POLIENCEPHALITIS SUPERIOR OF WERNICKE, WITH REPORT OF A CASE

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Encephalitis has been described either under this title or under the title of phrenitis since early in the seventeenth century, but not until Virchow began his pathological work in the first half of the nineteenth century was any extensive progress made in the knowledge of the condition. Since then many types of encephalitis have been described based on the pathology.

In 1881 Wernicke in the "*Lehrbuch der Gehirnkrankheiten*," described the type which he called acute hemorrhagic poli-encephalitis superior, the pathology of which was the formation of numerous punctate hemorrhages with acute inflammation about the Sylvian aqueduct and the third and fourth ventricles. He reported at this time three cases, though these were not the first reported of this type, since in 1875 Gayet had reported a case, but apparently had not recognized its complete identity. He called his case "*affection encéphalique*." In 1892 Boedeker found eleven cases reported, three of a non-alcoholic origin and eight alcoholic. Of Wernicke's three cases, two were alcoholic and the other was in a young girl who had attempted suicide with sulphuric acid some two months before the onset of the disease.

Acute hemorrhagic encephalitis, taken in its widest sense is in the great majority of cases caused by infection. Many cases have been reported during epidemics of influenza. It has also been seen following measles, scarlet fever, pneumonia, erysipelas, whooping cough, and in a case reported by Putnam it developed after an attack of mumps.

Oppenheim believes that post-diphtheritic paralysis is frequently caused by hemorrhagic encephalitis. It has been observed after ulcerative endocarditis and in middle ear suppuration, here often associated with serous meningitis. The acute poli-encephalitis of Wernicke may be divided into two general classes according to the etiological factor involved; one class being due to long-standing ingestion of alcohol, and the other to various factors,

frequently influenza. The onset of the acute symptoms is usually rapid after a short premonitory spell of headache, vertigo and some irritability, though even these premonitory signs may be wanting. The patient then becomes in turn dazed, unconscious and stuporous, though the stupor is seldom deep enough to abolish the pupillary or tendon reflexes. The temperature is variable, elevated moderately, or at times sub-normal; the coma may deepen and death ensue in from twenty-four hours to several days without return to consciousness, or the disease may run a more protracted course of many weeks or even months.

In Gayet's case, five months passed from the time of the onset until its fatal termination. When the course of the disease is protracted the fever is usually irregular and symptoms of paralysis appear in the form of monoplegia or hemiplegia. There is usually a cerebellar ataxia with tremor, and at times difficulty of speech. The ocular signs consist of palsies of extra-ocular muscles, ptosis, nystagmus and alterations in the optic disc, varying from a papilledema to an atrophy of the papillo-macular bundle in the chronic alcoholic cases, and as a result, of course, vision is more or less affected. It is not unusual to have the poli-encephalitis of Wernicke descend the cord and become a poli-encephalomyelitis. Such cases have been reported by many, three cases in the *Boston Medical and Surgical Journal* for 1903 by Taylor being especially interesting. Wilbrand and Saenger have in 1900 analyzed twenty cases, alcoholic in origin, of Wernicke's encephalitis; ten had ptosis and in three cases no mention was made of the lid condition. In all the cases except two in which no mention was made of the condition of the extra-ocular muscles, those muscles were variously involved, from the palsy of a single muscle (external rectus) to a practically complete ophthalmoplegia, and the pupils varied from normal reaction to immobility. The fundus changes varied from papillitis to a pallor of parts of the disc. The post-mortem changes were those as described above.

Schroeder reports one typical case which at necropsy showed minute hemorrhages and attempts at phagocytic repair, similar to those found in other inflammatory conditions. He believes the condition was caused by vascular change following chronic alcoholism, possibly similar to the hemorrhages of anaphylaxis. In the alcoholic type the early symptoms may be mistaken for delirium tremens, usually occurring in middle-aged chronic alco-

holics, and it is in this type that the typical pallor of the papillomacular bundle is usually found. Of cases non-alcoholic in origin there are but few in the literature. Gayet's case followed three days after a boiler explosion, in which the patient, a man twenty years old, did not suffer bodily injury. There was present double third-nerve palsy with ptosis, weakness, hemiplegia, and death five months after the accident. Wernicke's case, as has already been mentioned, followed two months after the attempt of suicide by sulphuric acid, with death twenty days after the onset of symptoms. Other cases, non-alcoholic in origin, have been reported by Salomonsohn, by Luce where the condition was a complication of miliary tuberculosis, and by Murawieff, who reports a case three months after influenza complicated by a sarcoma of the precentral gyrus. Zingerle reports a case with chronic digestive disturbances and an abdominal tumor of long standing. In *Brain* for 1897 Wiener reports a case of acute hemorrhagic encephalitis which seems to be more of the type which Strümpell has described, though there are certain conditions which suggest inflammation of the periventricular region. Since Wernicke and Strümpell recognize a difference only in the localization of the lesion, it seems probable there is very often a mingling of the two conditions.

