

country and in the great work of organization, with not more than two exceptions, the spirit of compromise has been so dominant that prejudices, predilections, prerogatives and even minor principles have been willingly and gladly subordinated to the greater principles for the conservation of the greater good. This spirit—the very soul of fraternity, coupled with the spirit of industry, the spirit of culture, the spirit of patriotism—must be kept in the ascendant if we, as a profession, shall maintain our just position and discharge our full duty as citizens of this great republic.

Original Articles.

A CONTRIBUTION TO THE STUDY OF VISUAL DISTURBANCES IN BRAIN INJURY.*

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CHICAGO.

NARRATION OF CASE.¹

*Hospital Record.*²—Fred. L., was brought by patrol about 9:30 p. m., Feb. 11, 1899, with gunshot wound in the left temple, one and one-half inches back of left eye, one-half inch above zygoma (Fig. 1), the revolver having been held within a few feet of the head. Examination found the patient pale, pulse slow and full, pupils moderately dilated, cold perspiration, no focal symptoms (?); a round hole with edges blackened and inverted in the location above described. Hemorrhage severe. Can not find bullet by palpation. Next day found bullet had flattened against side of the skull, and was removed (?) by Dr. C. within a few minutes after the injury. Patient was unconscious for about 14 days; had partial aphonia.

No convulsions at any time. Summary of the pulse rate and temperature: During his hospital residence his temperature was above normal only on two occasions: on March 15, 100 F.; on March 16, 100.8 F. The pulse rate remained below normal until February 20, after which it remained normal, 72. The lowest rate, 52, was recorded February 18. No further record kept.

Diagnosed—Bullet wound of the head; concussion of the brain. Result—Cured. Discharged March 9, 1899.

Patient's Story.—The patient gives me the following history: For a few days after returning to consciousness he could not recognize familiar objects by sight; he did not know what the pictures on the wall were; on looking out of the window he could not recognize the streets or the locality. He was unable to count or name the letters of the alphabet. He was lost.

On his way from the hospital to his home he was still unable to recognize the familiar streets. He says this inability to recognize the streets lasted only two or three days after returning home. All other things that he saw he knew at this time. There were no hallucinations of vision. He was unable to call the names of the most familiar objects about the room, although toward the end of his hospital sojourn he knew the use of them. For example, he could not recall the name of a "chair," but he knew its use.

When he wanted to smoke he could not recall the name "cigar," but would indicate it by making the motions of holding and puffing a cigar. Neither the touch, taste or smell of the cigar would bring the name to his mind, but when "cigar" was mentioned to him he would immediately recognize and repeat it, but would soon forget it. The ticking of a watch

or ringing of a bell did not bring back the name of the object. He would speak of a street car as a "thing to ride in," etc. He could not recall the names of his most familiar friends, but he recognized them, for example, his brother-in-law, whose name is Robert, he would call "Lawrence" or other names.

For several months after the injury he was unable to read printed or written words. Three months after the injury he wrote a letter to his father, which his father answered. The patient was unable to read this letter after he had written it. He says he would forget the last word on going to the next.

Author's Findings After One Year.—Jan. 24, 1900, he came into my service at the Illinois Charitable Eye and Ear Infirmary, complaining of difficulty with his sight. Age 28 years. An intelligent, educated German. No history of previous disease of the eyes or dimness of vision; no history of syphilitic infection or evidence of tabes.



Fig. 1.—Showing entrance of bullet.

R. V. 20/20+, L. V. 20/20+. Pupils normal in size, and reacted normally to direct light, consensually and to accommodation. Discs slightly paler than normal. No diplopia present, right homonymous hemianopsia. Negative scotoma. Intact part of the field contracted for form and color (Fig. 2). I searched diligently for hemiopic pupillary rigidity, but it was not found.

He can not raise the right foot well, walks lame. Little reduction in strength of right hand (he is right-handed) as compared with the left. Right half of the body anesthetic, most pronounced in the foot. No paralysis of muscles of the right side of the face. Hearing normal in each ear. Taste on each side of the tongue unimpaired.

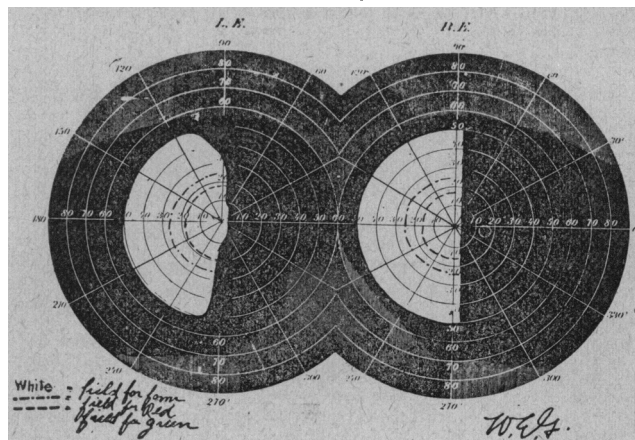


Figure 2.

