upon it as confirmative of those physiological experiments which point to the angular gyrus as in some way bound up with the function of vision."

On June 10th, 1884, I read another paper before the Royal Medical and Chirurgical Society on "Embolicism of the Right Middle Cerebral Artery producing Left Hemiplegia and Hemianæsthesia; Absorption of a Large Portion of the Right Hemisphere; Death Seven Years Later."<sup>3</sup> In this case the patient had been suddenly attacked, seven years before her death, with embolicism of the right middle cerebral artery, which resulted finally in the destruction of a large part of the right hemisphere. At the time of her attack she was in Guy's Hospital, where careful notes were taken. From these it appeared that after being unconscious for four days she was found to have, in addition to left hemiplegia and hemianæsthesia, dimness of vision in the left eye almost amounting to complete blindness, though the patient said that she saw perfectly before the attack. Hearing was good in the right ear, but almost absent in the left. Six and a half weeks after the commencement of her illness sight and hearing were completely restored. Seven years after this attack I performed a post-mortem examination on this patient and found that the following convolutions had been destroyed on the right side: the inferior frontal, the external portion of the orbital, the lower half of the two central convolutions, the inferior parietal lobule, the convolutions of the island of Reil, the angular

gyrus, the superior and middle temporo-sphenoidal, and part of the inferior occipito-temporal convolutions. The conclusions I drew from this case are as follows:—1. That if the angular gyrus and superior temporo-sphenoidal convolutions be respectively connected with sight and hearing on the opposite side of the body their destruction does not involve permanent loss of these special senses. Such an inference agrees with the results of experimental removal of the angular gyrus, an operation which has been found to produce crossed amblyopia, from which the animal soon recovers. 2. That each hemisphere is specially connected with the sight of the opposite eye. In a recent paper by Dr. Allen Starr on the Visual Area in the Brain the writer adopts the statement of Mauthner, made in 1881, that "there is no case well authenticated in which lesion of one hemisphere has produced blindness in the opposite eye"; and he concludes that "when a lesion of one hemisphere involves the optic fibres at any point partial blindness of both eyes, and not blindness of the opposite eye, is produced." Although I have seen cases where hemiplegia with hemianæsthesia, including loss of sight and other special senses, occurred on the same side of the body, and where a lesion of the opposite hemisphere appeared to be the cause of all, I have never before had the opportunity of verifying the fact at a post-mortem examination. As very careful notes of this case were made at Guy's, Hospital and on examination the left eye was found to be amblyopic and the right not, the conclusion that each hemisphere has a more special connexion with the opposite retina than with that on the same side seems to me to be legitimate.

During the early part of last year Dr. Wainwright, senior resident medical officer of the Evelina Hospital, brought a brain to Mr. Shattock, and the latter very kindly handed it over to me to make what I could of it. I saw at once that the specimen might prove to be of great interest, and I accordingly examined it carefully after due preparation. I found that there were bilateral softening and atrophy of a very marked kind involving the brain in the region of the Sylvian fissure symmetrically, but in unequal degrees on the two sides. The area involved was most extensive in the left hemisphere (Fig. 1). The ascending frontal and ascending parietal convolutions, the inferior parietal lobule, the angular gyrus, and the greater part of the temporo-sphenoidal lobe were practically gone, being atrophied and shrunken to the last degree, and presenting the rich brown tints so often present in cerebral softening. On the right side (Fig. 2) the ascending frontal and the greater part of the ascending parietal convolutions were intact, as were the two annectant convolutions forming the superior parietal lobule. The inferior parietal lobule, the angular gyrus, the superior temporo-sphenoidal, and the neighbouring parts of the middle temporo-sphenoidal convolutions were much atrophied, discoloured, and degenerate. The left crus cerebri was much smaller than the right. On both right and left side the occipital lobe was quite intact. The central ganglia on the left side were atrophied and much smaller than those on the right, which appeared to be normal. The softening in the left hemisphere was evidently extensive both in superficial area and in depth, while that on the right appeared to be very superficial and confined to the cortex. On both sides the occipital lobes were quite natural in appearance. In order to see whether the fibres which pass from the occipital lobe to the internal capsule were diseased or intact a horizontal section was made which passed through the middle of the softened area and into the central ganglia, displaying the whole course of fibres in question (Fig. 3). This was done on both sides of the brain and very successfully brought to view the radiating fibres of the occipital lobe and the long bundle formed by them on their way to the internal capsule. The softening in the right hemisphere proved to be quite superficial; it had not reached or altered in the least degree the occipital fibres. In the left hemisphere these fibres appeared also to be quite healthy; only at one spot in the section did the orange yellow colour of the softening reach them, and even there the fibres appeared to pass along without any alteration. Mr. Shattock, who made the sections in my presence, confirmed these observations. The result of the examination therefore amounted to this, that in the left hemisphere there was a wide area of destruction affecting the parietal lobe and the angular and temporo-sphenoidal convolutions; in the right there were softening and shrinking of the inferior parietal lobule and of the angular and temporo-sphenoidal convolutions only, while