In the *Journal of Nervous and Mental Disease* for 1894, Wolfe reports a typical case of Wernicke's encephalitis, the paper having been read before this Society, January 22, 1894. It was in a male thirty-nine years of age, who after many attacks of vertigo, numbness in the limbs and finally diplopia, sought medical advice. The temperature was found to be one hundred, eyes immobile and fixed in mid-position, loss of power and tone of the facial muscles and alterations of speech, partial ptosis, slight exophthalmos and loss of smell and taste. No changes in the fundus, vision being $\frac{6}{9}$, due to the moderate hypermetropia. He complained of a "pins and needles" sensation in both arms and legs. In four weeks after first being seen, the symptoms had very nearly cleared up. No symptoms of influenza infection had preceded the attack, though it occurred during an epidemic of the disease, and Wolfe believes this the possible etiological factor. The prognosis as first given by Wernicke was always grave, as he believed it usually fatal in from eight to fourteen days. That this is not always justified several cases on record show: notably this one of Wolfe's and the one which I report in this paper.

H. Y. P., age seventeen, came to me referred by his physician on April 12, 1906, giving the following history:

He had been in good health until late in January of that year, when he had a severe attack of influenza which lasted about three weeks. After this he felt fairly well until the middle of March, when he began to have difficulty in moving his limbs, as they were stiff and motion painful. This trouble seemed to develop gradually. Three weeks before his visit the soles of his feet became very sore whenever he stood on them. He said they felt like raw meat. This pain was only when standing or walking, and disappeared on lying down. He had some little headache. On Saturday, April 7, and the following day, his vision seemed a little dim to him. He went to his work on Monday, but his vision became so bad he had to return home, since which time it seemed to have remained unaltered.

On examination the central vision of the right eye was $\frac{3}{35}$ and of the left eye $\frac{1}{35}$. The pupils were semidilated, each being six millimeters in diameter, and responded very sluggishly and slightly to light, but promptly to accommodation and convergence.

FIG. 1. Visual Fields of H. Y. P., April 12, 1906.

His visual fields as shown by the charts were very markedly cut, the left field being obliterated, only a small triangle of the right field remaining in the left eye, and a much larger patch in the corresponding portion in that of the right eye. There was some ptosis with full and equal extraocular rotations, except upward motion which was decidedly limited. There was some pain on pressing the eyeballs back in the orbits. Ophthalmoscopically, both media were clear and what fundus alterations were present were confined to the right eye, the veins being rather full and the disc very hyperemic with veiled margins but not swollen; the left disc was normal.

His patellar reflexes were decidedly diminished, his station

swaying; there was no clonus. A diagnosis of acute retrobulbar neuritis was made and he was advised to enter the University Hospital, which advice he accepted and was admitted the same afternoon. He was placed in bed on a light diet, and given mercurial inunction, sodium salicylate, and sweats with free purgation. Dr. G. E. de Schweinitz saw him with me late the same afternoon, and agreed as to the probability of the diagnosis. His temperature on admission was 97.4, pulse 80, and respirations 20. The treatment as outlined was continued with the addition of a fly blister on the temples until the third day after admission when all medication by the mouth was stopped. His vision gradually fell until on the fifteenth, three days after admission O. D. was blind and O. S. had but scant light perception. Both discs were then hyperemic but not swollen, his weakness increasing and some headache and vomiting coming on. Dr. Wm. G. Spiller was asked to see him. The following is a transcript of the notes dictated by Dr. Spiller:

"He is weak in all limbs, not excessively, and is not paralyzed in any part. The sensations of touch and pain are normal in the face. The facial nerve supply is normal on each side. The tongue is normal. Muscles of mastication are normal in each side. He hears the voice clearly in each ear when the other ear is closed. He has almost absolute paralysis of upward associated movements of the eyeballs. The left eyeball possibly may be directed a little upward either voluntarily or in following the fingers, but the movement is questionable. Downward movement or movement to either side is well performed. Convergence is imperfect. The grasp of each hand is somewhat feeble. Sensations of touch and pain are normal in all the limbs and face. The muscles are not tender to pressure anywhere. The biceps tendon reflex is present but is weak on the right side and uncertain on the left. The triceps tendon reflex is not obtained on either side. He can move his lower limbs freely at all parts and with very little weakness. Patellar tendon reflex and Achilles tendon reflex are lost on each side, even with reinforcement. Babinski reflex is not present on either side. The toes, including the big toe, are flexed. His gait and station are good even with his eyes closed. At present there is no rigidity of the muscles anywhere."

Dr. Spiller, relying on the ocular findings, and his own examination made the diagnosis of acute hemorrhagic poliencephalitis superior. On the following day the vision had slightly improved and there was light perception in each eye. The vision continued to improve and in the next twenty-four hours the man could count fingers at a meter in each eye. On Wednesday, the eighteenth, the following day, he complained of a severe headache in the frontal region and there was nausea and some vomiting. This lasted for three days, during which time very little nourishment

was taken. His ocular condition had continued to improve steadily and by the nineteenth the discs were much clearer, upward rotation was nearly normal and convergence was stronger. Ten days later his vision was $\frac{4}{35}$ in each eye. He left the hospital May 3 with O. D. $\frac{6}{45}$ and O. S. V. $\frac{6}{30}$, upward rotation and convergence normal, discs rather pale. He was placed on strychn-

FIG. 2. Visual Fields of H. Y. P., May 3, 1906. Form, blue, red and green not seen. Absolute control scotoma for red in O. D.

nia sulphate and Bland's pills. When he left the hospital his form fields were full with concentric contraction for color, with a central scotoma for red in the right eye and with no recognition of green. In a week the scotoma had disappeared and the form fields were full; blue and red being still concentrically cut and green recognized at fixation only. By early June the fields were entirely normal as well as the central vision. The pallor of the discs gradually increased and by August they presented the appearance of a complete atrophy, but central and peripheral vision was normal, a condition which still continues.