He had severe headaches (not localized) for ten months after the injury. He has difficulty in remembering proper names; for example, he can not remember my name, although he knows me well. On handing him my card he has a little difficulty in reading it. He can not give the name of this hospital nor of the street, although he is acquainted with the locality. He writes spontaneously and less freely from dictation.

In view of the presence of hemiparesis, hemianesthesia and hemianopsia, I could not accept the statement of the physician who first saw the patient that the bullet had not penetrated the skull. Consequently, I did not agree with the diagnosis—"concussion of the brain"—made at the hospital. Radiographs were taken, plainly showing the bullet in the occipital region, left hemisphere (Figs. 3 and 4).

* Read at the Fifty-fourth Annual Session of the American Medical Association, in the Section on Nervous and Mental Diseases, and approved for publication by the Executive Committee: Drs. Richard Dewey, H. A. Tomlinson and F. W. Langdon.

1. Case reported to Chicago Ophthalmological and Otological Society, May 13, 1902.

2. Taken from Alexian Brothers' Hospital case record.

Condition After Three Years.—Feb. 19, 1902, three years from date of injury, he visited me again. V. R. 20/20—. V. L. 20/20—. Pupillary action normal. Field of vision same as when taken two years ago (Fig. 2). He still has difficulty in remembering names, as, or instance, he can not give my name, although when it is written he reads it.

There is no hemiplegia or hemianesthesia, but there is paresthesia of the ball of the right foot, and especially of the second toe. He locates a "sore disagreeable feeling" in the left occipitoparietal region, about the size of a silver half dollar. He has had this feeling more or less since injury. His grasp in the right hand is greater than in the left. He is not lame nor has he any difficulty in walking.

He is a finisher and painter, and is now painting, on a scaffold, the outside of one of the sky-scrapers in this city. He tells me he has great difficulty mixing paints. He can select the color if the name is given him, but he can not give the name of the color that he wants (anmesic color blindness), and as occasionally the mixing is done on an upper floor, he has to go to the basement himself instead of sending a helper to get the "color" he desires. This is especially true at the present time with shades of color that have compound names, such as bismark-brown, yellow-ochre, burnt-umber, chrome-yellow, vandyke-brown, etc. He can now give the names, red, yellow, green, blue, without difficulty when he sees them or wishes to recall the particular color. When he began paint-

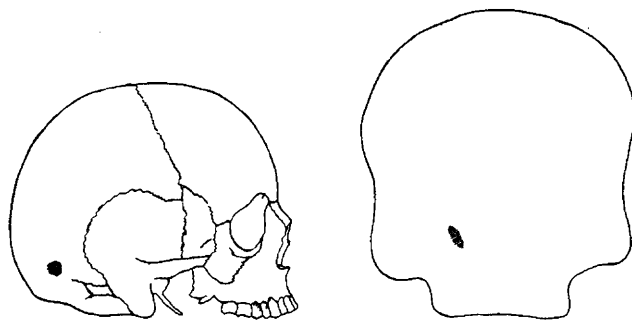


Fig. 3.

Fig. 4.

Figs. 3 and 4.—Lateral and posterior views of skull, with shaded area showing bullet. Drawings accurately reduced from radiographs.

ing again after his injury he could not even do this. He performs the Holmgren test perfectly. It should be added that while he was explaining to me his difficulty in getting the name of the particular shade he saw or desired, he enumerated a long list of names of special shades without hesitation. In this list were the names I have used above.

Appearance of Optic Discs.—In the right eye the bluish-white lamina cribrosa shows through the greater part of the optic disc, especially on the temporal side. The borders are sharply defined and the vessels but slightly changed; veins possibly a little engorged. Some connective tissue is seen at the bifurcation of the main artery, probably congenital. The left optic disc is pale, but does not have the bluish appearance of the right, and the lamina cribrosa is but dimly seen. There is some connective tissue at the bifurcation of the central artery. The outline of the margin is pronounced, with no signs of choroidal atrophy in the margin. Veins are possibly slightly engorged. No other change was observed.

Condition After Four Years.—Feb. 26, 1903. R. V. 20/20—. L. V. 20/20—. Field same as on last examination. Current speech seems to be about normal. When asked "Who is President of the United States?" he replied, "Garfield," and finally, after much effort and mispronunciation, got the name of the President correctly. For the last several months he has set himself to the task of increasing his vocabulary for names. By constant repetition he is slowly becoming able to retain permanently the names of the people he most often needs to address. He no longer forgets my name. He can now recall at will the names of a few of the principal streets, although to-day he was unable to give the name of one of two streets

passing a shop where he has worked since last November. He carries a street directory with him and underscores the name of the street he wishes to reach. Sometimes he can remember the first letter and recognize the name when he finds it on the printed list.

