The Montgomery Lectures IN OPHTHALMOLOGY.

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LECTURE I.—THE CORTICAL LOCALIZATION OF VISION.

In my first lecture I will attempt to place before you the evidence we now possess as to the regions of the surface of the brain where the images of objects that fall on the retinae excite visual sensations.

Owing to the decussation of a portion of the fibres of the optic nerves in the chiasma, each optic tract conveys impressions from the same sides of both eyes—that is, impressions excited by the retinal images of objects in the opposite halves of the visual fields. And since each tract terminates in the primary optic centres of the same side, and the optic radiations that spring from here pass to the same side of the brain, a total lesion of any part of the one optic system above the chiasma produces hemianopia, or blindness in the opposite halves of the visual fields.

It has only been within recent years that, as a result of many anatomical, physiological, clinical, and pathological investigations, we have acquired any accurate information as to the region of the cortex where retinal impressions excite visual sensations. These investigations indicate that the visual centres lie within and along the lips of the posterior parts of the calcarine fissures, and that they probably correspond to that portion of the cortex characterized by the presence of Gennari's line, which Professor Elliot Smith has called the area striata. In most brains this striate area extends around the occipital pole on to the lateral surface of the hemisphere.

Until lately, too, we possessed very little evidence of the manner in which the different segments of the retinae are represented in the visual cortex, but, owing to the large number of cerebral injuries that have been produced by gunshot wounds, the late war has given us an unequalled opportunity to investigate this problem. Numerous observations by myself and others have provided fuller data on which to discuss it. In March, 1916, I published, in collaboration with my colleague, Sir W. T. Lister, a communication on this subject; last year I was able to amplify the views which we then put forward, and to-day I wish to place before you such final conclusions as I can draw from a very large number of observations.

Two facts tend to make these conclusions less convincing than they might be. In the first place my earlier observations were made in France, where it was not possible to keep patients long under observation, and the defects of vision that were then recorded might be, to some extent, due to the temporary dynamic changes in the brain generally associated with gunshot injuries of the skull rather than to local destructions of the visual cortex. That areas of local blindness did not, as a rule, alter materially after the first two or three weeks is a strong argument that they were not produced only by such transient disturbances, and this assumption is confirmed by the fact that in some of the patients I have been able to examine as long as three and a half years later the state of the visual fields was practically unaltered. I have also had the opportunity of supplementing my earlier observations by the examination of several cases in which the visual disturbances were of much longer standing.

In the second place, since I have been able to correlate my clinical observations with the causative anatomical lesions in a few cases only, it has been possible for the most part to determine the portion of the brain that was injured only by measurements of the sites and the depths of the wounds. This method is certainly inexact, though the position of the area striata can be determined approximately, for the inion, or external occipital protuberance, has a fairly constant relation to its posterior end. In mesial sagittal sections of several heads I found that the posterior ends of the calcarine fissures lie 2 cm., or $\frac{3}{4}$ in., above its tip, and that the fissures extend forwards and

upwards at an angle of about 25 degrees from there to a depth of about 4 cm. By careful measurement of the injury to the skull, or of the position of retained missiles as seen in stereoscopic radiographs, in relation to the inion the portion of the visual area that is injured may be determined approximately. Further information was obtained in many cases by examination during operations of the site and extent of the damage to the brain.

We must, however, await the opportunity of correlating the visual defects with the anatomical lesions that caused them in a large series of cases before finally receiving such conclusions as I can place before you into the body of accepted fact, though if these conclusions establish a hypothesis that can explain all our observations, or is at least not at variance with them, there will be strong presumptive evidence that it represents the truth.

I have selected as far as possible cases with long-standing or permanent visual defects to illustrate this lecture.

DESTRUCTION OF THE STRIATE AREA ON ONE SIDE.

Let us first consider the effect of a lesion which destroys the whole of the area striata of one hemisphere. A deeply penetrating gunshot wound of the occipital region immediately to one side of the middle line, and not more than 5 cm. above the inion, that passes forwards parallel to the mesial sagittal plane, must do so. I have seen several men with such wounds, and all had complete hemianopia, or total blindness to the opposite side of the fixation point. This blindness extends almost invariably up to the fixation point, or at least to within one degree of it, which is certainly within the margin of error of our clinical methods. The more accurate and delicate the methods employed the nearer is the blindness found to come to the point on which the eyes are directed. Observations on an ordinary perimeter cannot be exact enough to decide this question, but it can easily be verified by examination by a Bjerrum's or other screen which is placed at a distance of one or two metres from the patient's eye. This is the method I have employed to investigate vision in the neighbourhood of the fixation point.

In hemianopia due to vascular lesions of the visual cortex the blindness rarely extends up to the fixation point, and in this respect it differs from that produced by a traumatic injury of one occipital pole. The explanation of this difference will be considered later in this lecture. The area of blindness is liable to be more irregular, and does not involve central vision so constantly, when it is due to injury of the optic radiations.

We turn now to the cortical representation of those portions of the retinae which lie above and below the horizontal planes through the maculae. Local lesions in the region of the calcarine cortex frequently produce blindness in either the upper or the lower homonymous quadrants; the following case is an example of superior quadrantic hemianopia:

CASE I.

Pte. J. was wounded by a fragment of shell which penetrated his steel helmet in April, 1918. He was unconscious for a few minutes only, and on coming to noticed that his sight was affected. It improved rapidly after an operation that was performed on the same day, but he experienced some difficulty in reading when he was first examined three weeks later.

