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Tumor of the Brain, with Double Nasal
Hemianopsia.

CLINICAL LECTURE DELIVERED AT ST. LUKE'S HOSPITAL.

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Professor of Nervous and Mental Diseases in the Medical Department of the
University of Colorado; Neurologist to the Arapahoe County and
St. Luke's Hospitals, Denver, Colorado.



[REPRINTED FROM INTERNATIONAL CLINICS, VOL. I., SIXTH SERIES.]

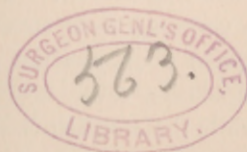
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GENTLEMEN,—I wish to call your attention to-day to this patient, Alexander A. He is twenty-three years of age, single, a Swede, and his occupation is that of a track-walker. He has been in Colorado six years. The family history is good,—or, in other words, negative,—as far as determined in relation to nervous troubles, insanity, cancer, or tuberculosis. He was always well up to nine or ten months ago, when he began to have headache, which was mostly frontal and just above the eyes. From the region above the eyes the pain extended over the entire forehead, and then to the back of the head. The headache, which he had at that time, lasted four days. It then was absent for two months, when it returned and was localized in the frontal and occipital regions. Since this time he has had headache at intervals lasting for a week or more at a time, and preventing sleep. He says the pain has generally been worse at night, but that he experienced pain in the morning as well. With the headache he would, occasionally, have a vomiting spell coming on in the morning. Just here I wish to impress upon you the significance of these two symptoms: persistent headache, severe enough to prevent sleep, and vomiting occurring while the stomach is empty. They are strong evidences of organic disease of the brain. He continued to work, as track-walker, until August 1, about five weeks ago, when his gait and sight became so impaired that he had to cease working. He entered this hospital August 18, 1894. I did not see him when he was first here, but I understand that it was impossible for him to walk or stand without assistance when he was admitted. After being here for a short time

he left the hospital greatly improved, after taking large doses of potassium iodide and mercuric bichloride. He returned to Pueblo, but he soon relapsed. He entered this hospital again August 23, with his sight more involved than when he left, and I was asked to see him. The following are my notes of the examination, as made at that time.

He walks backward and forward while his eyes are closed without showing any ataxic symptoms if he takes his time and is not hurried, but standing with his feet close together causes at first some swaying of the body. After remaining a short time in this position he maintains his equilibrium perfectly. Unsteadiness of the legs, which is slight, is most pronounced after hurried efforts in walking. After being on his feet for some time a slight tremor is observed in the legs. Prolonged or violent exertion of the arms causes a coarse jerky movement in the arm muscles, while simply holding the arms in an extended position for a short time gives rise to a fine tremor, which is a little more pronounced in the right arm than in the left. The leg and arm muscles are strong: dynamometer, right, 150; left, 130. Knee-jerks, both increased, the right a little more than the left. Ankle-clonus: right, a slight suggestion on first making forced dorsal flexion of the foot, but it soon ceases; left, absent. Plantar reflexes absent. Cremaster and abdominal reflexes absent. Deep reflexes of the arms, especially the biceps and triceps, increased. Tactile, pain, temperature, localization, muscular, pressure, posture, and joint senses normal. Taste and smell show no deviation from the normal. Hearing: watch, right, $\frac{1}{2}$; left, $\frac{1}{2}$; tuning-fork is heard better in the left ear. Eyes: right, vision = faint object-perception on the temporal side, nasal side is blind; pupil is widely dilated, reacts feebly to a bright light on temporal side, not at all when the light is on nasal side, but the whole iris responds fairly well to accommodation; all the external ocular muscles act normally; ophthalmoscope shows marked papillitis, with narrowing of the arteries, distended veins, and abundant exudate which completely covers most of the vessels as they pass over the disk; the swelling is great, requiring + 7 dioptries to see the centre of the nerve distinctly. Left, vision = faint light-perception on temporal side, nasal side is blind; pupil same size, or a little larger than right; reacts feebly to a bright light on temporal side, not at all on nasal side, but the entire iris responds fairly well to accommodation; all the external ocular muscles act normally; ophthalmoscope shows marked papillitis and beginning atrophy, with arteries very small and veins less distended than in right eye, and considerable exudate, + 6 dioptries being necessary to see the nerve distinctly.

His mental condition is normal. He suffers with headache and vomiting considerably.

He was placed upon increasing doses of potassium iodide and one-twelfth grain of mercuric bichloride, notwithstanding he denied syphilitic infection.