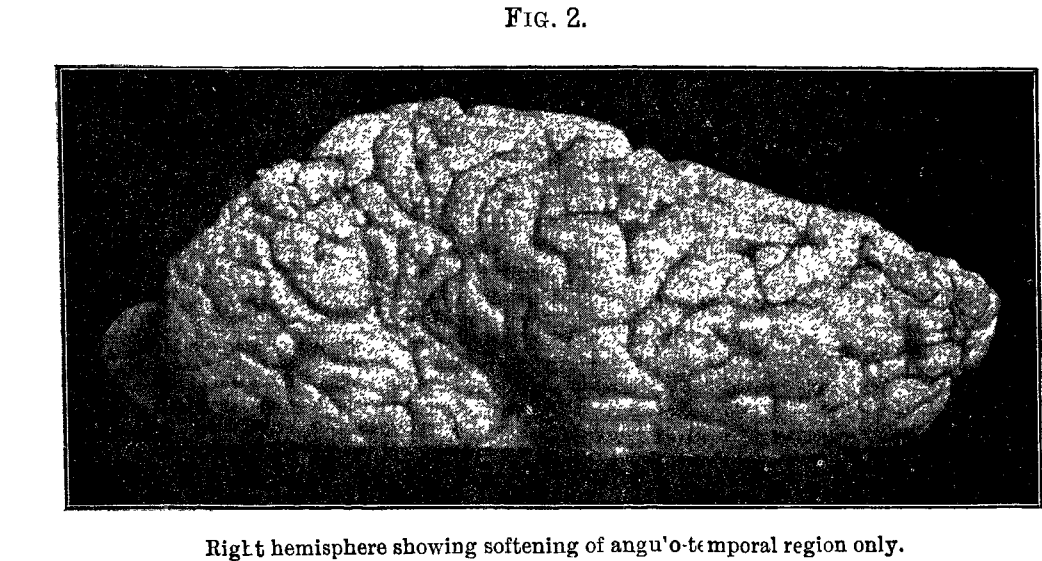
A paper read before the Neurological Society

<sup>2</sup> THE LANCET, May 12th, 1883.

<sup>3</sup> THE LANCET, June 14th, 1884.

on both sides the occipital lobe was intact. The symmetrical lesion in this child's brain suggested that it might have originated in instrumental delivery. The atrophied convolutions included these which have been bilaterally removed in animals for the purpose of investigating the localisation of sight and hearing in the hemispheres. Just such an experiment as this by disease is what has long been wanted, and on seeing this specimen great hopes were awakened in me as to its probable value. I wrote therefore to Dr. Wainwright and asked him to be kind enough to send me the child's history. He replied that the child was two and a half years of age and had been quite well until six years old, when he had a fit and lost power in the right arm and leg. After that these limbs gradually got rigid. The general health, however, was pretty good until fourteen days before admission, when cough and