The wound was represented by a small defect in the skull immediately to the right of the middle line and just above the lateral sinus. An oblong piece of shell casing, which measured $3 \times 1 \times 1$ cm., and several fragments of bone were removed from the brain, which was softened and disintegrated for a considerable distance beneath the fracture.

When he was seen three weeks after he had received the wound there was complete left superior quadrantic hemianopia, which reached exactly up to the fixation point, and, in addition, a peripheral contraction of the left lower quadrants. The latter gradually disappeared, so that five weeks later vision was lost in the upper quadrants only. The blindness still extended up to the fixation point, but it did not everywhere reach the horizontal meridians; there was a narrow zone along these in which a small white object was indistinct and frequently not perceived (Fig. 1). In the lower left quadrants white appeared greyish, and the colour of red and green test objects 10 mm. in diameter could not be recognized. Central vision was $\frac{3}{4}$ in each eye.

Here we have a case in which a penetrating wound close to the middle line, immediately above the lateral sinus, and consequently below the level of the right calcarine fissure, produced an absolute superior quadrantic hemianopia. It is therefore evidence that the lower halves of the retinae are projected on to the lower parts of the

area striata. The loss of colour vision in the inferior quadrants was associated, as is the rule, with the slight degree of amblyopia there, which was indicated by the apparent indistinctness and greyness of a white test object in this portion of the field.

On the other hand, when blindness of the lower homonymous quadrants of the visual fields occurs as a result of cortical injuries, it is always associated with wounds above the level of the calcarine fissures. I have already recorded several cases with this condition, and it is not sufficiently rare to need specific illustration here. These cases go to prove that the upper halves of the retinae are represented in the upper or dorsal parts of the visual cortex, just as the lower segments of the retinae are projected on to its lower portion.

If a lesion of one occipital lobe above the level of the calcarine fissure produces an inferior quadrantic hemianopia, bilateral lesions in this position should cause blindness below the fixation point. I have seen eight cases of more or less pure inferior horizontal hemianopia, and in all of these the cerebral wounds were above the level of the calcarine fissures and involved, as far as could be determined, the upper portions of the striate areas. I have chosen the following case as an example, as it was possible to observe the patient ten months after the infliction of the wound, and its site indicated that it was the visual cortex rather than the optic radiations that were injured.

FIG. 1.—Case I.

CASE II.
Pte. L. was wounded in March, 1918, by a machine-gun bullet. He was unconscious for a short time. During the first few days he could distinguish only light from darkness, but his vision then improved rapidly. Its condition became stationary after an operation was performed three weeks later in a German hospital, for he had been made a prisoner of war. He states that when he tried to read during the next three months he always seemed to see the figure of a white hen to the left of the point to which his eyes were directed. I examined him in January, 1919.

The wound was represented by a healed and depressed scar, under which a trephine opening about 2 cm. in diameter could be felt. This involved the middle line of the skull; its centre was 5 cm. above the inion. Radiographic plates did not reveal any foreign bodies within the skull.

His central vision was $\frac{3}{4}$ in each eye. There was a large area of blindness in both lower quadrants, which was more extensive on the left than on the right side, and in the seeing portions of the left lower quadrants vision was misty and indistinct. (Fig. 2.)

In this case the injury of the brain was obviously above the level of the calcarine fissures, and it was consequently the dorsal portions of the areae striatae that must have been most severely injured.

I have not observed a pure case of superior horizontal hemianopia; the probable reason is that gunshot wounds involving the lower portions of both striate areas would necessarily produce such injury of the torcula as would be probably fatal.

CORTICAL LOCALIZATION OF CENTRAL VISION.

If the evidence from these and similar cases shows that the upper halves of the retinae are represented in the upper or dorsal parts of the calcarine areas and the lower in the lower or ventral, we may now turn to the consideration of the cortical localization of macular or central vision. The most satisfactory evidence on which to base conclusions would be obtained from cortical lesions that produce complete loss of central vision in both eyes. I have seen and recorded a certain number of these. The case that is cited here as an illustration was the only patient who was observed at a long period after the infliction of the wound.

CASE III.

Pte. G. was wounded by a large portion of shell that penetrated his steel helmet in May, 1918. He was unconscious for seventeen hours. In the casualty clearing station, where an operation was performed on the same day, a depressed fracture of the skull was found in the occipital region, and a mass of bone was removed from the longitudinal sinus and from the brain on each side of it. He was blind for the first three days, but his vision then rapidly improved to its present state. A month after the infliction of the wound his visual fields were examined by Mr. J. B. Lawford, who discovered a scotoma almost identical in size and position with that which I found nine months later.

Ten months after the injury the wound was firmly cicatrized; beneath the scar there was a more or less circular defect in the middle line of the skull and to the left, which involved the base of the inion and extended $4\frac{1}{2}$ cm. upwards from it. Its lateral diameter was 5 cm. The vision was less than $\frac{3}{8}$ in each eye (according to his records it was $\frac{3}{8}$ when he enlisted in November, 1915), and he could not even recognize the other men in the ward. He saw nothing plainly, and had some difficulty in recognizing the distance of objects from him.

His peripheral vision was unaffected; he could see a circular white object 10 mm. in diameter to the natural limits in both eyes, but examination with a perimeter and by a screen revealed a large area of absolute blindness, which extended about 3 degrees to the left of the fixation point and from 12 to 18 degrees to its right. Central vision was totally abolished. (Fig. 3.)