While before his injury he was an omnivorous reader, he now reads at most disconnected newspaper paragraphs and letters from his father. In each letter there are words which he is unable to make out, and he usually writes his father for an explanation (alexia). The patient is of the opinion that the rate of progress from complete alexia (pure), in 1899, to his present attainment has been less during the last year than formerly. He has made no systematic effort to train this faculty. He says he has especial difficulty with long words: that seeing the left half of the word, and then, as he turns his eyes to increase his field, seeing the other half, he forgets the first, this explanation does not entirely satisfy him, but he is unable to add to it. At 15 inches from his face he is unable to keep in his eye a word longer than $\frac{3}{4}$ of an inch. At closer distance the length of the word seen is more limited. He thinks his chief difficulty is with long and (to him) unfamiliar words. Formerly he had the same difficulty with short words. By intently looking at the word which puzzles him he is usually

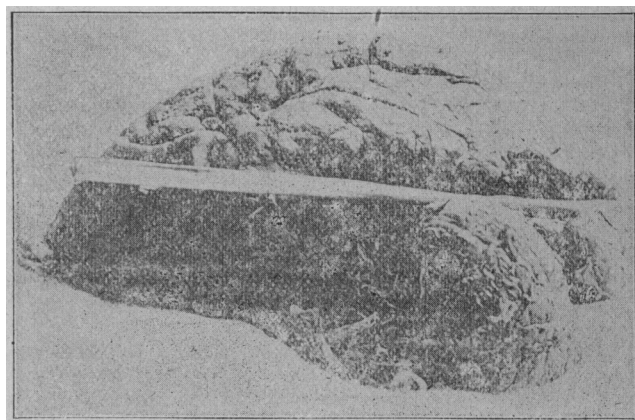


Fig. 5.—View showing assumed line of transit of bullet. The third and fourth temporal and middle convolutions suffered.

able to read it after a time. He has had no trouble in writing at any time.

In determining the probable localization of the lesion, I have had the good fortune to secure the co-operation of my friend and colleague, Dr. William T. Eckley, professor of anatomy in the College of Physicians and Surgeons, the School of Medicine of the University of Illinois. Dr. Eckley also secured the assistance of Prof. Harry E. Santee. Their methods and conclusions are given below.

Anatomic Report.—"With the able assistance of Dr. Harry E. Santee, we are prepared to submit the following report, in which we duplicate to the best of our ability, on a cadaver, the line of transit and brain tissue traversed by a bullet in the above case.

"As indicated by the skiagraphs submitted by you to us, the bullet now occupies in your patient a space three-quarters of an inch above and one inch and a quarter to the left of the external occipital protuberance, in the left cerebral hemisphere in contact with or very close to (probably $\frac{1}{4}$ inch) the skull in this region. A scar on the patient, just above the zygomatic arch and in the junctional angle of the frontal and zygomatic processes of the malar bone, marks the place of entry of the bullet. On a cadaver's head, conforming in measurements to the head of the patient, we located the entry of the bullet and its resting place, by comparison with head of patient and skiagraphs; and by trephining through the skull at these extreme points, succeeded in passing a steel spike $\frac{1}{4}$ inch in diameter through the brain in the probable line followed by the bullet. The hypothetical line of transit

passes just above, but does not touch the superior border of the petrous part of the temporal bone; and this being the only structure in the area under consideration, capable of producing deflection of the missile, we concluded the same followed a straight line, and on this assumption we venture to enumerate the brain regions necessarily traversed by the bullet.

"(a) The greater part of the third temporal convolution, in which the so-called naming center is located, was destroyed.

"(b) The fourth temporal convolution is almost destroyed, the upper part especially.

"(c) The anterior end of the inferior occipital convolution is bruised.

"(d) The outer surface of the middle occipital convolution is penetrated.

"(e) The perpendicular fasciculus of association fibers was in part cut off. This band of long association fibers, while of doubtful function, extends from the inferior parietal to the second and third temporal convolutions; and also joins the

manent lesion, the hemianopsia in this case being due to severance of all the visual bundle of radiations of Gratiolet in this hemisphere.

The *mind-blindness* can readily be accounted for by general circulatory and consequent nutritional disturbance of the brain resulting from the injury.

The *verbal amnesia* would seem to be of the visual type. It can not, however, clearly be classified as "visual aphasia," as described by Freund¹ and others, because names of objects seen are not recalled by aid of other senses. For example, the smoking of the cigar which would call into co-operation with sight the senses of touch, taste and smell, did not bring back the name. Listening to a watch or a bell did not bring back the name "watch" or "bell." However, his loss of memory for substantives, especially proper nouns, has been classified as visual aphasia by some authors, the name being immediately recognized when heard but soon forgotten.