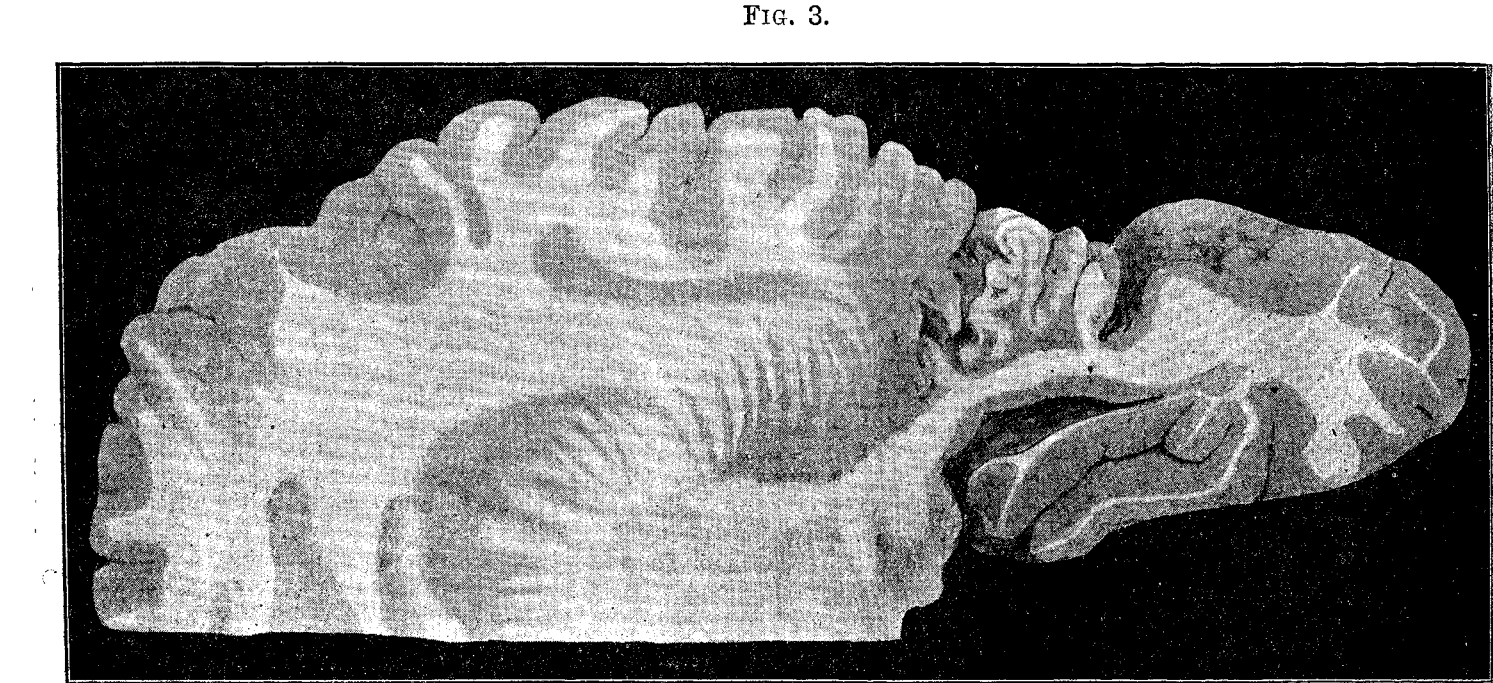
neither optic neuritis nor atrophy. The child appeared to be stone deaf. He died gradually after some weeks. His birth had been perfectly easy and natural and no instruments were used. As I wanted to get the facts as regards deafness I wrote again to Dr. Wainwright and also inquired as to vision. He replied: "As regards deafness, I found it impossible to rouse the child by any amount of noise; it would not even look round, though it answered quickly to touch. The nurses also failed entirely to find any evidence of hearing. At one time it was put in a bed with another child who was screaming in a most frantic manner; though quite awake it did not even turn towards it. The child was always considered to be quite blind, but it had some sensation to light in the left eye, I think, as it used to get worried if one focussed light on it for some time." I wrote again to Dr. Wainwright asking him to give me the facts which convinced him that the child was blind. He replied: "I spent a good deal of time on several occasions examining the child's eyes for optic changes and tubercles and it never took the least notice of the light, never showing the least tendency to object or even to blink except in the case of the left eye, which sometimes after a while seemed to be irritated, and it would rub it. It was very



Left cerebral hemisphere showing extensive destruction of the convolutions of the parietal lobe and of the angulo-temporal region.