To-day the dynamometer registers, right, 130; left, 110. Before, the record was: right, 150; left, 130. The knee-jerks are exaggerated over what was found on the previous examination, and the right is still increased more than the left. The left eye is absolutely blind, but he still has light-perception on the temporal side of the right eye, although this field is greatly contracted, so that the pupil only reacts to light when the candle is brought near to the perpendicular axis of the eye from the temporal side. The swelling of the optic disks is less than on the former examination, and atrophy has begun to be apparent in the intraocular portion of each optic nerve. There is no discoverable disease of the thoracic or abdominal viscera.

In the light of the history and examination of this man's condition, I will ask you to follow me in an analysis of the symptoms.

The symptoms point so obtrusively to an affection of the central nervous system that the first question that we need consider is whether the cord or brain is the primary seat of the trouble. The only affection of the cord with which acute optic-nerve changes are associated is acute diffuse myelitis, and in a few of these rare cases the optic-nerve inflammation has preceded the spinal-cord affection by several days. In none of the cases reported has there been prominent swelling of the disks, and in all the myelitis has been violent, unmistakable, and has run a rapid and usually fatal course in a few days or a few weeks. The presence, then, of well-marked choked disks and the absence of all symptoms of myelitis enable us to locate this man's trouble in the brain.

The next question which naturally suggests itself is, are the symptoms due to an organic or functional trouble? The most pronounced symptom in the present instance is the double optic neuritis, with well-marked swelling of the disks. Papillitis—or choked disk, as this condition is usually termed—occurs from three conditions other than organic brain-disease,—anæmia, kidney-disease, and acute or sub-acute lead-poisoning. The anæmia that gives rise to optic neuritis is usually profound, the subject, most commonly a young female, often complaining of great weakness, breathlessness, and cardiac palpitation, and the swelling of the disks is probably never as great as we find it in the subject before us. Our patient has rosy lips and a ruddy com-

plexion, so that all suspicion of anæmia playing any part in this man's eye trouble may be at once dismissed. In the absence of all evidence of renal disease, such as albuminuria, tube casts, hypertrophy of the left heart, increased tension of the pulse, œdema, and the peculiar facial expression so commonly seen in serious kidney trouble, we are justified in attributing the papillitis to other causes; besides, the swelling of the optic disks from kidney-disease is probably never very great, and degeneration of the retina, albuminuric retinitis, usually accompanies choked disk due to disease of the kidneys. Lead-poisoning giving rise to distinct papillitis is attended with pronounced cerebral symptoms, such as great cephalalgia, delirium, and convulsions, and well-marked gastro-intestinal disorders, such as nausea, vomiting, severe colicky pains, diarrhœa or constipation, and other evidences of saturnism, most of which are absent in the man before us. We have, then, in the choked disks in this instance positive evidence of organic brain disease. There are other symptoms in this case which point to organic disease of the brain; but persistent headache, worse at night and severe enough to prevent sleep, especially when associated with apparently causeless vomiting, is one of the most significant. Having excluded anæmia, renal disease, and lead-poisoning in our patient, the association of double optic neuritis, hemianopsia, severe headache, and causeless vomiting is about as strong evidence as we need to make the diagnosis of organic disease of the brain.

Accepting the evidence as conclusive that our patient is suffering from organic disease of the brain of some kind, our next procedure will be to inquire into its nature. For diagnostic purposes these lesions may be divided into sudden, acute, and chronic. Sudden lesions are vascular in their nature, due to hemorrhage, embolism, or thrombosis, and produce decided cerebral disturbance in a few minutes to a few hours. There is, then, no difficulty in excluding a vascular lesion as the cause of this man's trouble. The acute lesions comprise the acute inflammations of the brain and its membranes. These are usually attended with fever, headache, and delirium, and reach their height during the first or second week. The gradual onset of this man's brain trouble and the chronicity of the symptoms, now extending over a period of eight or nine months, compel us to class his affection in the chronic variety.

Without going into a discussion of the various chronic organic lesions of the brain for the purpose of differential diagnosis, the presence of the enormous degree of swelling of the optic disks in the present instance justifies us in excluding, without further consideration, all

chronic diseases save abscess and tumor. It is not always easy to differentiate chronic abscess of the brain from tumor. Both are often attended with headache, vomiting, and optic neuritis, although it is rare to find an extreme degree of swelling of the optic disks in abscess. Injuries to the head may be the cause of either. The early history of chronic abscess in some cases is that of acute organic brain-disease, attended with rigor, fever, and other well-pronounced symptoms, followed by a period of variable length during which cerebral symptoms are more or less obscured until the explosive or terminal stage is reached. I wish, however, to call your attention to the fact that a normal or subnormal temperature is common throughout the course of chronic abscess of the brain, up to the beginning or near the end of the terminal period. Tumor is gradual in its onset and development, with a steady augmentation of symptoms indicating the serious nature of the lesion. In the absence of injury to the head, ear disease, or other causes of suppuration which are commonly found in abscess, the symptoms present in this case point to tumor of the brain, rather than abscess.