It is a significant fact that in 25 cases of aphasia in various forms, in otitic abscess of the left temporal region, collected by Merckens,² the only symptom common to all, was visual aphasia. Oppenheim³ says this symptom "appears to be caused by lesions at the border of the left occipital and temporal lobes, and

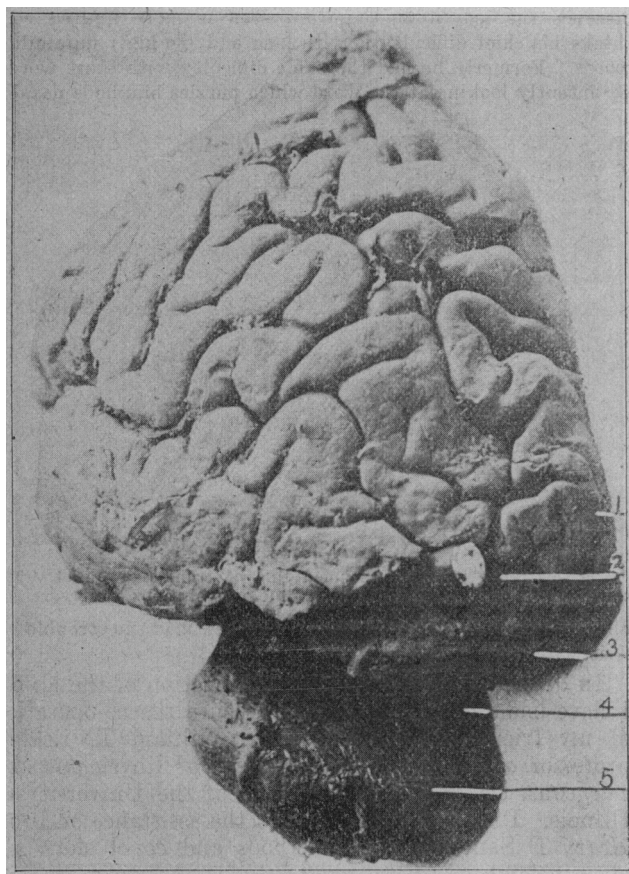


Fig. 6.—Showing distance of posterior part of wound from the cuneus, about one inch. 1. Sup. oc. convolution. 2. Middle oc. convolution. 3. Inf. oc. convolution. 4. Transverse fissure. 5. Cerebellum.

superior with the middle and inferior occipital convolutions and with the fusiform gyrus of the temporal lobe.

"(f) The association fibers between the tentorial and convex surfaces of the occipital lobe have been in part cut off.

"(g) The inferior longitudinal fasciculus connecting the temporal and occipital lobes, thus associating the center of hearing and visual memory respectively, may have been cut off or injured by concussions."

ANALYSIS OF FINDINGS.

This lesion, not involving the internal capsule (three-fourths inch away), the hemianesthesia and hemiplegia, principally confined to the right leg, being temporary, could be accounted for by hemorrhagic disturbance of the tracts leading from the occipital cortex as they pass forward to enter the internal capsule; and not by per-

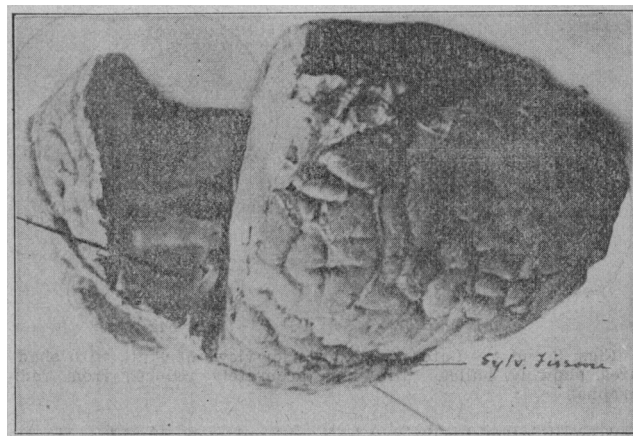


Fig. 7.—The needle represents (a) course of bullet through third and fourth temporal convolutions; (b) relation of course of missile to descending and posterior horns of lateral ventricle; (c) relation to radiations of superior longitudinal fasciculus, optic radiations and inferior longitudinal fasciculus.

so extensive as to divide the conducting tracts leading from the posterior lobes to the center for tone pictures." C. K. Mills⁴ teaches that there is a concept area for names in the mid-temporal region, probably in the posterior fourth of the third temporal and the occipital (left) in this region, which he calls the "naming center."

It can not be definitely determined by the findings in this case whether the verbal amnesia is a result of destruction of the greater part of the cortex in the third and fourth temporal convolutions (posterior) and injury to the cortex of the anterior end of the inferior occipital convolution; or of subcortical lesion in the middle occipital convolution, extending backward nearly or entirely through it or a result of all the lesions.