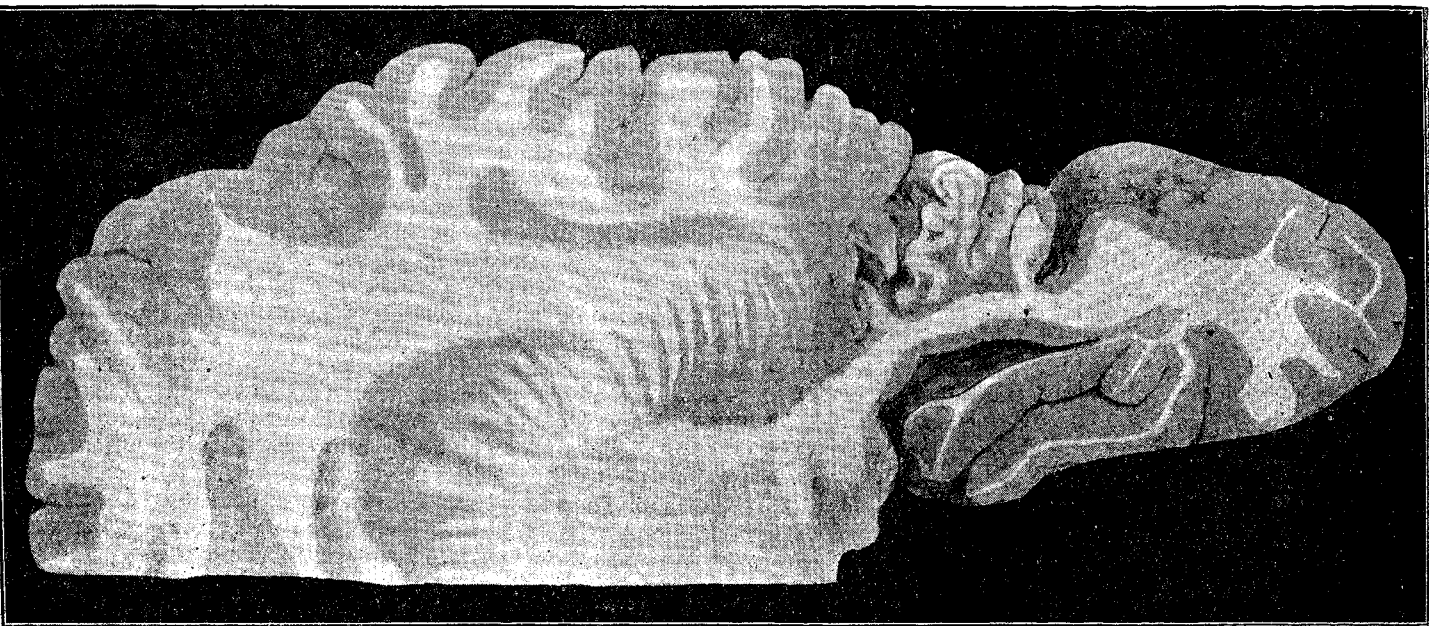
wasting set in. Advanced tuberculous disease of the lungs was then found, and the child was miserably wasted. The right arm and leg were contracted, and the contraction could only be overcome with difficulty and with the production of much pain. The right knee-jerk was absent; the left was active. The fundi of the eyes were healthy; there was

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Right hemisphere showing softening of angulo-temporal region only.

FIG. 3.



Horizontal section of right hemisphere showing the shrunken angulo-temporal convolutions and the intact occipital lobe and its fibres going to the internal capsule.

keen about its food, but unless its lips were touched it made no effort to get at it when a spoonful of anything was offered it. As circumstantial evidence, I may mention that the nurses say they always thought that everyone acknowledged that it was blind. As to your question about mental dulness and apathy, I may say it was not exactly lively, but it was quite sensible, and it never until quite the end showed any sign of coma, and it was in the hospital sixty days." The evidence as to blindness and deafness being satisfactory, I wrote again to Dr. Wainwright asking him to question the parents as to whether the child had ever seen and heard. Great difficulty was experienced in finding their whereabouts, as they had moved, and when they were at last traced they evinced considerable annoyance, as they had done before, at being bothered about the child, as they could not understand what good reason we could have for the trouble we were taking. However, both mother and father asserted that they were quite positive that the child used to see and hear well. Such are the anatomical and physiological facts which I have to record in this case. What gave rise to the superficial atrophy and softening of the right inferior parietal lobule and angulo-temporal convolutions I cannot say. If we disregard the hemiplegia on the right side of the body which was produced by the disease of the convolutions of the parietal lobe on the left, we have remaining a bilateral destruction of the inferior parietal lobule and angular and temporo-sphenoidal convolutions on both sides, while the occipital lobe and its radiations are intact. As far as one can see, that is so on both sides; even if this be doubtful in the left hemisphere it seems free from doubt as far as the right is concerned. Granting, then, that the original lesion in the left hemisphere which gave rise to the hemiplegia may have produced temporary blindness and deafness in the opposite side, which soon recovered, the facts of the case seem to indicate that when the inferior parietal lobule and angulo-temporal convolutions of the right hemisphere also became diseased the patient's sight and hearing were abolished.