Above and to the left of this scotoma there was a small area in which white objects could be seen, but they appeared dull and dim; colours could not be recognized here, nor in a zone of about 2 degrees to the left margin of the scotoma.

The site of the wound makes it probable that both occipital lobes were injured at the posterior ends of the areae striatae, which were opposite the centre of the trephine opening. And both the position of the wound and the clinical symptoms indicate that the injury was more extensive on the left than on the right side.

This case illustrates the type of blindness that is produced by superficial injuries of both occipital poles—that is, by wounds that injure the posterior parts of the striate areas. In all such cases we find peripheral vision intact and central vision abolished. They are consequently evidence that central or macular vision is represented in the more posterior parts of the visual areas, and that this region is not concerned with peripheral sight.

The evidence to be obtained from unilateral lesions confirms this opinion and also supports the conclusion we have already arrived at, that the upper parts of the retinae are connected with the upper parts of the visual cortex and the lower with the lower.

Homonymous paracentral scotomata are frequently produced by injuries of the polar region of one occipital

FIG. 2.—Case II.

lobe. The visual fields in the following case represent a characteristic type:

CASE IV.

Pte. S. was wounded by a fragment of shell in October, 1918, and was operated on in a casualty clearing station on the same day. He was unconscious, or at least remembers nothing that happened, during the first few days. Then he discovered that he could not see properly to his left, but he frequently noticed flashes of light to this side which he compared to ordinary gunflashes. He had also a feeling that "there was always something standing on the left side, a person or something," but he could distinguish no form. His vision improved quickly, but became stationary within a few weeks.

The wound was represented by a healed vertical scar in the occipital region immediately to the right of the middle line. Beneath it there was a defect in the skull, $4\frac{1}{2}$ cm. in vertical length and 2 cm. broad, which extended from 1 cm. above the inion upwards, parallel to the mesial sagittal plane of the head.

I examined him in February, 1919. His vision was $\frac{3}{8}$ in each eye. He complained that in reading he had difficulty in finding the beginning of a line of paragraph, and even the first letters

of a long word. In the street he often failed to see people in front of him and to his left. The peripheral limits of his vision to a circular white object 7 mm. in diameter were normal, but there was a large left paracentral scotoma which extended from the fixation point to about 20 degrees to the left. Its edges were remarkably sharp and definite. (Fig. 4.)

In this case an injury of the right occipital pole, which must have involved the posterior end of the area striata, caused a left homonymous paracentral scotoma. This is the condition of the visual fields most commonly associated with lesions of this region. The scotoma generally extends up to the fixation point, and usually its size can be seen to bear a close relation to the depth of the wound. Such cases supplement the evidence we have already obtained that the centre for macular and perimacular vision lies near the posterior ends of the hemispheres. In another case, with a similar defect in the left sides of the visual fields, total destruction of the posterior part of the right striate area was found on *post-mortem* examination, when death took place five weeks after the infliction of the wound.

Smaller paracentral scotomata are frequently limited to either the upper or lower homonymous quadrants. Their position in the field of vision always bears a close relation to the site of the wound—that is, they lie below the fixation point when the injury involves the upper and posterior end of the calcarine area, and above it when the ventral portion of the visual region is damaged. The following case is a typical example of an inferior paracentral scotoma.

CASE V.

Lient. H. was wounded in November, 1915, by a bullet. He found at once that he could not see the ground at his feet properly. When he was examined four days later his only complaint was that on trying to read he could not see the lines immediately below that on which his eyes were directed, though the rest of the page was visible to him. There was a tangential scalp wound with its entry 2.5 cm. above and 4 cm. to the right of theinion, while the exit was in the middle line 2 cm. above theinion.

Peripheral vision was undisturbed, but there was a small absolute paracentral scotoma in the left lower quadrants, which extended from the fixation point outwards and downwards to about 9 degrees. This patient was examined again recently, more than three and a half years after the infliction of the wound. Peripheral vision to both white and colours is unrestricted, and his visual acuity is $\frac{5}{6}$ in both eyes, but the paracentral scotoma remains practically unaltered, the only change being that he can now recognize a moving white object between 6 and 9 degrees from the fixation point, where he was at first totally blind. But in this narrow zone vision for white is still very defective, and colours cannot be recognized. (Fig. 5.) The only inconvenience he experiences is in his work as an accountant; in "running up" a column of figures he adds them correctly, but in "running down" a column he often fails to see the left-sided figures; that is, the hundred and the thousand units.

CASE VI.

This case is an instance of a superior paracentral scotoma. Pte. A. was wounded in September, 1918, by a fragment of shell which burst behind him while he was riding. He was dazed for a time but did not lose consciousness, and was able to ride alone to the first aid post, though his sight was considerably affected. He noticed no subjective visual phenomena. He was immediately transferred to a casualty clearing station, where part of the missile that was embedded in his skull, and splinters of bone that had been driven through the dura mater into the right occipital lobe, were removed. The wound healed rapidly.

The wound, five months later when I examined him, was represented by a scar slightly above and to the right of theinion, with roughened bone and a defect in the skull beneath it. Radiographic plates revealed a small circular trephine hole, 2 cm. in diameter, its centre being $1\frac{1}{2}$ cm. above theinion.

