

Society Reports.

AMERICAN NEUROLOGICAL ASSOCIATION.

Sixteenth Annual Meeting, held at Philadelphia, June 4th, 5th, and 6th, 1890.

(CONCLUDED.)

Dr. BURT G. WILDER, of Ithaca, N. Y., exhibited the left hemisphere of Chauncey Wright, and made the following remarks:

Mr. Wright was a well-known philosophical writer and general critic, who died in Boston in 1875, at the age of forty-five. His brain was removed by Dr. Thomas Dwight, now professor of anatomy in the Harvard Medical School, who published a brief description of it, with a diagrammatic figure of the left side, in a paper entitled "Remarks upon the Brain of a Distinguished Man" ("American Academy Proceedings," 1877, xiii., 210-215). It has since been generously loaned to the writer by Prof. Dwight for fuller study and publication.

A large number of photographs of the two hemispheres have been taken, from all aspects, oblique as well as direct, and copies of these photographs are herewith submitted. In the "Reference Handbook of the Medical Sciences," viii., the lateral aspect of the left hemisphere is shown in Fig. 4779, and in Fig. 4781 the dorsal aspect of the entire cerebrum with the ends of the occipital fissures and the central fissures, which latter are completely interrupted by an isthmus at about the junction of the middle and dorsal thirds. The present exhibition is mainly for the sake of enabling members of the Association to see an example of this rare fissural anomaly, the number of cases observed hitherto not exceeding fourteen out of the thousands of brains examined. It seems probable, however, as remarked by Heschl and Dwight, that there is not infrequently an incomplete interruption of the central fissure, constituting a *vadum* (shallow, or "concealed *pli de passage*"), and this should be looked for when practicable.

About fifteen points of interest respecting this brain are noted in the "Handbook," p. 158. Pending a future complete description, the following additions or corrections are made now:

The semicircular fissure just ventra of the postcentral is probably the *subcentral*, first observed by the writer in brains numbered 385 and 2268, in the museum of Cornell University ("Handbook," viii., 152, note). The partial exposure of the insula in this brain of a highly intellectual person is especially noteworthy in connection with its similar exposure in an uneducated mulatto, and its complete occlusion in most apes and monkeys. A careful study of the insula will be made by the removal of the several operculums, kindly authorized by Prof. Dwight.

In the published figure the bifurcated fissure between the orbital and the ventral end of the precentral is identified as the presylvian ("ascending branch"). Recent studies of this region have thrown doubts upon this determination and upon Herve's affirmation as to the greater constancy of the subsylvian ("horizontal branch") in Primates.

In this connection the attention of members of the Association was asked to the printed "Commentary upon Fissural Diagrams;"¹ also to the series of large wall-maps (about two metres square), recently made under his immediate direction, by Mrs. S. H. Gage, from specimens prepared by him for Cornell University. They embrace: (*a*) the mesal aspect of the entire head of a man and a chimpanzee whose brains were hardened in the cranium by continuous alinjection; (*b*) the lateral aspect of the brain of the chimpanzee and an adult mulatto; (*c*) the brain of a child at birth; (*d*) six human foetal brains at as many stages of development, illustrating especially the formation of the Sylvian fissure.

THE CONTRACTION OF THE HEART AND ORDINARY STRIATED MUSCLE,

was the title of a paper, presented by Dr. Thomas J. Mays. It is well known that under certain conditions the degree of contraction of a skeleton muscle varies with the stimulus applied to it. Inquiries into the nature of cardiac contraction (Bowditch, Kronecker, Stirling) have taught us that the heart differs widely in its mode of contraction from that of striated or skeleton muscle. The feeblest stimulus which is capable of calling forth a contraction acts like the strongest—the most complete or maximum contraction being, therefore, induced by the weakest or minimum stimulus (Landois). The cause of this behavior is supposed to be inherent in the structure of the heart muscle itself. The

¹ Copies may be had upon application to Dr. Burt G. Wilder, Ithaca, N Y.

muscular elements of the heart partake of the function of nerve fibres (Aubert), and on account of the anatomical variations the heart differs in its function from that of other striated muscles.