Hemianopsia, with hemiopic pupillary reaction, does not occur from abscess. The gradual increase of cerebral symptoms, the pronounced swelling of the optic disks, the hemianopsia, with hemiopic loss of pupillary reaction to light, in the absence of all causes and most of the leading symptoms of chronic abscess, justify us in making the diagnosis of tumor of the brain.

Having satisfied ourselves that this man is suffering from the effects of a growth in the brain, are we able to localize it with any degree of accuracy? A tumor in any part of the brain may give rise to headache, vomiting, and papillitis. Headache is rarely absent in tumor of the brain, but its seat of greatest intensity by no means corresponds to the location of the morbid process. The pain, as a rule, is most severe when the growth is near the surface of the brain, and when the meninges are involved its seat of greatest intensity often corresponds to the location of the tumor; especially do we find this verified in growths situated on the convex surface of the brain. When the centrum ovale is the seat of the morbid process frontal headache is said to be most common; basilar growths are usually attended with frontal and occipital headache; when the tumor is below the tentorium occipital pain, which often extends down the back of the neck, is common. Unilateral occipital pain usually corresponds to the side of the brain on which the growth is situated. I wish to remind you that, while the headache of tumor of the brain is an aid in the

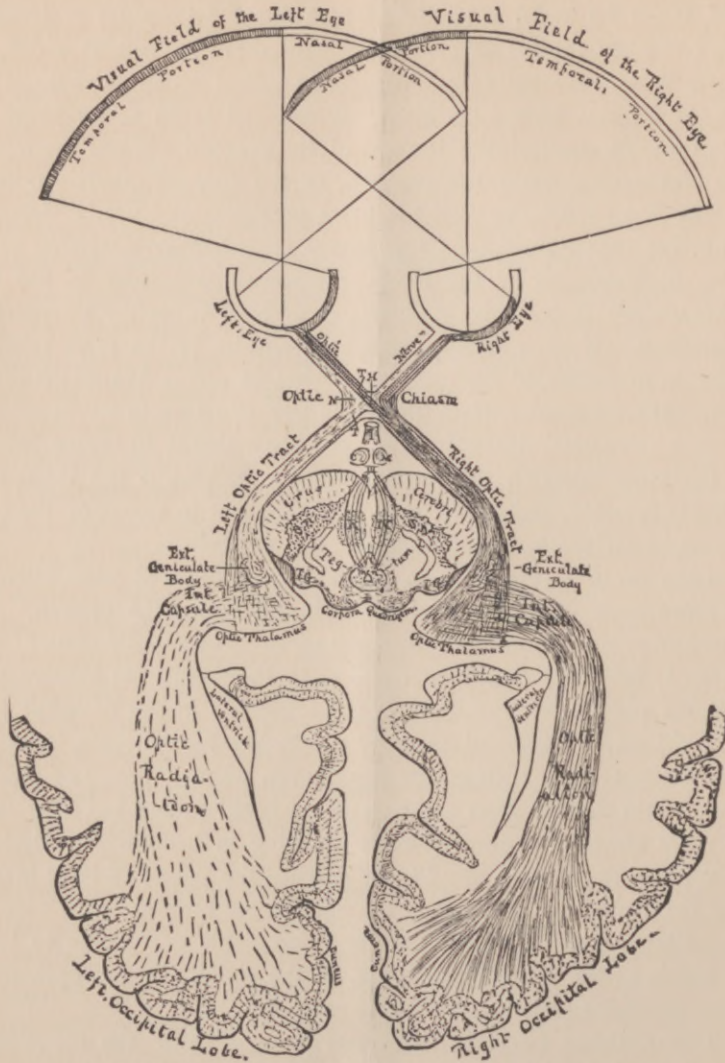
localization of the growth, it is rarely a reliable one, for not infrequently we find that a tumor on one side of the brain gives rise to pain on the opposite side, and that occipital growths sometimes cause severe frontal pain. The headache from which this patient has suffered, taken by itself, is of almost no value for purposes of localization, but when studied in connection with other symptoms which are present it is corroborative. The vomiting, from which our patient has suffered, has not been a marked symptom, and is on this account of little value in determining the seat of the growth. When the middle lobe of the cerebellum, the medulla, or the corpora quadrigemina is the seat of a growth, vomiting is often persistent and very distressing, and under such circumstances possesses considerable localizing value. Optic neuritis, one of the most pronounced symptoms in the case which we are engaged in studying, is of least value for purposes of localization. Optic neuritis, next to headache, is the most constant symptom of tumor of the brain, occurring in about five-sixths of the cases. It may occur from a growth in any situation in the membrane or brain substance, although it is more often absent in tumor involving the membranes on the convex surface than when the brain substance is invaded, and is probably most frequent in tumor of the base, corpora quadrigemina, pons, medulla, and cerebellum.