The three cases which I have brought forward supply important evidence, each in its own way, of the relation of the region of the angular gyrus to vision. The first or anatomical specimen, though very suggestive, is lacking on the physiological side. In the other two cases we have both the anatomical appearances and the physiological changes which accompanied them, and I think they deserve the title of striking cases. But they have the defects which almost all such cases must have; anatomically it is difficult to state the precise limitations of disease; and physiologically we would like to have some further details. But it must, I fear, generally be so with our observations on man. In experiments on animals, however, even if the difficulties of correctly estimating physiological changes be surmounted, which are more than ordinarily great in experiments relating to the functions of the brain, and if the exact limit of the injuries inflicted can be determined there still remains the fact that the knowledge gained of cerebral physiology in animals cannot be directly transferred to man. It serves as a beacon to guide our observations at the bedside and in the post-mortem room, but it still lacks that confirmation which alone can justify us in accepting it as a part of human physiology. Therefore, defective as the three cases may be, they appear to be worthy of forming links in the chain of evidence which finally leads to the solution of such questions. I suppose most clinical observers will admit, as I do, that the occipital lobes are very intimately connected with vision, and are, in fact, "half vision" centres. But it would be interesting to know whether the members of the society are inclined to limit the visual area in them to their internal or central aspect. To me it seems that the facts of disease do not warrant such a conclusion, but that hemianopsia results from destruction of the cortex on the external aspect as well. Hemianopsia is so persistent when once developed that it must be allowed that one occipital lobe cannot make amends for disease in the other, whereas one angular convolution can speedily enough make up for the temporary blindness produced by destruction of the other. How is it, then, that although each angular region seems to be able to subserve vision in both eyes it cannot make up for hemianopic defect produced by disease of the occipital lobe? The only explanation I can see is that the occipital lobe is a lower centre through which visual impulses must pass on their way to the higher centres in the region of the angular

gyrus, and that disease of the lower centre or of the fibres in connexion with it intercepts the visual impulses on their way to the higher centres. But each higher centre being connected with the whole of both occipital lower centres, destruction of one does not interfere permanently with vision in either eye. Considering the important part which is played by mental vision in the higher intellectual processes in man it is not to be wondered at if he is found to possess high visual centres in an advanced state of development, although such centres may be non-existent or only rudimentary in animals below him in the scale of existence; indeed, clinical experience seems to show that mental blindness, and especially that variety of it which is called "verbal blindness," is associated with disease of the inferior parietal lobule which lies in front of the termination of the fissure of Sylvius. This convolution is continuous above and behind with the angular gyrus. The conclusion to which my cases seem to point is that the convolutions surrounding the posterior extremity of the fissure of Sylvius are specially concerned with the vision of the opposite eye, and in such a direct manner that destruction of them in one hemisphere produces transitory blindness of the opposite eye, while bilateral destruction of them causes permanent blindness in both eyes.

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## ON VARIOUS FORMS OF TALIPES AS DEPICTED BY X RAYS.

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(Concluded from page 309.)

At first, and while those changes in the position of bones depicted in my last paper are still recent, acquired valgus and flat foot may be temporarily re-dressed by comparatively slight pressure with the hands. Herein it differs from the congenital form, which is stiff and unyielding from the very first.<sup>1</sup> The post-natal acquisition of valgus was traced in my last paper, and was shown to be due to insufficient, rather than to exaggerated, action of certain muscles, especially and primarily to weakness or sluggishness of the flexor longus hallucis permitting the os calcis to roll over on to its inner side, thus allowing the sustentaculum tali to droop and causing the head of the astragalus to fall into the sole. Such fall of necessity entails abduction on each other of the two halves, anterior and posterior, of the tarsus. When this false position has lasted a certain time the fasciæ and ligaments on the inner side of the foot become loose and thin, the tibial muscles weak and lengthened, while the fibrous structures on the outer side as also the peronei contract and adapt themselves to the acquired shortness. Thus after a certain period the foot is so firmly fixed in malposture that it cannot without certain adjuvants be rectified or much improved in shape by the use of any moderate manual force, thus in that matter of fixity simulating very much more the congenital type of the deformity. We have then, in formulating methods of treatment to deal with very different degrees of the evil, the slightest of which requires very little more than certain modifications of the foot-gear and better regulation of walking. Hence, though some of the details here mentioned may seem mere trivialities, they are not really so; their intelligent and persevering use will cure a large proportion of all but the more severe class of cases. Many young people, more commonly girls, are brought to me with lengthened feet almost devoid of instep, with straight or concave outer border, the internal malleolus almost effaced, and the head of the astragalus quite visible and projecting (see Fig. 13<sup>2</sup>). Such patients in walking turn the feet too much out, and a large proportion of them have the great toe forced outwards, the second toe either lying upon it or less commonly beneath it. This posture of the big toe may be, indeed is, very frequently the starting point and cause of the whole trouble,

<sup>1</sup> It has been already pointed out that congenital valgus is always, or very nearly always, combined with calcaneus, or less commonly with equinus. These conditions are not our subject just now (see THE LANCET, Jan. 30th, 1897). In future the one word "valgus" will be used to include also flat foot.

<sup>2</sup> THE LANCET, Jan. 30th, 1897.