The experiments of Wundt and Walton show that during strychnine poisoning "a stimulus which is strong enough to produce any reflex contraction in a muscle will not react more strongly if the greatest possible stimulus is applied. The range of stimuli through which the contraction varies with the intensity of the stimulus becomes shorter as the effect of the poison increases, and when a certain grade of poisoning is reached the step is infinitesimal from a stimulus which produces no contraction to one which produces a maximum." (Walton.)

When irritability is viewed, therefore, as it exists normally in voluntary muscle, it is seen that varying degrees of contraction are obtained from this organ when varying degrees of electric stimulation are applied to it; but this differential responsiveness vanishes so soon as its irritability is increased through the influence of strychnine. The irritability of the normal heart he regarded as being analogous to that of voluntary muscle under the influence of toxic doses of strychnine, for here, in virtue of the heightened irritability of the heart, the degree of electric stimulation which provokes a cardiac contraction at all is too powerful to produce anything else than a maximum contraction.

DIPHTHERIA AND DIPHTHERITIC PARALYSIS.

was the title of a paper, read by Dr. James Hendrie Lloyd. The weight of opinion seems to be that the specific poison of diphtheria is either a bacterium or the ptomaine generated by it. This gives us at least a working theory, upon which an active and even aggressive treatment can be established. Diphtheritic poison acts distinctly like other morbid agents upon nerve tissue. There is little doubt that it acts especially upon what Gowers calls the lower "segment" of the nervous system; that is, the large cells in the anterior horns and the nerve fibres running out from them to form the nerve trunks. That some observers have located the lesions in the horns and others in the nerve fibres only, furnishes additional evidence that these two parts form really one anatomical organ, and that the diphtheritic poison acts probably upon the whole. It might even be claimed on good clinical evidence that it is a general protoplasmic poison which does not confine itself to one group of cells. Arsenic, alcohol, and some other substances attack many tissues of

the body and are similar in their effects to the diphtheritic poison. If a violent protoplasmic poison is being generated in the body, the most important indication is to prevent its generation by early local treatment. Secondly, having the lesions of a peripheral neuritis, it is possible to give an intelligent prognosis, and to carry out efficient though somewhat expectant treatment.

Chlorate of potassium seems to be losing the confidence of the profession in the treatment of diphtheria, because large doses have an injurious effect upon the kidneys. Chloride of ammonium will do all that chlorate of potassium can do, without its bad effects. Calomel is a good remedy, without the same prompt results as the combined chlorides of ammonium and iron. The corrosive chloride of mercury, both locally and internally, appears on some accounts the ideal remedy for diphtheria.

Nervous phenomena must not be regarded as sequelæ of diphtheria, but as a part of the general symptom group. Bernhart pointed out that the knee-jerk is abolished in many cases of diphtheria which do not exhibit distinctly paralytic symptoms. But the broader fact is that a toxic agent begins early and probably in all cases, to threaten the integrity of the peripheral nervous system. Heart failure, due probably to involvement of the vagus, is the most alarming symptom, and occurs without other paralytic manifestations. In general multiple neuritis of diphtheritic origin, the prominent symptoms may not be noticed for several weeks after the disappearance of the primary disease. The treatment of the paralysis itself is in the main expectant. Nerve lesions demonstrated in autopsies are degenerative—destructive. There is destruction of the axillary fibre, segmentation of the medullary substance, and proliferation of the cell-elements in the nerve-sheath. The repair must be by a gradual and rather slow nutritive process.

Strychnine has been recommended, and digitalis in heart failure. Alcohol, as a more diffusible stimulant, is better than digitalis. The most important indication is to feed; or, as has been said, "to keep the blood-vessels and lymph spaces full." The phosphates, especially in the old form of Parrish, are invaluable in the treatment of diphtheritic paralysis.

