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Report of a Case in Which the Patient Survived for More Than Thirty Years

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HISTORY OF THE CASE

By Dr. Keen

I shall give a summary of the case,[1] and at greater length than I would otherwise do, because it was my first modern brain case; because it shows our technic at that time; because it was one of the earliest operations on such tumors, following by only two years Godlee's in 1885, the very first ever done for a tumor of the brain; and because of the great length of time between the operation and the death of the patient.

History.—T. D, a man, aged 26, first consulted me in May, 1887, at the request of Dr. M. L. Davis of Lancaster, Pa., who furnished me with the following history: When 3 years old the patient fell out of a window, his head striking on some bricks several feet below. The skull on the left side was indented, and (as was disclosed by operation) there was a small fragment of bone detached from the inner table without fracture of the outer table. Evidently the child had struck on the pointed corner of a brick. A scar in the scalp, one-quarter inch long, marks the site of the injury.

He lay motionless for quite a long time as if dead. His recovery was slow, but apparently complete. At intervals, ever since he was 5 years of age, he had had a discharge from the right ear. In 1886 he became partially deaf in the left ear. He had long suffered from frontal headache. In February, 1885, when 24 years old and twenty-one years after

1. The full report of the case is given in the Transactions of the American Surgical Association, 1888, and the American Journal of the Medical Sciences, October, 1888.

the fall, violent epileptic attacks set in. They were followed by intense pain in the head. The attacks took place once or twice a week. By the end of April, 1885, the right arm, leg and face, in succession and in the order named, became paralyzed.

Dr. Davis first visited the patient at the hospital in Lancaster, June 8, 1885. As he approached the hospital he heard him screaming with pain, which was located on the left side of the head. The pain was increased by pressure. The right pupil was dilated and not responsive to light. The left was normal and responsive. Vision in the left eye was good, but in the right eye was imperfect. Aphasia was marked so that he could not converse. The pulse was 60 and irregular; the respirations were 16; there was obstinate constipation and coated tongue, but no fever. Syphilis was eventually excluded. The diagnosis was "pressure on the anterior lobe of the left hemisphere involving the third convolution and

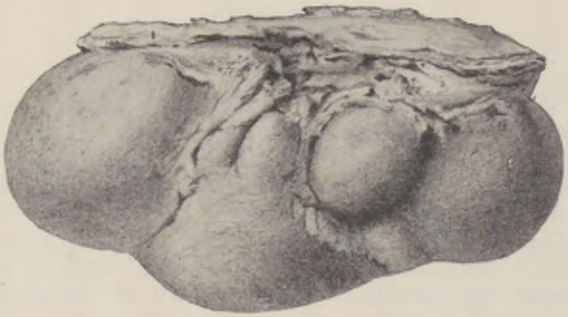

Fig. 1.—Lateral surface of brain tumor, natural size.

extending backward, due to exostosis, tumor or possibly only thickening of the dura." Later and before operation, the diagnosis was more definitely limited to a tumor. The treatment was potassium iodid with arsenic and laxatives.

The patient's eyesight rapidly failed, and by August, 1885, he had become totally blind, first on the right side and then on the left. In two months his left eye improved sufficiently so that he was able to walk on the street alone. The right eye remained blind for several months, when sight suddenly returned, but vanished in a few hours. This intermittence of vision continued in both eyes. The urine was normal. His mentality was much impaired.

When I first saw the patient, May 30, 1887, in St. Mary's Hospital, Philadelphia, his paralysis had improved so that he could use his arm and walk, though halting very slightly. He was slow of thought and speech, peevish, fretful, and seemed at times dazed. His gait was not spastic. There was moderate deafness, the right pupil was slightly the larger,

and the right eye deviated slightly outward and upward. The small scar was 3 ¾ inches above the midzygoma and 1 ⅝ inches in front of the interauricular line.

June 7, 1887. He had six epileptic attacks while in the hospital, the first since November, 1885. Other attacks occurred at intervals of a few days. Dr. Oliver saw some of them and made careful ophthalmoscopic records. He was seen in consultation also by Drs. S. Weir Mitchell, Morris J. Lewis and George C. Harlan. The conclusion reached was that though the evidence was probably in favor of tumor, it would be wiser to continue for a time a course of potassium iodid. If he was not better in the autumn and he still desired operation, I told him I would operate.

