A CASE OF TRAUMATIC EPILEPSY.

From the Mental and Surgical Services of the Albany Hospital.

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This case is reported because of the typical symptoms pointing to a localized cortical lesion, the availability of the latter for surgical interference, its removal by operation, and the recrudescence of the disease arising from the connective tissue proliferation incident upon the process of healing.

CASE 3599, PAVILION F RECORDS, ALBANY HOSPITAL.

The patient was a muscular, well developed boy, who was brought to the hospital by his mother on January 7, 1916, because of a mental condition which rendered him unmanageable at home. He was born October 22, 1905, with a history of tuberculosis in his father's family. His father had died of tubercular meningitis at fifty-two. On the mother's side the antecedents were good. There had been no other children. He was a frail baby, but after a few weeks improved, and with the exception of an attack of acute lymph-adenitis, which lasted about a week, had always been healthy and rugged.

In October, 1910, he fell about six feet and was found dazed. No after-effect was noted. In January, 1913, he fell again about ten feet. Both falls were purely accidental. After the second fall he lay a few seconds, and there appeared no immediate effects. He did not complain of pain in the head. On the day following the second fall, while standing near the dining table, he suddenly appeared dazed and said to an aunt, "Why, where are you? It is all black and I cannot see you." Six months later while talking he suddenly stopped. His mother approached him, noticed that he was "lost," and after a few seconds he said he "had been somewhere." In September, 1913, while standing near the telephone switchboard at which his mother was employed, he again suddenly stopped talking. After that the attacks occurred sometimes as often as three times a day. In school he would stop reading, and then begin again after a pause, but after a short time he was able to pick up the reading where he left it.
He was given bromide and had some relief during the following year. During this period he had occasional attacks, characterized by turning his head and eyes to the left, but there was no spasm of any kind. On one occasion he left his desk and went into a class of which he was not a member.

For the three months preceding his admission to the hospital he had none of these attacks. In November, 1915, he became irritable and profane, and had mental seizures in which he seemed to wish to kill himself, and on several occasions asked for a gun to kill some children who had annoyed him; he carried a toy pistol for self-defense. These attacks began suddenly, and sometimes lasted for half a day. His appetite failed, he was unable to maintain himself in his studies, and it was particularly noticed that he made peculiar errors in spelling and reading. He did not understand what he read, but seemed to comprehend when he was told. He spelled his name “J-h-o-n-s-o-n,” invariably displacing the “h,” although he had previously known how to spell it. He also lost his ability to write. His sleep was irregular and often restless, but he manifested no excitement and was not known to have attacks during the night.

The above history as given by the mother does not vary essentially from that given by her to Dr. Hun, whom she consulted on March 24, 1915. At that time it was stated that the attacks of petit mal began in October, 1913, and during the ensuing eighteen months, had been repeated from twenty-five to thirty-five times. In some attacks his head was drawn so strongly to the left that he called to his mother for assistance, and did not become unconscious. In the attacks of petit mal he lost consciousness for a few seconds, and there had been only one major attack so far as known. There had been no local spasm beyond the deviation from the head to the left. The physical examination at that time was negative. Dr. Hun advised operation.

For three days after admission to the hospital the boy was drowsy and inactive, and there was difficulty in arousing him. On the 10th of January some peculiar manifestations were noticed, and the nurses stated that “he seemed to follow something along the wall.” On the following day he had two slight epileptiform convulsions which lasted four or five minutes. On the 12th there were three attacks of ambiguous character, and it was noticed that his head and eyes were turned to the left. The
attacks had apparently been controlled by bromide, for at this
time they became very frequent, and on the 14th about twelve
seizures were noted, varying in character from petit mal to major
attacks, and often in the spasmodic condition with rotation of
the head and eyes to the left. On the 15th I was fortunate in
seeing a more typical and illuminating seizure than any that had
been reported. The boy was brought to my office by his mother,
and while sitting quietly awaiting consultation the attack began.
His head and eyes turned slowly to the left and became fixed in
extreme left lateral rotation with the lids closed. The left upper
extremity was elevated slightly in position of adduction and semi­
flexion of the forearm, without marked rigidity. Toward the end
of the attack there was a deep respiration, the head relaxed in
moderate clonic spasm, and the boy suddenly awoke. The clonic
stage did not affect the extremity. There was no involvment of
the lower extremity or of the opposite limbs. There was a sug­
gestion of coma during the clonic stage, but there was no coma
after the spasm subsided, and the patient became normal and
active at once. The boy returned to the hospital, and the mother
reported that during their absence of four hours he had had nine
seizures. The Jacksonian and other attacks became more and
more frequent until the operation.

The frequency and character of the attacks are indicated in
the following extracts from the nurses' notes:

January 7. 6 p. m. Got up to walk, but instead of walking
straight ahead, he turned around and around.

January 8. 3 to 6 a. m. Sits up in bed and looks around but
still in sound sleep.

12 p. m. Asleep with eyes open and face twitches.

January 10. 1 p. m. Seemed to follow something along the
wall; did not feel when hand was pinched.

January 11. 10 a. m. Attack like an epileptic fit: twitched and
jerked all over; voided in his clothes; yawned when coming out
of it; lasted four or five minutes.