He stated that his vision, which was at first seriously impaired, improved quickly, and that it was as good when he reached England a fortnight after being wounded as it was when I saw him. He complained only that he had difficulty in reading, as he generally missed the first few letters of long words.

His central vision was $\frac{5}{6}$ in each eye, and on examination with a perimeter the peripheral limits of the fields were found to be normal. He had, however, a small absolute scotoma to both white and colours to the left of the fixation point. It was carefully mapped out by the aid of a screen; the greater part of it lay in the left upper quadrants, into which it extended 5 to 6 degrees from the fixation point, but a small part projected into the lower quadrants. Its edges were remarkably sharp, and were identical to white and colours; an object moved from it into the seeing portions of his fields appeared suddenly as if it had come from behind a screen. The blindness extended directly up to the fixation point. (Fig. 6.)

In Case v the inferior paracentral scotoma was obviously due to an injury of the visual cortex produced by the wound, which was situated above the level of the calcarine fissure; while in Case vi, where the scotoma lay chiefly in the upper quadrants, the causative lesion must have affected the lower and posterior part of the striate area, as the wound was immediately above theinion.

Bilateral paracentral scotomata are less common, but when they occur they furnish further confirmatory evidence of the conclusions to which we have already come. The following is a good illustrative case:

CASE VII.

Pte. B. was wounded by a machine-gun bullet which passed through his steel helmet in July, 1916. He was dazed or semi-conscious for a short time. He then walked alone to the dressing station, but repeatedly fell into shell holes and across obstacles as he could not see the ground before his feet. An operation was performed on the third day after the infliction of the wound, and a fortnight later he was transferred to England, where he remained in hospital for four months. During this time he stated he could see quite well, and was not aware that his vision was in any way affected. He volunteered for active service, and came to France again in July, 1918. I examined him in August of that year, more than two years after he was wounded.

The wound was indicated by a transverse horizontal scar, 8 cm. in length, which was bisected by the middle line and crossed it 4 cm. above theinion. Radiographic plates revealed two small circular openings in the skull, one on each side of the middle line; their lower margins were $2\frac{1}{2}$ cm. above the point of theinion.

His central vision was $\frac{5}{6}$ in each eye and the peripheral limits of his visual fields for both white and colours were normal. There was, however, a large absolute bilateral inferior paracentral scotoma, more extensive in the right than in the left quadrants, which on the right came to one degree from the fixation point, and $2\frac{1}{2}$ degrees from it on the left. This area of total blindness was surrounded by a zone in which a moving object could be seen, though it appeared grey and indistinct, and in which the colours of test objects could not be recognized. Except in this amblyopic area the colour fields were normal. (Fig. 7.)

In this man both hemispheres were obviously injured above the level of the calcarine fissures, and the resulting blindness was limited to the inferior quadrants. The fact that the blindness did not extend into the immediate area of the fixation point is probably to be attributed to the escape of the posterior extremities of the visual cortex.

Not infrequently two or more scotomata coexist, which may be separate from one another or united into a complex figure. It is on these cases that we can most easily test our hypothesis, and if by it we can explain the occurrence of the blind areas as a result of the anatomical sites of the wounds we have a strong argument that it is correct. The following case is an instance:

CASE VIII.

In this patient I found a right inferior paracentral scotoma, while to the left of the fixation point there was a similar

FIG. 3.—Case III.

FIG. 4.—Case IV.

area of blindness, chiefly above the horizontal plane. This observation was made on a man six weeks after he received an oblique gutter wound which crossed the middle line of his skull 2 cm. above theinion; it passed from below upwards and to the left, and consequently injured the lower portion of the area striata on the right side, producing the left superior scotoma, and the upper part of the visual cortex on the left hemisphere, causing the defect in the right inferior quadrants. (Fig. 8.)

This series of cases illustrates the evidence that my observations can furnish on the cortical localization of central and pericentral vision. It proves that the macular and perimacular regions of the retinae are represented at or near to the occipital poles of the hemispheres. It is probable, indeed, that the portion of the area striata which frequently spreads over the occipital pole and on to the lateral surface of the brain may be the centre of macular sight. The frequency of paracentral scotomata is consequently accounted for by the more exposed position of this part of the visual area than of that which lies on the mesial surfaces of the hemispheres. It seems very probable, too, that the maculae have anatomically larger representations in the cortex than the less highly specialized peripheral zones of the retinae, and in this we may find the explanation of the frequency of small areas of blindness in the neighbourhood of the fixation point, though the causal lesions may be relatively gross.

LOCALIZATION OF PERIPHERAL VISION.

If all our experience points to a posterior representation of macular vision in the calcarine regions we may by exclusion assume that the centre for peripheral vision lies in the anterior part of the visual areas. There is, however, relatively little direct evidence of this. It is true that this region of the brain is not uncommonly injured by gunshot wounds, but as the anterior part of the calcarine area is surrounded on each side by the optic radiations that carry all visual impressions corticalwards, the visual defect produced by such a cortical lesion would be complicated by the presence of blindness due to interruption of the fibres of the radiations. Riddoch has, however, observed a case in which peripheral contraction of both halves of the visual fields was associated with the presence of a missile and fragments of bone between the mesial surfaces of the occipital lobes, in such a position that the anterior parts of the areae striatae were probably injured. I have also recorded a case with loss of peripheral vision in the homonymous fields in which a missile was localized at the junction of the calcarine and parieto-occipital sulci. The following case is further evidence in this question:

CASE IX.