Fig. 2.—Inferior surface of brain tumor, natural size.

Operation.—No improvement having taken place, the patient returned to the hospital for operation in the late autumn. The operation was fixed for Dec. 15, 1887. The hospital had formerly been a dwelling house, and was still quite primitive. Before the operation the carpet was taken up, and the walls, ceiling and floor were thoroughly scrubbed with phenol (carbolic acid). New, clean, marine sponges had been kept in phenol, but were used in 1:1,000 mercuric chlorid solution. The instruments were boiled in an open vessel for two hours before the operation; at later dressings they were only soaked in phenol, 1:20, for half an hour, and then transferred to boiled water suitably cooled. The phenol spray was used all the morning of the operation, but not during the operation itself or at any of the redressings. The hands were disinfected with soap and water, alcohol and mercuric chlorid.

For the operation a 1 ½ inch trephine was used. This was considered an enormous size, for the largest I had ever used

before was only ½ inch in diameter. Removal of the first button exposed the tumor, but it was much larger than the opening. A second button was removed, and this opening at that time considered large, was still further enlarged by the rongeur until it measured 3 by 2 ½ inches. The upper margin was ¾ inch from the midline. The tumor dipped behind the squamous portion of the temporal bone for half an inch. The dura was adherent to the brain except at the margin of this large opening.

I have quoted this rather fully so as to give an idea of the startling size of this growth and to justify the trepidation which I felt in entering on this *terra incognita,* for this was my very first modern brain operation. My heart "sank down

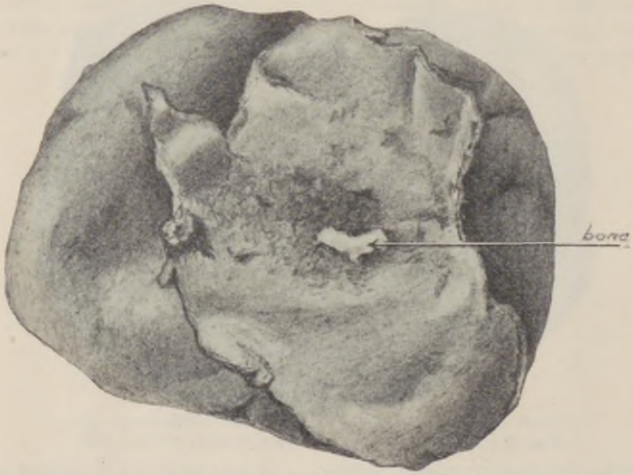

Fig. 3.—Superior surface of brain tumor. This shows the removed portion of the dura and the small fragment of bone which at operation was found detached from the inner table of the skull, natural size.

into my boots." But dangerous as the procedure might be I *had* to go ahead. I incised the dura one-quarter inch from the margin of the opening in the bone, and with my little finger, to my surprise and relief, enucleated the tumor with as little difficulty as one scoops an egg out of its shell. The hemorrhage was free but not alarming.

It is worthy of note that during the time occupied by controlling the hemorrhage the large cavity left by the removal of the tumor was half filled up by the resilient brain tissue. The bottom of this deep cavity consisted of softened and in parts shreddy brain tissue. Evidently the roof of the lateral ventricle was intact. The tumor was practically a foreign body starting at the dura as a result of the constant irritation from the little loose fragment of the inner table broken off

at the time of the accident and never consolidated with the skull. As the elastic skull recoiled from the blow, evidently the fragment did not resume its original position, but was in contact with the normal inner table and so could not unite with the bone. Every inspiration and especially every impulse from the throbbing heart produced a slight movement of this fragment. The tumor, a fibroma, slowly but constantly growing, pushed the brain tissue downward.

At the conclusion of the operation, two rubber drainage tubes were inserted and an abundant gauze dressing applied. The bone could not be replaced, as the dura was gone.

The tumor weighed 3 ounces, 49 grains, almost a quarter of a pound. It displaced 2½ ounces of water. Its size was 2⅞ by 2½ by 1¾ inches. Its circumference was 7¼ by 6 inches. Its posterior border reached backward nearly to the rolandic fissure. Pathologically it proved to be a pure fibroma (Figs. 1, 2 and 3).