11 a. m. Walks peculiarly on toes; drags feet.

3 p. m. Unconscious for a few seconds.

6 p. m. Walked around and around to his left with head turned
over left shoulder.

9.30 p. m. Head turned to left; body stiff; unconscious;
twitching.

10 p. m. Stood in middle of floor with head turned over left
shoulder, and turned around and around for two minutes.
January 12. 6 A. M. Shook hard, attempted to bite tongue; cried, said he could not get his breath and was dizzy.
12 M. Fell to floor.
9 P. M. Dropped to floor; stiff; with head and eyes turned to side; three minutes.
January 13. Three attacks with rotation of head and eyes to left.
January 14. Eleven attacks of brief duration with rotation.
January 15. 4 A. M. A severe attack, lasting ten minutes; cried, said he was scared and dizzy; could not sit up; staggered, almost to falling. Fifteen other attacks.
January 16. 4 A. M. Attack—made strange noise; shook hard; cried at top of voice; could not walk alone; followed by very ugly mood.
8 A. M. Head turned to right(?); eyes closed tight; shook; muscles rigid; mouth open, with grunting noise; both arms bent at elbow and fists clenched; face flushed, white about nose; hands blue and cold; followed by difficulty in breathing.
Twenty-one other attacks during the day, several severe and many light.
January 18. 4 A. M. A major attack.
11 A. M. To operating room.

Dr. Elting kindly gives the following description of the operation:

At the operation on January 18th, 1916, an extensive osteoplastic resection of the right side of the skull was done. The dura appeared to be normal on its exterior surface as did the inner surface of the bone flap. On palpation, however, the dura seemed abnormally tense as though there was increased intracranial pressure. The dura was freely incised when it was found that very extensive old adhesions existed between the dura and pia. In some places these adhesions were very dense and tough so that some of the larger cortical veins were torn in the process of loosening the adhesions, requiring several sutures. The area of adhesions was roughly about eight centimetres in its diameters and covered practically the entire motor area of the cortex. The adhesions were carefully loosened over the entire adherent area and it was found that beyond this area there were no adhesions and no evidence of any abnormality. The cortex was carefully palpated but no evidence of any other cortical or subcortical disturbance could be found other than the adhesions. No attempt was made to suture the dura, which would have been quite impossible owing to the increase of intracranial pressure. The
osteoplastic flap was replaced and the wound was carefully closed without drainage. There was a moderate amount of shock from which the patient soon rallied and he had a most uneventful convalescence. Almost twelve hours after operation, the nurse reported that he had a slight convulsive seizure of the left arm, but no other convulsive seizure of any description occurred thereafter during the sojourn in the hospital. For three or four days after the operation there was some moderate paresis of the left arm and left leg, but this speedily disappeared. The patient's entire disposition showed a marked transformation. During the early days after operation, he was rather self-willed, petulant and at times difficult to manage, but these characteristics gradually disappeared and when he left the hospital four weeks after the operation, he appeared normal in every way. There was no evidence of any muscular weakness, the mental processes seemed rather abnormally acute, his disposition was excellent and he appeared like an orderly, well-behaved, unusually bright and intelligent boy.

The patient left the hospital on February 5th, apparently well. Some errors of statement occurred, suggesting the mental state preceding the operation, but these responded to correction, and there was no real agraphia or alexia. Nor was there any motor defect. But any jubilation over his restoration was short-lived. Exactly two months after his discharge the mother described re-crudesence of the attacks after the manner of the original symptoms. The first demonstration is best described in her own words, under date of April 11, 1916:

"He has been all right until Sunday morning; he was getting ready to get up and was laughing and talking to me; he stopped and had a queer look on his face and commenced to say queer things. It did not last but a minute and he realized he had lost himself. Yesterday at dinner he dropped his bread and head and turned to the left. He knows he has been lost and says, 'I cannot be as I was.' He eats very heartily."

April 17, 1916: "He is getting nervous and fretful again."

May 4, 1916: "Has his lost spells, talks strange and mutters. Says he will drown himself if he is growing bad again."

May 23, 1916: "Sometimes he will have a spell just after breakfast after eating a good meal, and then he declares he has not had a mouthful and will eat as much more. One day he was playing the phonograph; I heard a noise and found him on the floor. He is growing very fat."

October 24, 1916: "Some days he does not know me. He weighs 121 pounds. He is very ugly at times. He again talks of killing himself."
The symptoms of an irritative lesion of the cortex are well known to be more ambiguous than those which result in paralysis, and many attempts at the determination of a focal lesion have failed when too much confidence has been placed in spasmodic conditions which seem to point to a definite cortical center. In this case there were decided and repeated attacks of conjugate deviation of the eyes and head to the left, and these were thought to have some localizing value. On the other hand, the loss of intelligence as represented by agraphia and alexia, suggested a destructive condition in an entirely different region of the brain, the spasmodic condition suggesting an irritation of the right hemisphere and the defect pointing to the left hemisphere.

