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WALTER TIMME, M.D., *President*

A CASE OF LETHARGIC ENCEPHALITIS. Presented by DR. ISADORE ABRAHAMSON.

An unusual case of lethargic encephalitis was presented for the purpose of showing how intensive study of this disease had shed light on many obscure points in the pathology of diseases of the nervous system. New names are frequently attached to unusual variations of one disease, and discoveries of distinct disease entities are heralded without cause. The patient illustrating the complexity of encephalitis manifestations was an Italian butcher. His previous personal history was negative. The initial symptoms were pain in the mastoid, left occiput and left shoulder. There was fever for three days, followed by a lethargic sleep for two weeks, from which he was easily aroused, answering in a typical fashion. Movements set in, being first present in the left shoulder and afterward in the left index finger. The other hand was soon affected and the speech became markedly involved, being dysarthric, not aphasic. There was no fine tremor. The legs were involved later. Pains and paresthesia in the suprascapular region and nape of the neck have persisted from the time of the onset nine months ago until the present. The patient complained of formication about the left shoulder. Neurologic examination showed the right pupil ovoid up and down and sluggish to light; accommodation was normal. The left pupil was ovoid from side to side; accommodation was not so good; it was sluggish to light.

The most marked feature exhibited by this patient was the nature of the movements of the upper extremities, the left especially. The movements were slower than those of chorea and resembled intentional movements, being slow, coordinated and asymmetrical. There was no fibrillation or myoclonia. Every movement of a muscle is followed by movement of a part, both antagonistic and synergic. There is thus presented in this patient, as a late sign of an undoubted lethargic encephalitis, a disturbance of motility, without ataxia, without loss of power, atrophy or dysmetria, showing no dyssynergia or myoclonia. There are evidences of sensory irritation still persisting. The movements are neither choreic nor paramyoclonic, nor do they resemble those of dyssynergia cerebellaris. They are not hysterical. They are not the movements of dystonia. There is no torsion spasm of the pelvis or neck. They are extrapyramidal, not induced by pain or paresthesia. It is impossible to designate the affliction by any accurate form in the recognized categories. The accepted classification of disease according to manifestations is here clearly seen to be untenable.

A CASE OF LETHARGIC ENCEPHALITIS. Presented by DR. THOMAS K. DAVIS.

The patient was a young woman in whom encephalitis had been a complication of pregnancy. The encephalitis began three weeks before the birth of a child in February, 1920, and quite early took the pseudoparkinsonian form. With this, the arms assumed a constant tonic contracture across the chest, each elbow being strongly flexed. They could be passively extended only with great difficulty. The physician who saw her at that time placed the arms in full extension in plaster casts, which were worn for three weeks. This resulted in the arms becoming rigid at full extension. It has been quite impossible to flex either elbow since.

There is usually a fine tremor of the hands. There have been no pyramidal tract signs.

A roentgenogram of the right arm showed a faint suggestion of new bone formation at the elbow, possibly beginning ankylosis. In the left arm, bony changes were absent and the high degree of muscular tone apparently alone produced the rigidity of this arm.

DISCUSSION

DR. FOSTER KENNEDY pointed out that this second case was practically the reverse of that shown by Dr. Abrahamson. There was apparently overtoneing of the cervical muscles. The trunk muscles were not affected.

DR. ISADORE ABRAHAMSON called to mind a patient with spondylos rhizomelique who exhibited the same pains, attitude and rigidities as the second patient, in whom the condition had been the result of immobilization. There were, however, no signs of cord or brain implication. Dr. Abrahamson regarded the case presented as both lethargic and spondylotic.

DR. C. B. CRAIG had seen the first patient at the Neurological Institute for a period of two weeks in June. His condition had not changed from that time. It was observed that the motility ceased during sleep. Faradism had proved to be beneficial, its effects lasting from one to three hours. This was probably a fatigue phenomenon. The case was diagnosed as chorea following lethargic encephalitis.