Fig. 4.—Hernia cerebri soon after operation. (From An American Textbook of Surgery, Ed. 4, p. 586.)

Eight days after the operation the floor of the cavity which was also the roof of the ventricle evidently gave way, thus opening the ventricle. From the eighth day until the end of the fifth week the cerebrospinal fluid continued to escape very freely.

Postoperative Course.—No motor symptoms followed. The temperature was 100 ± for a week. Considerable disintegrated clot and shreddy cerebral tissue escaped, estimated at 4 ounces. All but two of the sutures had been removed by the eighth day. On the eighth day the patient's aphasia was greatly worse, the flap bulged more and more, the right arm became paretic, and the dressings—this is to be especially noted—were "saturated with a watery discharge but no pus." On the tenth day the temperature rose to 104.2 F., the face and arm were paralyzed, and by the next day the right leg was paralyzed. The patient had also a sharp diar-

rhea with fetid stools. Alarmed by these threatening indications of intracranial pressure and fearing also infection, I reopened the wound to nearly half of its original size—an error of judgment. But as I had no experience in such operations—and for that matter I might almost say that no one else had—I may be pardoned.

By the fourteenth day a moderate crescentic hernia cerebri had appeared (Fig. 4). The paralysis and aphasia gradually bettered, and the temperature became normal. The hernia cerebri at first increased, and by two small pin-holes at its middle a large amount of clear fluid escaped, evidently the cerebrospinal fluid. This fluid continued to escape until the end of the fifth week. The patient was sitting up out of bed at the end of four weeks. I had stained the nails of both hands with nitric acid and found that the nails on the right hand had grown "decidedly less than the left."

Fig. 5.—Depression in the head, sixteen years after operation. (From An American Textbook of Surgery, Ed. 4, p. 586.)

The hernia cerebri after a short time slowly subsided, till on the seventy-first day it was nearly on a level with the skull. The next dressing was four days later on the seventy-fifth day, when, to our surprise, instead of an elevation, as it had always been, it had changed to a deep hollow. (In 1908, twenty-one years after the operation, I noted that the depth of this depression was 5.5 cm. This had been its usual depth [Fig. 5]. The patient went home on the eighty-fourth day. The nails of the right hand were still half stained; on the left hand a barely perceptible line of discoloration was visible.

I noted then, what was for years, and up to the last time I saw the patient a few years ago, an always striking phenomenon. When he sat upright, any muscular effort—for example, using the dynamometer, and forcibly resisted expiratory effort—and a change of posture, as in leaning forward, caused the usually deep hollow noted above immediately to

bulge nearly an inch beyond the level of the skull. To protect his brain against injury I bent a piece of tin to fit the surface of his head, covered it with black silk, and sewed this to the inside of a skull-cap which he always wore.

Only three cerebral fibromas appear in Bernhardt and Hale White's table of 580 intracranial tumors. I gave an "entirely favorable prognosis" at the time of the operation, and thirty years have fully justified it.

The patient's later history is at first of a stationary condition and then of a slow deterioration. The epilepsy was markedly bettered. In 1902 he wrote me that he had not had an epileptic attack for two years, and from 1902 to 1908 he passed six years more without a convulsion, but his eyesight was gradually failing. He was, however, able to come to my office from Lancaster alone up to about two or three years ago. He died, Jan. 29, 1918, thirty years and forty-five days after the operation.

Dr. M. L. Davis at once advised me of his death, and the next morning Dr. Aller G. Ellis went up to Lancaster and procured the brain, which the patient had promised to me many years before.

My hearty thanks are due to Messrs. Harry S. Ziegler and S. Gordon Smyth, both of Base Hospital No. 38 (Jefferson Medical College Unit), for the admirable drawings of the tumor and the ventricle.