Conjugate deviation of the head and eyes due to irritation has been attributed to irritative lesions of many parts of the cortex, and at different times has been assigned to lesions of the frontal, the temporal and occipital lobes. For the case under consideration it is not necessary to consider the center in the pons to which this symptom may be attributed, as none of the symptoms indicated lesion of the pons. The first definite statement as to a center for conjugate deviation of the head and eyes was made by Ferrier, whose experiments on monkeys revealed that stimulation of the posterior half of the superior and middle frontal convolutions resulted in deviation of the head and eyes toward the opposite side, with dilatation of the pupils and wide separation of the lids. This symptom has been emphasized in its relation with epilepsy, and Vorkastner gives it a prominent place in the beginning of the tonic stage of idiopathic attacks, and undoubtedly in many cases of epilepsy this definite phase is associated intimately with the spasms of the limbs. Further corroboration of the existence of such a center may be derived from symptoms occurring in paralytic conditions, for any destructive lesion affecting such a center destroys the cooperation of symmetrical balancing muscles, and in such a case the uninvolved antagonistic muscles act to rotate the head and eyes toward the affected side of the brain.

The whole matter is summed up in Hun's Atlas in which he states that conjugate deviation in which the eyes are turned to the side of the lesion may follow a paralytic lesion in almost any part of the brain, and especially one occurring in the posterior
part of the frontal lobe; whereas the eyes are turned away from the side of the lesion when there is an irritative lesion of the cortex.

Whatever doubt may have been felt as to the suggestiveness of this symptom was removed by the one attack in which the conjugate deviation was associated with the spasm of the left upper extremity. This seemed to indicate clearly enough an irritation spreading from the middle frontal convolution to the arm area in the precentral convolution of the right side. This was regarded as sufficiently definite to justify an operation at this point, and the operation was consequently performed.

Focal epilepsies are always instructive, particularly when determined by a definite cortical lesion. The mimicry of a circumscribed affection by the idiopathic disease, or of the latter by the former, may lead to disappointment from attempt at relief by surgical means. Even a definite traumatism may arouse neuropathic or epileptoid characteristics plainly predisposing to the disease. For the common formula, "the healthy child, the blow upon the head, the epilepsy," a skeptical editorial writer in the Medical Record (May 12, 1917) would substitute, "the epileptic makeup, the blow on the head, coincidental or precipitating, and the epilepsy." From his viewpoint, "if the blow on the head had not been forthcoming the development of the epilepsy would have been delayed until a later stress." True traumatic epilepsy should be independent of all preliminary taint, for "there are a very few cases of adults receiving depressed fractures of the skull with a spicule of bone pressing on some motor area and developing convulsions limited to or at least starting in the groups of muscles controlled by this area."

Surgery, however, is a highly aggressive art, and structures uncovered by the scalpel may disclose unsuspected facts and modify opinions elaborated upon no more substantial basis than theory. Kocher has even advocated decompression operations for the relief of idiopathic epilepsy, and asserts that an opening "large enough to prevent the increase in tension which he believes precedes the attacks" has a favorable effect. The possible benefit of this procedure is attributed by Frazier (Journal of the American Medical Association, June 5, 1909) to better discrimination between true and symptomatic epilepsy. He has found, in cases without a suggestion of a focal lesion, a large percentage
in which the brain when uncovered presented some gross pathologic lesion (adhesions, oedema, the remnants of an old pachymeningitis, etc.) “so frequently that it would appear as though the so-called idiopathic type was a little less prevalent than we were led to believe before we had so many opportunities as now to study the living pathology of the brain and its membranes.”

The case which has been made the subject of this article strongly emphasizes Frazier’s contention. The history of repeated traumatisms was suggestive, but not conclusive to the doubter who would attribute the falls to initial seizures. The attacks, considered singly, may be assumed to have represented wide-spread disease of idiopathic nature. Persistent alexia, agraphia and amnesia were surely misleading and implied deterioration. Petit mal, general severe convulsions, and psychic phenomena were variations too broad to be accepted as of Jacksonian type. But prolonged observation gradually brought to light some distinctive characteristics. The initial phenomenon was conjugate deviation of the head and eyes to the left; this was often followed by flexion of the left upper extremity, and in more severe seizures was amplified into genuine *epilepsia pro-cursiva rotatoria*, the propulsion being likewise to the left. A broad interpretation of Frazier’s work would have justified surgical interference without the history of traumatism.

The immediate effect of the operation is instructive. The complete relief of the seizures and the restoration of the boy’s intelligence are sufficient proof of a difference between acquired and idiopathic epilepsy even at this susceptible period. Convulsive seizures are rare in adult life, no matter how vicious the lesion. Because there are hundreds of cases of delirium tremens with only an occasional convulsion, there can be no reasonable argument that these occasional attacks are not due to alcoholism. On the other hand the casual incidence of convulsions in childhood from relatively slight causes, does not imply the presence of epilepsy.

Kocher’s intrepidity in subjecting idiopathic cases to the knife adds to the many futile attempts to relieve epilepsy which constitute one of the most dramatic chapters of the long history of therapeutics. But if the clinical evidence in the case points to a lesion which can be reached, there is hope in surgery. There may be faith that technique will be found to anticipate the cicatricial disasters of the healing of the wound.