Thus far the symptoms to which I have directed your attention have not been of any special value in localizing the growth. Are there any present in this case that will serve us in this direction? Yes, two,—(1) hemiopic pupillary reaction to light, and (2) hemianopsia. A lesion posterior to the corpora quadrigemina in one occipital lobe, optic chiasm, or internal capsule, producing hemianopsia, does not abolish the reflex action of the pupil, even when a pencil of light is thrown into the eye from the side of the blind fields; but a lesion in the optic tracts, chiasm, or nerves, resulting in hemianopsia, destroys the reflex action of the pupil to light when the pencil of light is carefully thrown into the eye from the side of the blind fields. The loss of pupillary reaction to light in the blind fields enables us to locate the lesion at the base of the brain, in the vicinity of the optic-nerve fibres, anterior to the corpora quadrigemina.

This drawing (Fig. 1) illustrates the course of the nerve-fibres from the occipital lobes to the retinae, and you perceive that it requires a double lesion, one involving each of the external fibres of the optic chiasm, to produce binasal hemianopsia. The external fibres of the chiasm go to the temporal side of each eye, without decussating. In the patient before us the temporal half of each retina is blind (bilateral

temporal hemiopia), but the nasal fields of the eyes are the blind ones (bilateral nasal hemianopsia). This is an extremely rare form of hemi-

FIG. 1.



Course of the fibres in the optic nerves, tracts, and radiations.—After Starr.

anopsia. Professor Knapp, of New York, has reported such a case, due to thickening and enlargement of the internal carotid arteries. (Opera Minora, p. 328, Seguin.)

I regret that I did not have an opportunity to examine the fields of vision earlier in this case. When I first saw the patient he only had faint light-perception on the temporal side of the left eye, but it was sufficient to enable me to elicit pupillary reaction to light on this side, while no such response to light was obtainable on the nasal field. To-day the left eye is entirely blind, but the right presents light-perception in the temporal field. We see, then, that the character of the hemianopsia locates the lesion at the optic chiasm, beginning from the external surfaces, and this is further corroborated by the loss of pupillary reflex to light in the blind fields, while the headache, vomiting, and optic neuritis are readily accounted for from a lesion in this situation.

The jerky incoördination observed in this man's arms usually occurs from lesions in the cerebrum involving the motor tracts indirectly, but not sufficiently to produce distinct paralysis. It is of importance in some cases lest it should be mistaken for the jerky tremor of multiple sclerosis of the brain and cord. It is probably due, in the present case, to irritation of the crura.

We will next study the probable nature of the growth. It is sometimes easy, and at other times most difficult, to determine the nature of a growth in the brain. In the absence of growths in other portions of the body, the most probable nature of a cerebral tumor in a man of his age is syphilitic, tubercular, or gliomatous. In adults it is rare to have tubercular growths in the brain without the presence of tubercles in other portions of the body, especially in the lungs. In children this is not so, as it is not uncommon in them to have tubercular growths in the brain without the lungs showing any signs of tuberculosis. Gliomata are rare at the optic chiasm, and two gliomata situated so as to impinge upon the external surfaces of the chiasm would, in all probability, have given rise to paralytic symptoms affecting the cranial nerves before this time. The most common sites for gliomata are in the cerebral hemispheres, cerebellum, central ganglia, pons, medulla, crura, and corpora quadrigemina, in point of frequency in the order in which I have named them. What evidence have we that this man is suffering from syphilis, seeing that he denies any infection of this nature? Just here I wish to remind you that we must not lay too much stress upon the absence of syphilitic history. Some contract syphilis, and deny it, although the constitutional symptoms have been experienced; some suffer from the initial lesion, but the constitutional symptoms are so slight that they supposed themselves to have been cured; while others may have a chancre in the urethra or