NECROPSY REPORT

By Dr. Ellis

The body was that of a well developed adult white man. Rigor mortis was present. There was a slightly oval, cup-shaped depression in the scalp on the left side of the head, just back of the hair line, that measured 5 by 6 cm. The center of this was 4 cm. deep in the recumbent position. The scalp was adherent to the margin of this depressed area, and its separation revealed a nearly circular opening in the skull. The longest diameter of this opening, taken from a point at the median line posteriorly to the most lateral point anteriorly, was 8 cm.; at right angles to this line the diameter was 7 cm. The depression in the scalp was smaller than this because of a crescentic ledge of very firm periosteum and dura 2 cm. wide that extended inward from the lateral margin of the bone and on a level with it.

Reflection of the scalp revealed it adherent at the margin of the opening in the bone, but fairly easily separated from the underlying tissue over the remainder of the opening. This underlying tissue was a gray, quite thick membrane that in many respects resembled the dura, but which was somewhat thinner than was the dura elsewhere. That this

was not dura was clear because the dura was removed with the tumor. It must, therefore, have been a newly formed fibrous membrane. It was not adherent to the brain.

Turning back this membrane exposed a crater-like cavity in the brain having as its deepest part the floor of the left

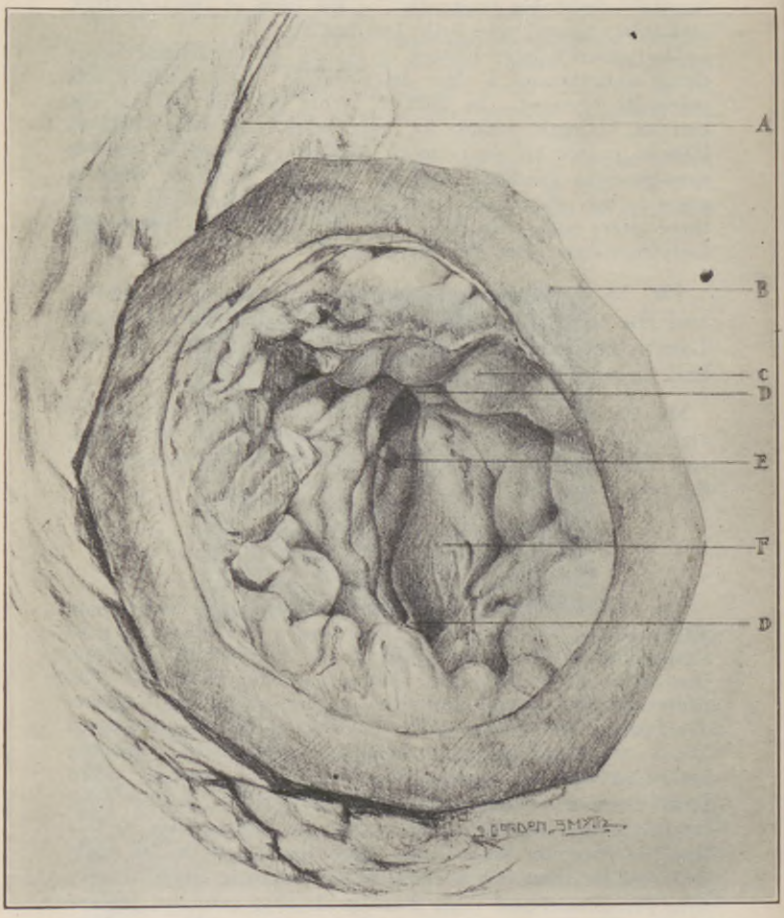

Fig. 6.—*A*, longitudinal fissure of brain; *B*, narrow zone of skull removed to preserve contour of operative opening; *C*, one of the cerebral convolutions forming wall of cavity; *D*, *D*, anterior and posterior boundaries of wide open left lateral ventricle; these points are 5 cm. apart; *E*, foramen of Monro; *F*, left corpus striatum.

lateral ventricle (Fig. 6). The floor of the ventricle was exposed for a length of 5 cm., the foramen of Monro being plainly visible 2 cm. from the posterior point of exposure. The

corpus striatum formed a part of the sloping floor and lateral boundary of the cavity, as the internal portion of the groove was deeper than the ganglion itself. The surface of the corpus had a number of tiny elevations. The thalamus was also visible at the posterior portion of the ventricle.