However, in view of the fact that there is little or no evidence heretofore brought forward of subcortical lesion in the inferior occipital convolution producing this symptom; and on the other hand, much

1. Freund: *Archiv. für Psych. u. Nervenkrankh.*, xx, S. 276

2. Merckens: *Deut. Zeit. f. Chir.*, B. 60, S. 417.

3. Oppenheim: *Diseases of the Nervous System*, Ed. 1900, p. 464.

4. Mills: *THE JOURNAL A. M. A.*, October, 1902. Mills and J. W. O'Connell: *Jour. of Nerv. and Mental Dis.*, vol. xx, 1895.

evidence has been accumulated showing that cortical lesion in the third and fourth temporal convolutions produces this symptom, this case furnishes strong corroborative evidence in favor of the teaching of Professor Mills and others, that there is a concept area for names in the third or fourth temporal convolutions and anterior part of inferior occipital convolution, or in all.

The ability to increase his vocabulary for proper names shows that there are other cells that have this function, though undeveloped, and for the reason given below in connection with word-blindness, this undeveloped word center is probably in the right hemisphere.

Amnesic Color Blindness.—The patient was unable to name any color at sight for many months. At the present time (four years after date of injury) he is unable to give the names of particular shades, such as bismark-brown, chrome-yellow, etc., although being a painter, if given the name he can select any shade that he uses, but he can name the ordinary colors when he sees them, such as brown, yellow, etc. His color sense is normal, as he performs the Holmgren test perfectly. Therefore it is strictly an aphasic symptom, and has nothing to do with disturbance of the color sense.

The presence of this symptom in lesion of the left middle occipital and third and fourth temporal convolutions proves conclusively the correctness of Willbrand's⁵ supposition that the lesion must be in the left hemisphere in a right-handed person. But in addition it shows this to be not the whole truth, for his partial reacquirement of this function by persistent training during a period of four years, can be explained only as an education of an undeveloped center in the right hemisphere for the same reasons as those given below for the reacquirement of names and the reacquirement of the ability to read. This lesion is evidently produced by severance of the path leading from the center for color vision at the bottom of the calcarine fissure (Henschen),⁶ anteriorly to the speech center.

Word-Blindness.—The pure word-blindness, according to the patient's explanation is probably partly a forgetfulness of parts or the whole of a word preceding the part in mind—a type of verbal amnesia. This aphasic disturbance was complete for several months (more than three months). Improvement has been gradual until at the present time he can read, although with difficulty. The gradual recovery from this aphasic condition can not be accounted for by a reparative process taking place in the left middle occipital and third and fourth temporal convolutions, the bullet being larger than 22-caliber.

Phelps' reports a case of gunshot wound of the brain, in which thirteen years afterward the bullet tract was converted into a membranous canal. Doctors Sanger Brown⁸ and Schaefer found, six months after removing the occipital lobe of a monkey, that the cut surfaces had remained unchanged, the edema *ex vacuo* taking the place of the lost part. It is hardly necessary to add that evidence is lacking of reparative process in lesion of nerve tissue in brain and cord.

We are compelled to assume that other specialized nerve cells have taken on this function, and from our knowledge of the bilateral nature of other functions of

the brain it is reasonable to assume, according to Bastian's⁹ teaching, that the right hemisphere has an undeveloped word center which is capable of education, contrary to the views of Dejerine¹⁰ and Lichtheim that only the left hemisphere is concerned in word-blindness. In the three other cases of pure word blindness (Dejerine,¹⁰ Redlich and Wyllie) so far reported, in which autopsies were obtained, the lesion found in each case was in the white substance in occipital region (left) together with damage to lingual fusiform hippocampal gyrus and cuneus. In these cases the cortical region injured is destruction of the greater part (posterior) of the third and fourth temporal convolutions and the bruising of extreme anterior end of left inferior occipital convolution, the cortical area common to this case and another case reported being the fusiform gyrus.

Therefore, in this case, it is altogether probable that the lesion causing this alexia is mainly due to the injury of the white substance in the occipital region, involving the lower part of the inferior longitudinal fasciculus (Dejerine¹⁰) and not due to direct cortical lesion.

Dyslexia.—The distaste which the patient experiences for reading (dyslexia) is partly due to the great reduction of the field resulting from the hemianopsia and from the concentric contraction of this half field to 50" for form (20" for red and 10" for green); and partly due to partial word-blindness and also to fatigue which comes from developing a function in the right hemisphere comparable to the discomfort evinced by a child in learning to read.

Optic Atrophy.—There being no history of previous severe illness nor dimness of vision or other ocular trouble, nor evidence of tabes nor syphilitic infection present, it must therefore be assumed that the gunshot wound is the cause of the change in the discs. Primary atrophy of optic discs, if we adhere to the doctrine of the neuron theory, is almost inexplicable when the lesion is behind the central ganglia, as it is in this case. The "indirect"¹¹ theory, lately put forth is the only explanation so far offered.

Prognosis.—The unusual fact of the toleration of the bullet by the brain for a period of four years may be due to its present location, being possibly on the tentorium. The irritation of this membrane may have produced proliferation of the connective tissue about the bullet, encysting it. If this be true, for this reason the prognosis in this case is good. Surgical interference would probably improve prognosis.