Corporal W. was wounded by a fragment of shell in October, 1918. He was dazed for a few moments, but noticed no affection of his sight. He was operated upon in a casualty clearing station next morning. His wound healed quickly, and he remained free from symptoms, except headache, till February, 1919. Then one morning his right side became suddenly numb and weak, and a dull black spot appeared in vision; he referred it to the lower margin of the outer canthus of his right eye, and repeatedly tried to rub it away without success. After some minutes he began to see flashes of light, "like lightning, uncoloured," which flashed from the black spot towards the centre of his eye; these persisted for ten or fifteen minutes. Then he lost consciousness and was probably convulsed. This was the only severe fit he has had, but two minor attacks on an average have occurred every day since then. These always commence with the appearance of one or two black spots, which he refers to the lower margin of the right outer canthus. They are at first very faint, but become more and more distinct, and then flashes of light begin to shoot from them towards the

centre of his right eye; these may persist as long as thirty minutes, and appear whether his eyes are closed or open. In these attacks he feels strange, as if he were going out of his mind, and after them he has considerable headache.

He was examined in May, 1919, seven months after the infliction of the wound.

The wound was represented by a small circular trephine opening immediately to the left of the middle line of the skull, with its centre slightly above the lambda—that is, 8 cm. above theinion. Radiographs revealed an irregular piece of metal in the brain; in antero-posterior views it was seen slightly to the left of the middle line, and when measured on lateral stereoscopic plates it was localized in the antero-inferior part of the left cuneus, immediately posterior to the junction of the calcarine and parieto-occipital sulci.

His visual fields were repeatedly examined. He perceived a circular white object 10 mm. in diameter to the natural limits of his fields, but in the periphery of the lower right quadrants it was blurred, indistinct, and greyish, and often disappeared

from vision for a moment, especially when it was not moved. It also seemed much smaller in this region, which extended to about 45 degrees from the fixation point. He could, however, distinguish the relative sizes of two objects presented to him in succession in this area as accurately as in homologous parts of his left fields, and recognize the shape of test objects, though the recognition was less certain. When an object 2 mm. in diameter was employed this amblyopic area was more extensive, especially near the vertical meridians. (Fig. 9.)

The fields for red and green objects, 10 mm. in diameter, were approximately normal and equal in the right and left temporal fields; but when a large red object 30 mm. in diameter was used he failed to distinguish its colour, or saw it only as indefinite greyish-red, in the amblyopic area, though its colour was distinct out to 70 degrees in the lower left quadrants.

The radiographic localization of the missile in this case shows that it was in or near the anterior part of the area striata of the left hemisphere, and the occurrence of amblyopia in the periphery of the lower right quadrants suggests that this is the part of the visual cortex that is concerned with peripheral vision below the horizontal. Further, it was here that the visual spectra, which were undoubtedly due to discharges from the neighbourhood of the irritant lesion, always first appeared.

The missile was removed three weeks ago by Lieut.-Colonel P. Sargent. It was found on the left surface of the falx and probably lay in the anterior and inferior part of the cuneus. Since the operation he has had only one visual attack, which occurred on the second day after it.

Our observations so far indicate that the upper homonymous parts of each retina are projected on to the upper portion of the opposite visual area, and the lower halves of the retinae on to its lower. Further, all our facts go to prove that

the centre for macular or central vision lies posteriorly in the visual cortex, while that for peripheral vision is situated anteriorly in it. The representation of the intermediate zones of the visual fields from the fixation point outwards are probably arranged in this order from behind forwards in the visual cortex.

There are numerous observations, however, which make it probable that a further localization is possible within the visual cortex. It is, for instance, not uncommon to find blindness in the homonymous visual fields limited to areas in the region of the vertical meridians. In most of these cases that I have seen this loss was associated with hemianopia, or at least a large area of blindness, in the opposite halves of the fields, and the injuries that produced them were penetrating wounds of one occipital lobe, which probably caused a superficial damage on the mesial surface of the other hemisphere. The following case is an example of this condition.

FIG. 5.—Case V.

FIG. 6.—Case VI.

CASE X.

Pte. H. was wounded in September, 1918; he stated that he was unconscious for several days. At the operation, which was performed on the day of the wound, a depressed fracture was found in the left occipital bone a short distance above the lateral sinus, and several fragments of depressed bone were removed from a deep track in the brain. I examined him six months after the infliction of the injury.

The wound was represented by a circular trephine opening immediately to the left of the middle line of the skull, with its centre 4 cm. above the inion. No foreign bodies or retained missiles were visible in radiographic plates.

His central vision was in each eye, and he believed as good as it had ever been; but he had an absolute right homonymous hemianopia, which came exactly to the fixation point. He was also completely blind in the mesial portions of the left lower quadrants, except in a small area within 3 degrees of the fixation point. This blind area lay approximately between the inferior vertical meridians and the radii at 45 degrees from them. (Fig. 10.) Vision to both white and colours was unimpaired in the remaining portions of the left halves of the fields.

In this case the right-sided hemianopia was obviously due to the deep penetrating wound of the left occipital lobe. The loss of sight in the left halves of the fields shows that the right visual cortex also must have suffered, and the position and the nature of the wound makes it probable that the damage to the mesial surface of this hemisphere was superficial and probably limited to the surface of the cuneus. That portion of the area striata that lines the walls and the floor of the calcarine sulcus, and consequently lies further lateralwards, probably escaped injury.