The wall of the cavity in general was of cerebral tissue that projected in rounded masses of varied sizes; these were firm and white, and looked like recently exposed brain tissue; in short, they presented the appearance of cerebral convolutions that evidently were displaced downward by the very slowly growing tumor. The falx cerebri formed a small part of the inner boundary where the cavity reached the midline superficially.

The base of the brain was unaffected, except that the optic commissure was decidedly smaller in size than is usual. The optic nerves also were small. Microscopically, sections of the left optic nerve showed decided atrophy, dense bands of fibrous tissue extending through it. In areas near the capsule, the fibrous tissue occupied much more space than did the nerve tissue itself.

Sections of the corpus striatum showed a covering of ependyma in an essentially normal condition. The tiny elevations on the surface were composed of neuroglia that underlay the ependyma.

Sections of one of the protuberances forming the boundary of the cavity were of cerebral tissue in which there were almost no pyramidal cells immediately under the surface and but few farther inward. Even those that were present were atrophied. There were a few more mononuclear cells along the course of the vessels than are commonly found. The surface next the cavity was of compressed neuroglia with no evidence of the formation of ependyma.

The tissue from which these sections were taken was somewhat distorted because of its position in the wall of the cavity; conclusions, therefore, must be guarded. It would seem, however, that the layer of small pyramidal cells was thinned and the large cells atrophied.

The pathologic diagnosis was extensive wound of skull and brain exposing almost the whole of the left lateral ventricle; atrophy of the optic commissure and nerves with extensive fibrosis of the latter, and atrophy of pyramidal cells of the cortex.

The point of special pathologic interest in this case is the extensive exposure of the interior of the left lateral ventricle for a period of over thirty years.

The ventricular area of the central nervous system was greatly increased. So far as the clinical history indicates, there was no symptomatology of changed intracranial pressure, either increase or decrease. The fact that the covering of the wound was depressed

when the patient was in the erect posture is anatomic evidence that the pressure was increased to no appreciable extent, if at all. When the patient stooped and the scalp protruded, the spinal fluid must have accumulated principally in the left lateral ventricular area. This fluid probably even came from around the spinal cord, but apparently without causing any clinical symptoms.

At necropsy there appeared to be no increased amount of fluid, and the dépression of the scalp during life was proof that the wound cavity was not filled by that fluid. The case is unique from the standpoint of duration, and to the best of my knowledge of extent of involvement of the lateral ventricle, and we therefore have none with which it may be compared.

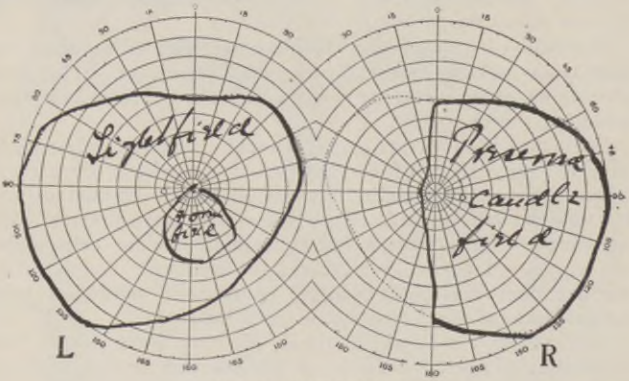

Fig. 7.—Visual fields.

The question arose as to whether the inner surface of the wound became covered by ependyma extending from the ventricle. This proved microscopically not to be the case. According to Weed, however, the ependyma plays a negligible rôle in the production of cerebrospinal fluid, and this would not be important from that standpoint. If, as is generally believed, the fluid is very largely the product of the choroid plexus, the condition obtaining here would be of more importance from the standpoint of pressure than from production of fluid. The case, therefore, was one of long-standing extensive increase of ventricular area of the brain without demonstrable clinical effect.

COMMENT

By Dr. Keen

In the length of life after the operation and the wide open ventricle, this case seems probably to be unique. The only roof to the ventricle was the thin fibrous membrane formed after operation. This was not adherent to the surface of the brain. Therefore, the cerebrospinal fluid had free access to the space between the dura and the pia-arachnoid, as well as to the subarachnoid space.