DR. BERNARD SACHS remarked on the interest attached to two such diametrically opposed cases. He said he hoped to gain anatomic information from them and was especially interested to know what part of the brain was responsible for the disturbances of motility. The condition of the last patient resembled certain infantile hemiplegias that he had seen. In Dr. Abrahamson's case the movements were distinctly choreic, arrhythmical, more or less purposeful and of slightly explosive character. They were slower than ordinary chorea minor, and not so fast as electric chorea, yet they were distinctly choreic as opposed to myoclonic and myotonic. It would be interesting to know the part of the nervous system that was responsible. The movements were evidently the result of encephalitic processes and were certainly not hysterical. Dr. Sachs' belief was that it was a ganglionic disturbance near one of the larger ganglions and not a cortical disturbance.

DR. MICHAEL OSNATO stated that he had had the first patient under observation since May, 1920, when he was shown to the students at Columbia as a case of encephalitis. He had noticed a rhythmicity of movement, the movements occurring four or five to the second. A considerable improvement had

undoubtedly occurred. Malarial parasites had been detected in the patient's blood and suitable treatment had been given.

DR. SMITH ELY JELLIFFE said that the syndromes of postinfluenzal and post-lethargic encephalitis were becoming more interesting, and they were turning up in greater abundance in private practice, as the increasing attention given to them, in the advancing masses of the literature, was making physicians more observant of the possibilities. These cases had been in existence for many years but heretofore had been distributed into larger and looser nosologic conceptions; and their etiologic relationships to the infections, especially to that of influenza, were largely overlooked.

In the cases under consideration, particularly the case of Dr. Davis, emphasis had been laid on the segmental distribution of the tonic fixation. It involved an entire limb. One point of possible localization had been omitted thus far in the discussion to which Dr. Jelliffe thought attention might be directed. This was the possibility of cerebellar pathway disturbance in certain types of cases, of which Dr. Abrahamson's case might be an example. The studies of Bolk and Rynberk have cleared up much of our knowledge concerning the localization of function of limbs in the cerebellum, and some cases were on record in which segmental motor disturbances of a choreic or tic-like character, as seen in Dr. Abrahamson's case, could be referred back to foci of destruction in the cerebellar mechanisms. Thus Klein (*Monatschr. f. Psychiat. u. Neurol.*, 1918) reported an interesting choreic-like disturbance from a small cyst in the dentate nucleus. As the fatal cases are usually fulminating and rarely afford opportunity to clear up the complete pathology, it would probably take many years before the valuable localizing lessons could be gained from the study of these residuals of mesencephalic involvement.

DR. ABRAHAMSON, in summarizing the discussion on his patient, said that he doubted the correctness of the localization in the cerebellum, since the patient showed none of the symptoms of cerebellar disease. His case was distinctly one of disease of the communicating pathways and not of the centers.

A CASE OF PYRAMIDAL SCLEROSIS FOLLOWING ELECTRIC BURNS. Presented by DR. FOSTER KENNEDY.

The patient had been working in front of a dead cable when a charge of 1,100 volts was sent through it. He remembered nothing of the accident and was unconscious for eleven hours after it. The accident occurred six years ago, and he was paralyzed immediately thereafter. He now has spastic legs and double ankle clonus. He has control of the bladder, but incomplete control of the rectum. A large scar is visible on the left scapula, showing the point of ingress of the charge, and a smaller one is found on the left buttock at the point of exit. There were no neurologic signs of interest except the spastic paraplegia. The burn was a third degree burn. Within two years of the injury the patient began to have a double cataract. The lens in the right eye has been removed. Dr. Kennedy said that his information in regard to nervous system results of this type of injury was slight, and asked for any points of interest from the audience.

DR. SMITH ELY JELLIFFE said that by chance he had just finished a revision of the chapter on electric injuries in Peterson and Haynes' "Legal Medicine" and had thus had occasion to review the extensive literature. Results of electric injuries of this type producing myelitis from lumbar foci, were

extremely rare, but were well known. A review of the entire literature has been given by Jellinek quite recently.

DR. ISADORE ABRAHAMSON asked whether the pathology of this case would resemble cases of caisson disease.

DR. SMITH ELY JELLIFFE pointed out that the force of the current acted as a disruptive agent, whereas the gas in caisson disease had an eruptive effect.