CONCLUSIONS SUGGESTED.

The findings in this case suggest the following conclusions:

1. A cortical lesion the size of a 32-caliber (?) bullet, involving the greater part (posterior) of the third and fourth left temporal convolutions and bruising of extreme anterior end of inferior occipital convolution, and a subcortical lesion penetrating the middle occipital convolution at the same level, produces verbal amnesia, especially for names of objects seen and having been seen, particularly those which have proper names (concrete nouns), and for names of colors, especially names of special shades, such as bismark-brown, etc. (amnesic color blindness), and pure word-blindness (alexia), which may be in part a form of visual verbal amnesia.

5. Willbrand: Quoted from Swanzy, *A System of Diseases of the Eye*, Norris and Oliver, vol. iv, p. 562.

6. Henschen: "Brain," London, 1893, p. 170.

7. Phelps: "Traumatic Injuries of the Brain," p. 67.

8. Sanger Brown and Schaefer: *Philo. Trans. Royal Society of London*, vol. clxxix (1888) B., pp. 303-327.

9. Bastian: *Allbutt's System of Medicine*, vol. vii, p. 437.

10. Dejerine: Quoted from Swanzy, "A System of Diseases of the Eye," Norris and Oliver, vol. iv, p. 561.

11. Barker: *The Nervous System*, p. 18.

2. Verbal amnesia is a result of lesion in the cortex of third and fourth temporal convolutions, left hemisphere and injury to cortex of anterior end of inferior occipital convolution.

3. The amnesic color blindness and alexia result from subcortical lesion in left middle occipital convolution.

4. The reacquirement of these functions in a man 28 years of age is a slow and tedious process of education of the specialized though undeveloped cells of the right hemisphere, requiring years for even partial accomplishment.

5. Injury to the optic radiations of Gratiolet may produce negative scotoma, contrary to the teaching that such a lesion produces positive scotoma.

6. Primary atrophy of the optic discs occurs in lesions of the left temporo-occipital region.

DISCUSSION.

DR. H. T. PERSHING, Denver—It is somewhat precarious to infer the place of a lesion from an attempted reproduction of it on the cadaver of another person. From its clinical features, I think the case was one of subcortical visual aphasia. The fact that the patient could write showed that his visual memory was good, but the fact that he could not read what he had written showed that there was an interruption of the association fibers. This explanation would also account for his inability to recall nouns, especially proper nouns, because, according to the best theory we have to offer, a noun is recalled by the visual image of the corresponding object. My opinion, therefore, is that the case was one of subcortical visual aphasia, due to a lesion in the immediate vicinity of the angular gyrus.

DR. H. T. PATRICK, Chicago—I feel inclined to support Dr. Pershing's view. I understood from Dr. Gamble's paper that the last examination of the patient's eyes made recently showed a vision of 20/20 in either eye, and yet there was double optic atrophy. How does he reconcile these statements? Furthermore, how could this injury cause double optic atrophy? The course of the projectile, as traced by Dr. Gamble, would hardly account for the motor and sensory disturbances in the right lower extremity. In order to account for those symptoms, the bullet would have to go a little deeper and start a little further forward, which would be a much simpler explanation than to invoke the aid of disputed points in anatomy and physiology or theories that are as yet unproved. It has been shown by Horsley and others, especially since recent wars, that the injury inflicted by a projectile passing through the brain is not accurately represented by the track of the bullet. The latter has an explosive action, and this gives rise to widespread injury which is more or less in proportion to the velocity of the projectile. The injury inflicted by a bullet passing through the brain is not the same as that resulting, for example, from excision of a certain cortical area. By a rapidly moving bullet, as just stated, the brain is somewhat injured, not destroyed, to a certain extent outside the course of the projectile. Such an injury does not involve the question of the reuniting of nerve fibers or the regeneration of nerve cells. It simply involves a reparative process in the immediate neighborhood of the injury, such as occurs after traumatism or embolism or hemorrhage. There is only a partial injury to the brain tissue, and it is usually followed by at least partial repair. It is not necessary to invoke the theory of the re-education of latent nerve cells or centers in the adult.

DR. J. H. MCBRIDE, Los Angeles, Cal.—About thirty years ago there was reported in London the case of a right-handed child that suddenly became aphasic, indicating a lesion in the left motor speech center. Within a year, speech was entirely recovered. Two years later there was another attack of aphasia, which was permanent. The child died six months later, and two lesions were found in the brain, one involving the third left frontal convolution and the other the third right frontal. In 1890 there was admitted into the Milwaukee

Hospital a man who was supposed to be suffering from chronic epilepsy. He stated that twenty-seven years before he had been shot in the eye. The man died, and at the autopsy the bullet was found under the left temporal bone. It had lodged in the second left temporal convolution and gradually gravitated to the point where it was found.