In adults, diphtheritic paralysis can be mistaken for locomotor ataxia, as in the case of a man who had numbness of the extremities, slight ataxia, abolished patellar reflexes, and suspicious change in the pupils. The principal points

of distinction were the loss of muscular power, flabby muscles, absence of fulgurant, absence of Romberg's symptom and the history of earlier diphtheria. The changes in the pupils in diphtheritic paralysis are the opposite of those occurring in the Argyle-Robertson phenomenon of locomotor ataxia. In diphtheritic paralysis the power of accommodation is lost to near objects, and not the reflex to light.

Dr. KNAPP was much surprised at the statements made by Dr. Lloyd as to the treatment. That diphtheria was due to a localized infection might be granted. It was, however, of as little use to attack local manifestations of hysteria or the initial lesion of syphilis, in the hope of getting rid of those troubles by that means. Certainly Dr. Lloyd's method of using a long-handled brush down the patient's throat seemed very remarkable, and, for young children, absolutely impossible or likely to do more harm than good. The use of syringes, irrigation by the atomizers and with steam, laden with disinfectants, would certainly not irritate or excite the patients, and seemed infinitely better treatment. If there were marked constitutional symptoms, every effort must be made to keep up the strength. The speaker always used strychnine in large doses, and in almost all cases it had been well borne. The chief danger, apart from the heart failure, which was not as late a manifestation as the true diphtheritic paralysis seemed to be failure in nutrition from paralysis of the pharynx. This was a point which should be early guarded against.

Dr. LLOYD said that modern research showed that we had a local infectious disease to deal with, from which point the system became involved, and we must attack the disease locally. The brush was to be preferred to the spray. The latter was useful for the nasal chambers. Chloride of iron and ammonia should be used for the throat. He did not advocate caustic applications, but only mild antiseptics, lotions thoroughly applied to the local disease.

TUMOR OF THE QUADRIGEMINAL REGION, WITH SPECIAL REFERENCE TO OCULAR SYMPTOMS.

Dr. B. SACHS read a paper with this title. He had been fortunate enough to obtain two autopsies, during the past year, which bore upon this question, and also several cases which were subjected to careful clinical examination. His first case was one of unusually severe tuberculosis cerebri. The main points of the history, which he had been able to complete through the kindness of several colleagues, were these :

E. L.—, aged three years. When first seen she had double ptosis, but no other ocular paralysis was observed. She was dull and listless, and had a pulse that ranged from 145 to 160, but with normal temperature. The mother had noticed a change in the child's disposition since an attack of measles nine months previously. The child did not care to play, but preferred to sit quietly in a chair all day long. She staggered in walking and occasionally fell. She had no epileptic attacks, and had vomited but once. Knee-jerk was absent. The right hand was weaker than the left. No anæsthesia or ataxia. There was paresis of both levator palpebrarum, the pupils being half covered. No nystagmus. Pupils were equal, moderately dilated, and reacted well to light and accommodation.

December 28, 1889, the patient had come under Dr. Sachs' care. The condition at that time showed great changes. Examination disclosed double and almost complete ptosis. There was no upward or downward movement of either eye. Both external recti muscles were thrown into clonic spastic condition when the attempt was made to use them. The interni were capable of very slight movement, but all the other ocular muscles were completely paralyzed. The accommodative reflexes were still distinct, and there was slight contractility to light. There was also slight left facial paresis. The vision was very much impaired. Although in a semi-stupor, the child could be made to walk, and then exhibited most distinct cerebellar staggering, walking with a broad base, and almost falling to the right side. The oculist reported plaques of choroidal atrophy below the macula of left eye. The reflexes were exaggerated, and there was occipital headache. The diagnosis of tumor of the corpora quadrigemina was given. The tumor was supposed to be associated with a general tubercular meningitis. The child grew rapidly worse, and, after passing through several convulsive seizures, becoming blind, and finally developing left hemiplegia, she died February 4, 1889.