I have seen many cases in which superficial injuries of the mesial aspect of the one hemisphere were associated with blindness along the vertical meridians of the visual fields. This suggests that the part of the area striata which is exposed on the surface of the hemispheres is concerned with vision in the neighbourhood of the vertical meridians, while the visual cortex that lies more laterally in the walls and the floor of the fissures is that which subserves vision along the horizontal radii. Wilbrand and Henschen's celebrated case, in which a narrow homonymous scotoma that extended from the fixation point horizontally outwards to 60 deg. was associated with a softening of the floor of the fissure, is complementary evidence of this hypothesis. I have not seen such a scotoma produced by a war wound, but this is not surprising, as any missile which penetrated the brain to the bottom of the calcarine fissure would necessarily injure the fibres of the optic radiations that pass to other parts of the visual cortex, and consequently produce more extensive blindness.

Such observations as these, which are only selected illustrations from a large number of cases, indicate that there is a definite areal representation of the retinae in the visual cortex. There can be little doubt, too, of the general principles of this localization; the macular regions are represented posteriorly and the periphery of the retinae more anteriorly in the occipital lobes, while the upper and lower portions of the retinae are projected on to the corresponding parts of the visual cortex. Further, the mesial horizontal segments of the retinae are probably projected in the neighbourhood of the floors of the calcarine fissures.

And not only is each segment of the retinae represented in a definite portion of the visual cortex, but this representation is fixed and immutable, so that if a part of the visual cortex be totally destroyed there will be a permanent blindness of the corresponding segment of the visual fields. Only so can we explain the persistence of such a small scotoma as in Case v, which, when examined three and a half years after the infliction of the wound, was found

almost identical in size and position as within the first few days of the injury. In Case III, too, the blind area remained unaltered between one and ten months after the injury. There is no place here for the theory of vicarious representation of the function of parts destroyed by other regions of the brain.

It is true that the functional loss observed immediately or shortly after the infliction of the wound is generally considerably greater than the permanent blindness, but this can be only expected since, in addition to destruction of a portion of the brain, gunshot injuries produce less severe damage with temporary loss of function in a zone around it. Hine has studied the early recovery of this temporary functional loss and the rules that govern it, and I have had many opportunities of observing it too. It is, however, the rule, as far as the visual cortex is concerned,

that this temporary blindness or amblyopia disappears within a few weeks and leaves only the permanent defect, provided that there is no sepsis, inflammation, or hernia formation. For this reason observations made within a few weeks of the infliction of the wound can be utilized in the problem of visual localization. That sight may later return in portions of an area that was at first blind is no valid argument against

the employment of such cases in the problem of the cortical localization of vision, for any scheme of localization that is true must explain the form and distribution of these temporary changes as well as the permanent loss of function.

Escape of the Macula in Vascular Lesions.

There are other questions that must be considered shortly. The homonymous hemianopia that is produced by a penetrating gunshot wound of one occipital lobe differs from the ordinary form of hemianopia due to vascular lesions that is met with in civil practice. In the latter the blindness rarely reaches up to the fixation point; as a rule there is a zone of normal vision to about 3 degrees to 8 degrees wide around it. This escape of central vision has excited considerable controversy. The one school explained it by the hypothesis that the maculae are not represented

in the occipital visual areas, but elsewhere in the brain. But not only has no case, as far as I am aware, of macular blindness due to disease of this hypothetical centre been recorded, but all the observations on visual disturbances due to local wounds of the brain that have been made during the war are directly opposed to it. A second explanation was that there is a bilateral representation of maculae, that as each macula is represented in both hemi-

spheres of the brain a unilateral lesion cannot abolish or disturb central vision. This hypothesis, too, is untenable, as when hemianopia is due to traumatic destruction of one occipital lobe the blindness usually extends to the fixation point, and the more accurate the methods of investigation, the smaller is the apparent escape. (My observations were made on screens placed one or two metres from the patient, and the margin of error was consequently small.) The same is true with regard to paracentral scotomata; when the most posterior region of the area striata is destroyed the blindness comes, as a rule, immediately to the fixation point. The probable explanation of macular escape in hemianopia due to vascular lesions is to be found, as Lister and I suggested more than three years ago, in the anatomical arrangement of the arteries that supply the occipital poles. Here, in the region of the posterior end of the striate areas, where the centre of macular vision is placed, the middle and posterior cerebral arteries meet, and if the circulation in the branches of the one is blocked

FIG. 7.—Case VII.

FIG. 8.—Case VIII.

by an embolism or thrombus the portion of the brain in this watershed area may obtain arterial blood from the branches of the other, since there is always a certain amount of overlap. The cerebral softening due to the occlusion of an artery is rarely as extensive as its anatomical distribution. This explanation not only disposes of the difficulty of the macular escape, but is confirmatory evidence of the localization of central vision.

Loss of Colour Vision.

The second question we must consider is what elements of vision are represented in the area striata, which we assume to be its cortical centre. We may here deal particularly with the question of colour vision. In the cases I have selected to illustrate my arguments, the loss of vision was complete in the portions of the fields that were coloured black; here there was total loss of all visual perception. But in a few cases, as in Case ix, there was only amblyopia or diminution of sight, and in others, as in Case vi, a zone of diminished vision surrounded a patch of complete blindness. Areas of amblyopia are in fact very common.