PROF. W. T. ECKLEY, Chicago—Speaking on the basis of the examination that Dr. H. E. Santee and I made, the anatomic side of the discussion of this valuable contribution will give a brief summary of the known anatomy and physiology of the temporo-occipital region, looking to the pathologic possibilities incident to disease or trauma. We shall consider the commissures, the association fibers, and the sensorial cortical centers, which latter receive impulses from corresponding peripheral regions, and give origin to audition, olfaction, gustation and vision.

The commissural fibers connect portions of opposite hemispheres, forming definite structures—the corpus callosum, the anterior commissure and the hippocampal commissure.

The corpus callosum connects both similar and dissimilar parts in all regions of the hemisphere except the olfactory bulbs and the antero-inferior portions of the temporal lobes. The anterior commissure, supplemental to the corpus callosum, connects the two olfactory lobes and portions of the temporal lobes. The hippocampal commissure connects the uncus, optic thalamus and hippocampus major of one side with similar structures of the opposite side.

Association fibers in general connect parts of the cortex on the same side of the hemisphere. Short association fibers connect convolution with convolution; long association fibers connect lobe to lobe. The long association fibers are the cingulum, fornix, uncinate fasciculus, superior longitudinal fasciculus, inferior longitudinal fasciculus, occipito-frontal fasciculus, and perpendicular fasciculus.

The cingulum is in the falciform gyrus and almost completely surrounds the corpus callosum. It associates the frontal lobe and uncus. The fornix connects the corpus albicans and thalamus with the uncus and hippocampus major. The uncinate fasciculus arches over the stem of the sylvian fissure, connecting the uncus with the frontal pole and orbital convolutions. Lesions in cingulum, fornix or uncinate fasciculus produce disturbances in smell, because they are connected with the rhinencephalon.

The superior longitudinal fasciculus, on the outer surface of the foot of the corona radiata, connects the frontal temporal and occipital lobe. It passes over the insular region and at the posterior end of the sylvian fissure bends around the putamen and passes to the temporal lobe. It sends numerous radiating fibers into the occipital lobe. This fasciculus associates the motor speech center with the visual and auditory memory centers. If it is destroyed, the patient can see and hear perfectly and recognize things seen and heard, but he has no power to express in words his visual and auditory memories.

The inferior longitudinal fasciculus extends along the whole length of the temporal and occipital lobes, uniting the auditory and visual memory centers. In the occipital lobe it is to the outer side of the optic radiations, from which it is to be distinguished by the greater coarseness of its fibers.

The fasciculus occipito-frontalis is between the cingulum and superior longitudinal fasciculus, extending from the cortex of the entire frontal lobe to the occipital lobe, where its fibers diverge, fan-like, and enter the descending and posterior horns of the lateral ventricle.

The perpendicular fasciculus extends from the inferior parietal convolution to the second and third temporal convolutions anteriorly; posteriorly, it joins the superior, middle and inferior occipital convolutions with the fusiform gyrus of the temporal lobe.

The auditory sensory center is in the transverse temporal gyri and third and fourth fifths of the superior temporal convolution. It is represented peripherally by the cochlea, and impulses of hearing reach the auditory sensory center over the cochlear path.

The auditory memory center is intimately associated (1) with the auditory sensory center, by short association fibers; (2) with the motor speech center, by the superior longitudinal fasciculus; (3) probably, with the naming center, through a group of association fibers.

The olfactory center is probably in the mucus. It is represented peripherally by the schneiderian membrane. Impulses of smell reach the uncus through the fibers of the olfactory tract, cingulum and uncinate fasciculus.

The gustatory center is probably in the cortex of the hippocampal convolution, posterior to the olfactory center. It is represented peripherally by the end organs of the glossopharyngeal and chorda tympani nerves. Impulses of taste reach the center through the medial fillet and internal capsule.

The visual sensory center is in the cuneus and lingual convolution, on the mesial surface of the hemisphere, and extends somewhat into the convex surface of the occipital lobe. Its peripheral representative is the retina, and impulses here originating reach the higher visual sensory center, in the cortex of the cuneus, via the optic nerve, tract and radiation. The visual sensory center is connected to the visual memory center by short association fibers.

The visual memory center is in the angular and occipital convolutions. It is associated with the visual sensory center by short association fibers, and with the motor speech center, in the central gyri and inferior frontal convolution, through the superior longitudinal fasciculus. Injury of the visual sensory center produces blindness. Injury of the visual memory center produces mind blindness. Destruction of the tract, associating the visual memory center and the motor speech center produces sensory aphasia.