DR. MICHAEL OSNATO was interested to know why the current had selected the pyramidal tracts and had missed the others. It appeared strange that the other tracts and cells and columns of the cord had so completely recovered.

DR. SMITH ELY JELLIFFE said that the current was not selective but that now six years after the accident much of the condition had cleared up. Disturbances of vegetative function had existed, however, and were still in evidence, notably the cataract.

CLINICAL SYPHILIS OF THE CENTRAL NERVOUS SYSTEM. Presented by DR. JUNIUS W. STEPHENSON.

In a plea for the more fundamental instruction of medical students in the practical aspects of syphilology, a brief classification along lines suitable for complete and rapid instruction was given. In attacking the nervous system, Dr. Stephenson pointed out, syphilis rarely attacks en masse, but produces symptoms referable to a patch of meninges here or there, a blood vessel of small caliber, or a tract in the spinal cord. The accuracy of diagnosis largely depends on the individual's knowledge of the anatomy and physiology of the nervous system. The successful treatment of syphilis accordingly depends on proper discrimination as to what anatomicophysilogic destruction is caused by the initial invasion, the condition of certain structures being unalterable, while others would be amenable to treatment. Thus the importance of the early recognition of syphilis is to be emphasized. When the average patient reaches the neurologist, the damage is, usually, too extensive for much improvement. The ability to recognize the disease in the early stages is therefore of the utmost importance.

The one criterion of syphilis is the Argyll Robertson pupil, but this is not a preliminary sign. Any irregularity of the pupil in patients below the age of 40 years should be considered syphilitic until proved otherwise. A further sign of diagnostic value is one that Dr. Stephenson has styled the "dead man's pupil." Experience is necessary to recognize this sign.

The symptoms of syphilis within the cranium, those of meningitis, isolated gumma, cerebral vascular syphilis, thrombosis of the posterior inferior cerebellar artery, general paresis (meningeal and parenchymatous) and syphilis of the optic nerve, were briefly outlined, following which Dr. Stephenson surveyed the type of syphilis involving the spinal cord, touching on the principle diagnostic points and certain special tests and signs that he has found of value. The entire field of syphilitic disease was rapidly summarized and the earmarks of the various types set forth for rapid and easy assimilation.

DR. FOSTER KENNEDY, in discussing Dr. Stephenson's paper, spoke of the curious refractive phenomenon which caused what Dr. Stephenson had designated as the "dead man's pupil." Although, as he had said, it required skill in learning to recognize it, yet Dr. Kennedy felt that it had a diagnostic value.

STUTTERING: ETIOLOGY AND THERAPY. Presented by MISS HANNAH M. CREASY, by invitation.

Miss Creasy stated that it was her impression that speech specialists should endeavor to gain the cooperation of neurologists if the profession of speech correction were to assume the dignity and gain the respect that it deserved. In her work she had found that there was no royal road to recovery. Her object in presenting her methods was to show the philosophy underlying them.

Stuttering, including stammering, she defined as the inability to speak at will. It is a mental condition, a psychic neurosis, not physical, and frees itself in somatic symptoms, such as cramps and excessive tension of the organs of speech. There is lack of harmonious working between the brain and the speech organs. The speech mechanism is not at fault. It is the mind that is tied, not the tongue. Stuttering must accordingly be traced further back than the symptoms, or even the trauma producing the symptoms.

Every experience which produces a painful effect of fear, anxiety, shame, etc., may act as a psychic trauma, and may produce pathologic results, if it falls on fertile soil. "In order to create nervousness," says Charcot, "two factors are necessary—one permanent (the neuropathic predisposition), the other contingent (the provoking agent)." A shock may have brought out the symptoms, revealing the permanent neurotic background. After the effects of the shock may have been removed and the symptoms cured, another shock may again reveal the constitutional weakness and the cure be undone. This has occurred in many of the so-called shell shock cases in the war.