In these amblyopic areas the state of vision varies considerably. If the disturbance is severe only large white objects when moving may be recognized, but they appear "dim and misty," or "dirty and grey" instead of white, and the patient has no clear idea of their outline or size. When they cease to move he sees them no longer; they disappear. Smaller white objects may not be visible within this area or in some part of it; in fact the blind area generally varies indirectly in extent with the size and brightness of the object by which it is mapped out. In such areas colour cannot be perceived.

When the affection is less intense, white objects of moderate size can be seen even when at rest, though they appear indistinct and grayish, but small test objects may disappear momentarily from vision. The patient frequently states, too, that the objects presented to him seem smaller than they do in normal parts of his fields; but we have not here a specific loss of the appreciation of visible size, since the patient has always an idea of the magnitude of the object, if he can see it, and I have always found that he can distinguish the relative sizes of two test objects presented in succession almost as accurately as in homologous normal parts. The dimness and indistinctness of the visual image cause him to judge the object to be smaller than it is; he misinterprets the defective perception as smallness. In these cases colour perception is generally lost, too, or only relatively large areas of red and green can be recognized; but then their tints are usually pale and indefinite.

Finally, in the mildest degrees of amblyopia even small white objects can be perceived, whether in motion or at rest, though they usually seem dim; and red and green—the only colours that I have systematically used—do not appear in their natural tones; a deep red is often "yellowish red," and green "pale or greyish."

I have seen no case in which colour vision was lost in portions of the fields in which the perception of white was unaffected, and I know of no conclusive evidence that a purely isolated loss of colour vision ever results from lesions of the mesial surfaces of the hemispheres in the region of the visuo-sensory cortex.

EFFECT OF BLIND AREAS IN DAILY LIFE.

We may now consider how these large or smaller areas of blindness affect the subject. We know from ordinary clinical experience that a man with homonymous hemi-

anopia is frequently unaware that he has lost one half his seeing field, and that he learns to appreciate it only by experience. And the same is, as a rule, true with respect to blindness produced by traumatic cortical lesions. A man with complete hemianopia rarely complains spontaneously, when he is first examined, that he cannot see to the one side, and there is, as a rule, no subjective blackness or mistiness to this side unless central vision be affected; this part of visual space simply does not exist for him. And this holds true, too, for small patches of blindness to one side of the fixation point. A man with a left paracentral scotoma which extended to the fixation point constantly jolted into people who passed on his left side in the street; his consciousness seemed to be so little aware of his blindness that he rarely took precautions to avoid accidents which might occur through it. Even more

striking is the effect of a small paracentral scotoma limited to either the upper or to the lower quadrants. Case iv, for instance, saw only the feet or legs of a person approaching on his left side, and connected this part with the idea of a whole man only by a deliberate act of attention. Similarly in reading he and many others missed small words and letters, and Case v failed to include the left-sided figures in the column

he was adding; even after long experience he continued to miss them. These patients fail to realize their own blindness; the space not seen is simply space that does not exist for them, and they are no more conscious of its absence than is the normal person of his own blind spot. When, however, central vision is affected, so that the object on which the eyes are directed cannot be seen distinctly, the patients generally complain of a mistiness of vision, or of a blackness in front of their eyes when the blindness is here complete, though on careful questioning it is generally found that this is not a positive sensation of blackness but a state of not-seeing.

Positive Visual Phenomena.

Positive subjective visual phenomena are not uncommon during the first few weeks after the infliction of the wound. The most common form is the appearance of flashing

lights or stars, which may be either white or coloured, in a part of the blind or amblyopic area. It is rarer that these subjective phenomena occur at a later period; Case ix, which is recorded here, is an example. In another man the local epileptiform seizures with which he suffered always began with the appearance of lights of all colours on the margin of an area of peripheral blindness, which grew brighter and brighter

until they became intolerable as he lost consciousness; he exteriorized these centrally produced sensations so vividly that on one occasion he assaulted an orderly who stood beside him "for flashing those awful lights into my eyes." In a third case, brilliantly coloured stars appeared on the margin of a paracentral scotoma. Incidentally, the occurrence of colours in these sensations produced by discharging lesions in the calcarine region is an argument that colour perception depends on this portion of the brain.

In a second type of these visual sensations of central origin more complex figures appear in the blind area. These, which are more or less constant and do not move, are always extremely indefinite; a few men complained that there always seemed to be "a man or something" standing on their blind side, and one man with inferior horizontal hemianopia stated that when he tried to read there seemed to be a white hen on the lower part of the left page of the book. But when questioned these patients

FIG. 9.—Case ix.

FIG. 10.—Case x.

admitted that they could never recognize a shape, there was only a "feeling that there was something there," something too ill defined to describe. These indefinite forms appeared only in the early stages and generally in blind areas that were recovering. They are probably due to the misinterpretation of feeble and indefinite sensations excited in functionally depressed areas of the visual cortex.

A CASE OF SYMMETRICAL GANGRENE (RAYNAUD'S DISEASE).

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THE patient whose case is here related was admitted on May 22nd, 1919, to the Welsh Metropolitan War Hospital from the Bath War Hospital with a diagnosis of subacute mania, possibly general paralysis of the insane. It was reported that the pupils were unequal and sluggish, that there were facial and lingual tremors, and greasiness of the skin of the face. The duration of the mental attack was given as four days.

History.