Autopsy showed the dura adherent to the skull, and it had to be removed with the calvarium. The quantity of the sub-dural fluid was slightly increased. A solitary tubercle was at once discovered near the right lateral sinus, pressing into the lateral edge of the cerebellum and producing thrombosis of the lateral sinus. Other tubercles with large areas of softened tissue were found in the cerebellum. Deep examination of the brain showed the hemispheres to be healthy, with the exception of the small

tubercular deposits along the paths of the blood-vessels. The cerebellum was the seat of the most profound changes. The base presented several unusual conditions. There was great thickening of the pia, with small tubercular deposits between the corpora mamillaria and optic chiasm, and in the interpeduncular space. The thickening at this point was so great that both third nerves, instead of lying across the crura, after removal of the brain, pointed backward, and the right, the sixth, was twisted out of its position.

Section of the brain showed the tumor to occupy almost the centre of the tegmental division of the crus, and had left a very small portion of the corpora quadrigemina and the brachi intact. The occipital headaches and the cerebellar staggering were the only symptoms which could be ascribed to the large tubercles in the cerebellum, though both these symptoms might be due to the lesion of the quadrigemina region. It was probable that the sixth and seventh nerve-nuclei were responsible for the symptoms pointing to lesions of those nerves, or that the basilar meningitis was at fault. Certain it was that the latter condition was late in developing, for, for months, the symptoms had been distinctly nuclear. In spite of the manifold morbid conditions, it was most remarkable that the ciliary muscles and the sphincter irides had remained exempt during the entire period of observation. Considering the compactness of all cerebral structures in the crura, it would be supposed that there could be no difficulty in making a differential diagnosis between cases of tumor in this region and a chronic inflammatory process.

CRUS LESION.

This was the title of a second paper by Dr. SACHS. Crus lesions were rarer than many other cerebral lesions, but their symptoms were well marked. The case under consideration had some special interest, however, in connection with post-hemiplegic disturbances of motion, and from this point of view the results of the post-mortem examination were worthy of consideration.

Seven years ago the patient, a woman, about fifty years of age, had had a dizzy attack one morning, and had found her vision rather blurred. There was a recurrence of the attack in fifteen minutes. There was no unconsciousness nor difficulty with speech, but when the patient attempted to walk she found she could not with ease. By morning she had almost complete left hemiplegia; she could not open either eye. At that time speech was heavy and indistinct, but from this she had recovered in three weeks.

Hearing, taste, and smell were altogether normal. The hemiplegia was never recovered from, the patient became somewhat unruly and demented, and was finally taken to the Montefiore Home, where she had remained for many years.

A few further details of the patient's chronic condition were elicited in examination. There had been no history of syphilis, but there was very marked atheroma of the peripheral arteries. In addition to the left hemiplegia, the patient had suffered amputation of the right leg above the ankle, for old necrosis of the tibia, fully six years before. There was rigidity of the left leg, and increased knee-jerks of both sides. The wrist reflex was decidedly increased on the paralyzed side, but the left upper extremity was subject to the wildest ataxic movements. This would go on until the arm dropped from exhaustion, when it would remain quiet until aroused again by an effort to use the hand. She became extremely emotional, took very little nourishment, and finally died. The diagnosis of crus lesion of the right side, probably softening from thrombosis, was made, and confirmed by the autopsy.

NEW YORK NEUROLOGICAL SOCIETY.

Meeting of October 7th, 1890.

The PRESIDENT, Dr. L. C. GRAY, in the chair.

Dr. W. B. PRITCHARD presented a specimen, and the history of a case of "Tuberculous Meningitis," see p. 720.

CAN WE DIAGNOSE HYPERÆMIA OR ANÆMIA OF THE BRAIN AND CORD?

Dr. WILLIAM A. HAMMOND read a paper on this subject. The writer had for many years been familiar with a group of symptoms, which, from their etiology and general characteristics were indicative of cerebral disturbance, and some twenty-five years ago, after considerable observation and many experiments performed upon living animals and the human subject, he had come to the conclusion that they were the result of an increase in the amount of blood circulating in the vessels of the brain. His conclusions were first published in an article on "Insomnia," in 1865, various papers appearing on the subject at subsequent intervals, and lastly in a monograph issued in 1884, entitled "Cerebral Hyperæmia the Result of Over Mental Work or Emotional Disturbance," in which additional facts, the outcome