The dominant quality in the syndrome of stuttering is fear—fear of people, or of speaking before people. On entering school, the stutterer carries his fear over from his home environment to any one in authority, any one he thinks might be hostile and unsympathetic. In the best of environments intelligent sympathy may be lacking. The fear attached to stuttering is a morbid fear. It does not arise from the shock, it arises from an internal condition. The history of frights, shocks, accidents and other attributed causes of stuttering show that they might have resulted in a temporary normal fear. The patient's make-up has predetermined the morbidity of the fear. According to Frink, the manifestation of an anxiety hysteria is a morbid fear and the physical accompaniment thereof. Stuttering may be considered a manifestation of anxiety hysteria.

Accepting the premise that stuttering is a fear neurosis (anxiety hysteria), Miss Creasy's treatment follows along the lines laid down for other psycho-neuroses. The work is largely a matter of character building—reorganization of the emotional life. To counteract the attributes of fear, cowardice, and stubbornness, environmental trends are recognized to bring about a more normal reaction to life, to produce fearlessness, self control and moral courage. An example of this method given was that of a student who, when asked why he stuttered more than usual at one time, replied that he had been worsted in a fight. His tendency to expect defeat was pointed out to him and he was told to approach his next fight with his mind made up to win, and to stay at the job until he did so.

The personal equation of the doctor in this branch of therapy, as in all others, is of great importance. The understanding of the patient, and letting him know that the doctor understands his peculiar difficulties, will go far toward

altering his attitude toward others. Thus, with the friendly cooperation of teachers and schoolmates, Miss Creasy has found that fear will gradually give way.

The stutterer's bodily health and environment should have careful attention. Segregation of stutterers is not desirable. The symptoms may be removed under institutional care, but the problems in the unfamiliar outside world will cause them to return again. Children should be kept in school and in touch with normal life in every possible way.

To attain fearlessness the patient is given a new method of speech. The sufferer is unable to get over the initial consonant sound to the vowel. The psychic fear causes an overinnervation in the occlusive, consonant sounds, and an inability to coordinate the consonant with the vowel in the syllable. The patient is shown that normal speech differs from his in the weaker consonant obstruction. The work proceeds with the syllable, coordinating the consonants and the vowels. Vowel valuation is made the keynote of the work. The patient is trained to consider the vowels as life savers. The mental attitude of fearlessness and self confidence inculcated by the care of the treatment, together with the scientific aids to phonation, when made clear and reasonable to the stutterer, enabled the work to proceed surely and with lasting success.

DISCUSSION

Dr. A. A. Brill agreed that the genesis of the stuttering was in the situation of the patient. Many cases of stuttering starting after puberty were easy to cure, since the stuttering was a symptom of a neurotic condition. Too much time, he felt, should not be devoted to routine practice. Most of the sufferers should be treated as average psychoneurotics. The prognosis in stuttering is usually bad. The treatment is lengthy and trying. The permanency of the cure cannot be guaranteed.

Dr. Neustaedter congratulated Miss Creasy on her conservatism and perseverance. He remarked that stammerers do not stammer when they sing. If the patient can be kept talking slowly the stammering can be largely overcome.

Dr. Smith Ely Jelliffe agreed with Miss Creasy's final summary of the extreme complexity of the situation. He did not believe that even the majority of cases could be ranged in the anxiety hysteria group. The reasons for the types of results obtained lie in the nature of the beginning of the neurosis. The compulsive type, beginning in the auto-erotic period is treated with extreme difficulty, especially when the muscle erotic is involved. Such patients are prone to relapse and require much time. Miss Creasy's effort in establishing a transference is sane and advisable. The most promising part of her presentation, Dr. Jelliffe felt, was the beginning of cooperation between the layman and neurologist. There are not enough neurologically trained physicians to handle psychoneurotic problems in the community. If intelligent cooperation can be established between the neurologist and the sincere helper, much help can be obtained.

Miss Hannah Creasy, in closing, said that since the work in public schools had been developed, rapid advance had been made. Cooperation and team work made the advance more successful. The work in the schools was more favorable than in private work, since in the home there is backsliding. Until the parents will cooperate, therefore, training in schools is advisable. Team work is most necessary to combat the time required and the difficulty of the cure.