Patient was 42 years of age. The information supplied by his wife showed nothing of note in respect of family or personal history. His present illness was said to have supervened after five years' military service. The following are extracts from the medical case-sheet received on admission of the patient to this hospital, the medical notes having been taken whilst at a hospital in Cairo:

Admitted on March 26th, 1919, suffering from bronchitis, bronchopneumonia, and Raynaud's disease. Arterio-sclerosis noted, also patches of bronchopneumonia in both lungs, with pleuritic rub below right breast.

On April 11th diarrhoea, with blood and mucus in evacuations; no protozoa; no history of dysentery; urine alkaline, albumin present with phosphates.

On April 17th gangrene threatening the right foot, which is cold and blue; pain, with loss of sensation. Urine, occasional trace of albumin, no casts, no sugar.

On April 18th left foot bluish, right foot improved; little and fourth toes very purple, tender.

On April 20th gangrene threatening both hands, more in right; blue and cold, numb, painful.

On April 21st hands look like Raynaud's disease. Feet almost recovered.

On April 28th hands a little better; fingers still black.

On April 30th feet practically recovered, but little toe of right foot still bluish; fingers improving; still black, numb, and cold.

The patient was stated to have had malaria, but no data were given.

On admission to this hospital it was noted that he showed general wasting; the radial arteries were somewhat thickened; the temporals were not evident. No abnormality of heart; tremor of hands and tongue, and unsteadiness of gait; speech normal; pupils equal, react normally to light and accommodation; knee-jerks brisk; superficial reflexes normal. Systolic blood pressure 152, diastolic 130.

The blood was examined on May 29th and June 17th, with the following result:

Red blood corpuscles 4,933,333 and 5,360,000 to the c.mm.; white blood corpuscles 7,383 and 13,750; haemoglobin percentage, 85 and 95; colour index, 0.87 and 0.95; relative white cell count shows large lymphocytes 48.5 per cent.; small ditto 6.0 per cent.; neutrophils 43.5 per cent. On the second occasion these figures were, 34.5, 18.0; 43.25 per cent. (lymphocytosis). No other peculiarities.

The urine was examined on three occasions—

May 27th: Specific gravity 1028, acid; no sugar; no albumin.

May 30th: Specific gravity 1024, acid; no sugar; no albumin.

June 26th: Specific gravity 1036, slightly alkaline, no sugar, no albumin.

The "Wassermann" reaction, for which I am indebted to Captain H. A. Scholberg, R.A.M.C.(T.), was negative in serum and in cerebro-spinal fluid. Cells in cerebro-spinal fluid 2 to the c.mm. Nitrogen in 1 c.cm. of cerebro-spinal fluid (Stanford's method; for this estimate I am indebted to Dr. R. V. Stanford, research chemist at this hospital) 14.5 hundredths of a milligram.

Although the patient was restless, garrulous, excited and grandiose, he was not, according to the above laboratory findings, a general paralytic. The photographs sufficiently indicate the state of the fingers. The dark portions were gangrenous—black, dry, and insensitive; the nails were still preserved on each finger. The skin of the fingers above the gangrenous portions was reddish-blue, tense, shiny and sensitive to touch; the gangrenous portions, although insensitive to light touch, were sensitive to pin-prick and to deep pressure. The hands generally were somewhat livid; toes, nose, and ears not affected.

On June 30th, the date of writing this report, the patient appears to be almost well from what was presumably an attack of subacute mania. There are no physical indications of general paralysis. It is noted, however, that both pupils are slightly excentric, and there is greasiness of the facial skin. Systolic blood pressure 148, diastolic blood pressure 123; mummification of affected finger-tips; line of demarcation between living and dead portions very obvious.

Raynaud, in his original thesis, published in 1862, states that, out of 25 cases of symmetrical gangrene seen by him, 20 occurred in women and 5 in men; the patients were all under 30, excepting one aged 48. Oppenheim describes the disease as developing exclusively or principally on the basis of the "neuropathic diathesis." Amongst exciting causes, emotional disturbances—fear, etc.—take first place. Sometimes the condition has been preceded by infectious disease—typhoid, influenza. Young women up to 30 years of age are the usual victims, but of the 4 cases which Oppenheim had seen 3 were men. Photographs of the condition given in Oppenheim's work are almost identical with those accompanying the present description. In Raynaud's Plate 1, showing the toes of two feet affected by symmetrical gangrene, the condition is much less advanced than in the case here recorded. For the photographs accompanying these notes I am indebted to Dr. Stanford.

There is no question in this case of ergotism. The bread eaten by this patient whilst in Egypt was the same as that issued to the troops in general. I do not recollect a single instance of this disease amongst the cases of mental disorder seen by me, whether in public institutions or consulting practice, during my twenty-nine years' experience.

I desire to take this opportunity to draw attention to the micro-quantitative methods of estimating the constituents of cerebro-spinal fluid devised by Dr. R. V. Stanford. That for nitrogen is described in the *Zeitschrift für Physiologische Chemie* (Bd. 86, H. 3, of 1913). Details of the results in a large number of cases are given in that paper, and a great many more have been dealt with since. The conclusions are summarized as follows in a memorandum kindly supplied to me by Stanford:

The number of hundredths of a milligram of total nitrogen contained in 1 c.cm. of the cerebro-spinal fluid of a patient is called the "nitrogen number" (NN). In the normal sane individual the nitrogen number is about 15. In the great majority of insane patients, not suffering from general paralysis or gross brain lesion, the NN is between 15 and 20. In a few cases it was found to be higher, but in only one case above 25. In cases of general paralysis the nitrogen number is nearly