The cause of the formation of the fibroma, its very gradual displacement downward of the cerebral convolutions with disappearance of the pyramidal cells near the surface and their atrophy deeper down, and the great improvement in the epilepsy have been noted in the history. In spite of his early hemiplegia, the patient was able to walk about for years without a cane and with almost no evidence of any paralysis of leg, arm or face, unless possibly the slight deviation of the right eyeball was a result of the tumor. For one or two years before his death he was confined to his bed because of his increasing general weakness.

The hernia cerebri, and the cuplike depression at the site of the operation when the patient was erect, are shown in Figures 4 and 5. The changed condition from a cup over 2 inches deep to a rounded swelling nearly 1 inch in elevation brought about by muscular compression of the trunk and by the change from the erect to the horizontal position was evidently due partly to the almost instantaneous rush of the cerebrospinal fluid from the other ventricles and from around the spinal cord, and partly to the increase in the blood content of the cerebral veins. This ebb and flow of the cerebrospinal fluid was a most striking phenomenon, the like of which I have never seen in a large experience in cerebral surgery. Even the halting of the breath accompanying the use of the dynanometer, by coughing, etc., would either almost fill up the deep cup by the elevation of its floor, or if continued and with considerable force, would convert the cup into a hill. As in the erect posture the deep cavity was constantly present, it is evident that the total amount of cerebrospinal fluid was less than the normal, yet no cerebral symptoms ever resulted during more than thirty years of postoperative life.

Fortunately, fifteen years ago (Nov. 30, 1903), midway between the operation and the patient's death, Dr. de Schweinitz examined his eyes and gave me the subjoined brief report:

Ophthalmic Diagnosis: Postpapillitic atrophy of optic nerves.

Vision: Right eye, hand movements, eccentric; left eye, 1/30.

Pupils: Right, 8 mm.; left, 7 mm.; imperfect light reaction.

Muscle Balance: Vertical nystagmus.

Eyegrounds: Right eye, nerve head atrophic; both sets of retinal vessels smaller than normal, and perivascular lymph sheaths distended; numerous yellowish white (colloid?) spots in macular regions. Left eye, similar; less marked shrinking of retinal vessels.

Remarks: The appearances are those of postneuritic atrophy of the optic nerves, in the corresponding loss of direct peripheral vision.

The surgery of the lateral ventricles from this time on interested me very deeply.

Nov. 7, 1888, I read a paper[2] in which I proposed a formal operation to tap and drain the lateral ventricles, an operation which is now almost commonplace, but which then had not been reduced to the exact surgical technic which I formulated.

Jan. 11, 1889, on a boy, aged 4, with a tumor of the cerebellum, I first carried this operation into effect on one side.[3] For certain reasons there stated,[4] February 8, I tapped the opposite ventricle. On the thirty-second and thirty-fourth days after the first operation, I irrigated the two lateral ventricles from side to side, using 8 ounces of a warm 4 per cent. solution of boric acid. As soon as the warm solution began to flow freely through the brain, the child, who had been a little restless, quieted down, saying "it felt good." The choked disk had measured 2.3 mm. in each eye before the first operation; it subsided six days later to 1.09 mm. on both sides. He died on the fifty-second day.

In the same paper[3] I reported two other cases of tapping the ventricles.

At the International Medical Congress in Berlin, in 1890, I presented a complete paper on the surgery of the lateral ventricles, which was a study of ventricular surgery from the time of Paré to that date. Unfortu-

2. Keen, W. W.: Med. News, Philadelphia, Dec. 1, 1888.
3. Keen, W. W.: Proc. Philadelphia County Med. Soc., 1889, p. 50.
4. Keen, W. W.: Proc. Philadelphia County Med. Soc., 1889, p. 85.

nately, only an abstract has been published,[3] since the original paper was lost by the secretary of the Surgical Section.

I corrected seven instances of deliberate puncture of the ventricles, with two recoveries.

In fourteen cases of rupture of the ventricles from fracture and secondary opening of them (one of which was the present case), nine patients recovered.

I concluded, therefore, that such involvement of the ventricles was in no wise so dangerous as had been supposed, except when an abscess burst into a ventricle or a large hemorrhage into it took place. Instances of both of these conditions were cited. Every one was fatal.

Among all these and many other cases I did not find any one at all resembling the present one, nor have I ever seen any similar case reported since my paper in 1890.

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