Let us now analyze the sensorial cortical centers in the occipital and temporal lobes, with reference to destruction of (a) the sensory center; (b) the memory center; (c) the tract of association fibers connecting several memory centers with the motor speech center. If the visual sensory center is destroyed, there is blindness in the opposite eye. If the visual memory center is destroyed, vision remains, but the patient has no power to recognize through seeing. If the tract connecting the visual memory center with the motor speech center is destroyed, vision and recognition of things seen will be perfect, but the patient will be unable to express in words his visual memories. If the auditory sensory center is destroyed, there is loss of hearing in the ear of the opposite side. If the auditory memory center is destroyed, the patient hears, but there is failure of recognition, through hearing. If the tract connecting the auditory memory center with the motor speech center is destroyed, hearing and recognition of things heard will be perfect, but the patient will be unable to express in words the auditory memories. In the light of analogy there must be, linked to the olfactory and gustatory centers, corresponding olfactory and gustatory memory centers. If the olfactory sensory center is destroyed, there is loss of smell in the nose of the corresponding side. If the olfactory memory center is destroyed the patient smells, but can not recognize through smelling. If the tract connecting the olfactory memory center with the motor speech center is destroyed, smelling and recognition of things smelled will be perfect, but the patient will be unable to express in words his olfactory memories. If the gustatory sensory center is destroyed, there will be loss of taste. If the gustatory memory center is destroyed, the sense of taste is perfect, but the patient can not recognize things tasted. If the tract leading from the gustatory memory center to the motor speech center is destroyed, taste and recognition of things tasted will be perfect, but the patient will be unable to express in words his gustatory memories.

In the interesting clinical case under discussion, the patient could see and recognize things seen; hear and recognize things heard; smell and recognize things smelled; taste and recognize things tasted. Therefore, the respective sensory and memory centers of vision, audition, olfaction and gustation were unimpaired. The patient, however, could not express in words, visual, auditory, olfactory and gustatory memories. Therefore, there must have been interruption in the long association

fibers connecting the cortical memory centers of these special senses with the motor speech center; and the injury must have been to the radiating fibers in the occipital lobe where the superior longitudinal fasciculus turns forward into the temporal lobe, around the putamen. In view of the symptoms—visual, auditory, olfactory and gustatory aphasia—is not the easiest explanation of this group of symptoms to be found in the assumption of a common naming center, interposed somewhere between the motor speech center and the cortical memory centers previously mentioned?

DR. W. E. GAMBLE—The lesion producing the alexia is probably the subcortical injury in the left middle occipital convolution involving the inferior longitudinal fasciculus, and not the cortical lesion in the third and fourth temporal convolutions. I fail to see on what known or supposed physiologic basis Dr. Patrick assumed that the lesion in this case is higher up than Dr. Eckley has demonstrated, that is, involving the region of the angular gyrus. This region is held by Bastian and Gower to be the center for visual memories, while the later clinicopathologic work of Henschen leads him to the conclusion that the whole convex surface of the occipital lobe has this function. Wilbrand, Nothnagel and others believe that the whole occipital lobe, except where the center of vision resides, stores up visual memories. In my case the visual memories are intact, with the possible exception of alexia. In answer to Dr. Patrick's question: "How can your patient have optic atrophy and still see 20/20 with each eye?" At first thought such a statement would seem a paradox. However, by reference to the diagram of the field of vision (Fig. 2), it will be seen that his half fields are concentrically contracted for form to 50 degrees, which shows that a large part of the optic nerve (peripheral fibers) has lost its function. The maculopapillary bundle has remained intact, which accounts for the good central vision. I do not share in Dr. Patrick's difficulty of understanding how the patient had temporary hemiparesis and hemiplegia, especially confined to the right foot, in the lesion described. As I stated in my paper, the tracts passing from the occipital cortex to the internal capsule were more or less injured by the hemorrhage, not destroyed by the bullet, the internal capsule not having been injured.

A CASE OF PERFORATING WOUND OF BOTH CEREBRAL HEMISPHERES.*

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Last November, while attending a meeting of our county medical society I was summoned, in company with Drs. McClintock and Bowen, to a hotel near by in which a guest of the house was said to have attempted suicide. We found a young man about 19 years of age writhing on the floor with a bloody wound on the right temple, the result of a revolver shot fired about five minutes previously.

The hemorrhage was not great, but to prevent its becoming greater we tried to keep the patient quiet till an ambulance should arrive. In this we were not entirely successful, for he succeeded in getting up. He seemed very reticent on being questioned, although he talked enough to show mental coherence. We discovered that he had on only one cuff, whereon the patient informed us distinctly that the missing cuff was in his overcoat pocket, where it was found. Thus within a few minutes after the wound was inflicted, full mental operation was apparent, the senses of sight and hearing were normal, comprehension of spoken language was complete and the ability to frame and express a correct reply unimpaired. There was also manifest co-ordinate movements.

Examination at the hospital an hour later showed what was in effect a perforating wound of the brain, for the bullet, which was from a 32-caliber revolver, was found outside the skull on the left side, nearly opposite the point of entrance, which was

* Read at the Fifty-fourth Annual Session of the American Medical Association, in the Section on Nervous and Mental Diseases, and approved for publication by the Executive Committee: Drs. Richard Dewey, H. A. Tomlinson and F. W. Langdon.