in some other portion of the body, where the exact nature of the lesion has been entirely overlooked. Adults of this man's age (twenty-three years) who have never had any suspicious sore, and who have never exposed themselves, we may conclude are free from syphilis. While this patient does not deny exposure, he denies syphilis. There is a reason for his denial. He works for a corporation that does not give free care and treatment to its employees ill from venereal trouble. There are no marks of syphilis on the external surface of the body, although he has suffered from sore-throat and the hair of his head is rather thin. The results of treatment in his case favor the suspicion of syphilis. When he was first placed on potassium iodide and mercuric chloride his headache disappeared in a few days, and sight so improved within a week's time that he left the hospital to resume his occupation. On stopping the treatment he soon relapsed, and in a short time was worse than when he first entered the hospital. At the time of his second admission into the hospital, when I first saw him, the former treatment was resumed and pushed, and again his headache and vomiting ceased promptly; but irreparable damage had been done to the optic nerves, and the resulting atrophy has caused almost absolute blindness. You see then that the effects of treatment, while not positive, are presumptive, evidence of the syphilitic nature of the growth.

What is the prognosis in this case? The man will remain blind, or, if the vision improve, it will be so slight that he will find very little comfort from it. The optic nerves are practically destroyed. I suspect that we have in his case a tumor-like thickening of the pia in the region of the optic chiasm, which at first compressed the external nerve-fibres and gave rise to bilateral nasal hemianopsia, but later the contraction of the exudate has resulted in throttling all the nerve-fibres of the chiasm. Within a few months I have had the good fortune to witness two autopsies with similar morbid changes, both of which subjects I had seen some weeks before death, but neither before blindness was so complete as to render testing the fields impossible. With treatment from time to time, it is possible, and even probable, that this man's life may be prolonged for some years.

The treatment should consist of mercury mainly, as I have found that he bears this better than potassium iodide, and it seems to do him more good than the latter. I find the mercuric bichloride as efficient as any of the other preparations of mercury. He is taking one-twelfth of a grain thrice daily. At one time it was increased to one-eighth, but he did not tolerate this dose. In giving mercury,

where you wish to administer large doses over prolonged periods, keep the mouth and teeth clean. Have the patient use a stiff brush twice daily, and make the gums bleed. By this precaution you will be able to get the patient to take large quantities of mercury without suffering from salivation. As soon as the patient ceases to improve the mercury should be stopped for a number of weeks, and resumed again, but in smaller doses. It will be necessary for this man to take anti-syphilitic agents for three or four weeks, about three times a year, as long as he lives. In combating the ravages of syphilis with appropriate medicines pay attention to nutrition, and keep this in the best possible condition.

SUBSEQUENT HISTORY OF THE CASE, WITH AUTOPSY.

Soon after the lecture the patient was transferred from St. Luke's Hospital, Denver, to the hospital of the railroad company at Trinidad, Colorado, and to Dr. John R. Espey, the railroad surgeon at that point, I am indebted for the following notes.

The patient continued nearly rational most of the time up to his death, but there were periods in which he was more or less comatose. Dysphagia and weakness of the entire body developed, but no groups of muscles were entirely paralyzed. The prominent symptoms towards the last were almost continuous bleeding of the gums, congestion of the left eye, and marked retraction of the head, which was very pronounced the two weeks immediately preceding death. He emaciated greatly, and a sacral bed-sore formed about one week before death. He first lost control of the vesical and anal sphincters during the night, but after this had continued for about a month he soiled the bed, both night and day, for at least two months immediately preceding death. The doctor speaks of partial anæsthesia of the posterior portion of the trunk, but states that no systematic examination was made or regular notes of the case kept, so that it is probable that the lessened sensation was due to pressure. Death occurred February 24, 1895. The brain was removed and sent to me for dissection, but when it reached me it was torn and considerably lacerated, and the two hemispheres of the cerebrum had been separated in order to pack it into a small jar. A tumor was found in the left lobe of the cerebellum, which had extended forward and toward the median line, and evidently before death had exerted pressure upon the pons and medulla. The pia surrounding the optic chiasm was greatly thickened, and had pressed upon the optic nerves and chiasm. The tumor was encapsulated, and proved to be a glio-sarcoma. In parts it was dis-

tinely sarcomatous, with polymorphous cells. The endothelium and adventitia of blood-vessels showed proliferation. The latter was so marked in places as to compose the major part of the tissue. The result of the autopsy explained the clinical symptoms. Before I saw the patient there had been distinct symptoms of cerebellar tumor, with vomiting, headache, optic neuritis, and marked cerebellar ataxia, but his ataxic gait had entirely passed away under the administration of potassium iodide and mercury, leaving the general symptoms of tumor of the brain, optic neuritis, headache, and vomiting. At the time the patient came under my care the only localizing symptom was the peculiar hemiopia spoken of in the lecture, and this was caused by the thickened pia, which had compressed and partially destroyed the optic chiasm.

