HEMIPLEGIA WITH UNUSUAL SPASMODIC MOVEMENTS

C. S. PORRS presented a girl, twelve years of age, in whom a right-sided hemiplegia developed following a scarlatina at the age of five years. Mentally the patient was normal. She was highly emotional and the motor difficulties had increased recently. Neurological status showed a right hemiplegia with tremors. The muscular power was not much inferior to that of the left side. There was a smaller right side. No Babinski. Athetoid movements of the right hand were occasionally observed, and when the patient walked there was an associated flexion of the right arm with grasping of the hair by the left hand. This was accompanied by spasms in the left sternocleido mastoid which shows compensatory hypertrophy. The head was pulled to the right shoulder and backward. On excitement the patient's right arm and leg became rigid and showed clonic contractions. The case was interpreted as one of organic hemiplegia with hysterical exaggeration. The implication of the lenticular region was suggested.

Discussion.—C. K. MILLS emphasized the probable lenticular localization of the lesion. He deprecated operation for the torticollis.

S. D. INGHAM believed that the psychogenic factors were paramount and also denied the utility of operation.

NEW YORK NEUROLOGICAL SOCIETY

THE THREE HUNDRED AND EIGHTY-SECOND REGULAR MEETING,
HELD AT THE ACADEMY OF MEDICINE,
OCTOBER 5, 1920

The President, DR. WALTER TIMME, in the Chair

PRESENTATION OF TWO CASES OF ENCEPHALITIS
LETHARGICA

DR. ISADORE ABRAHAMSON presented an unusual case of encephalitis lethargica for the purpose of showing how intensive study of this disease had shed light upon many obscure points in the pathology of diseases of the mental system. New names were frequently attached to unusual variations of one disease, and discoveries of distinct disease entities heralded without cause. The patient illustrating the complexity of encephalitis manifestations was an Italian, a butcher by trade. His previous personal history was negative. The initial symptoms were pain in the mastoid, then left occiput into left shoulder. There was fever for three days, then a lethargic sleep for two weeks from which he was easily

aroused, answering in a typical fashion. Then movements set in, first in the left shoulder, then in the left index finger. The other hand was soon affected and the speech became markedly involved. It was dysarthric, not aphasic. There was no fine tremor. The legs were involved later. Pains and paresthesia have persisted from the time of the onset nine months ago to the present in the suprascapular region and nape of the neck. The patient complained of formication about the left shoulder. Neurological examination showed the right pupil ovoid up and down, very sluggish to light; accommodation normal. The left pupil was ovoid from side to side, accommodation not so good. Very sluggish to light.

The most marked feature exhibited by this patient was the nature of the movement of the upper extremities, the left especially. The movements were slower than those of chorea, resembled intentional movements, slow, coordinated, asymmetrical, there was no fibrillation, no 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, without atrophy, or dysmetria, showing no dysynergy, nor myoclonia. There are evidences of sensory irritation still persisting. The movements are neither choreic nor paramyoclonic, nor do they resemble those of dysynergy 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 term belonging in the recognized categories. The accepted classification of disease according to manifestations is here clearly shown to be untenable.

The case presented by DR. THOMAS K. DAVIS was that of 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 strongly flexed. They could be passively extended only with great difficulty. The physicians who saw her then placed the arms in full extension in plaster casts, 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.

X-ray of the right arm showed a faint suggestion of new bone formation at the elbow, possibly beginning ankylosis. In the left arm, bony changes are absent and the high degree of muscular tone apparently alone produces the rigidity of this arm.

In discussing these cases, DR. FOSTER KENNEDY pointed out that this second case was practically the reverse of that shown by Dr.

Abrahamson. There was here apparently overtoneing of the cervical group of muscles. The trunk muscles were not affected.

DR. ABRAHAMSON called to mind a case of spondylosis rhizomelia exhibiting the same pains and attitude and rigidities of the second case in which the conditions had been the result of immobilization. There were, however, no signs of cord or brain implication. He regarded the case presented as both lethargica and spondylosis.

DR. C. B. CRAIG had seen the first patient at the Neurological Institute for a period of two weeks in June. His condition was the same then as now. It was observed that the motility ceased during sleep. Faradism had proved a great relief, its effects lasting from one to three hours. This was probably a fatigue phenomenon. The case was diagnosed chorea following lethargic encephalitis.

DR. BERNARD SACHS remarked on the interest attaching to two such diametrically opposed cases. He hoped to gain anatomical information from them. He was especially interested to know what part of the brain was responsible for the disturbances of motility. The condition of the last patient resembled that of certain infantile hemiplegias, that he had seen. In Dr. Abrahamson's case the movements were distinctly choreic, arrhythmical, more or less purposeful, 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. The part of the nervous system that was responsible would be highly interesting to know. The movements were evidently the result of encephalitic processes and were certainly not hysterical. Dr. Sachs' belief was that of a ganglionic disturbance near one of the larger ganglia and not a cortical disturbance.

DR. MICHAEL OSNATO had also 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 post-influenzal and post-lethargic encephalitis were becoming more and more interesting and they were turning up in greater abundance in private practice as the increasing attention to them given in the advancing masses of the literature were 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 nosological conceptions and their etiological relationships to the infections largely overlooked, especially to that of influenza, since this disease was too exclusively conceived of as a respiratory disease.

In the cases under consideration, particularly the case of Dr. Davis, emphasis had been laid upon 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 possibilities of cerebellar pathway disturbances in certain types of cases, of which Dr. Abrahamson's case might be an example. The studies of Bolk and Rijnberg have cleared up much of our knowledge concerning the localization of function of limbs in the cerebellum, and some cases were on record where 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 had reported an interesting choreic-like disturbance from a small cyst in the dentate nucleus [*Monatschrift für Psychiatrie und Neurologie*, 1918]. Inasmuch as the fatal cases are usually fulminating and rarely afford opportunity to clear up the complete pathology, it would probably take 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.

PRESENTATION OF A CASE OF PYRAMIDAL SCLEROSIS FOLLOWING ELECTRIC BURNS

DR. FOSTER KENNEDY showed a patient in whom an almost pure pyramidal tract change had taken place. The patient had been working in front of a dead cable when a charge of 1,100 volts was sent through it. He remembers nothing of the accident and was unconscious for eleven hours after it. The accident occurred six years ago. He was paralyzed immediately thereafter. He has now spastic legs and double ankle clonus. There is control of the bladder, but incomplete control of the rectum. A large scar is visible on the left scapular showing point of ingress of the charge, and a smaller one is found on the left buttock at the point of exit. There are no neurological 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 lense 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. 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. ABRAHAMSON asked whether the pathology of this case would

resemble cases of caisson disease. DR. 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. OSNATO was interested in knowing why the current had selected the pyramidal tracts and had missed the others. It seemed strange that the other tracts and cells and columns of the cord had so completely recovered. DR. JELLIFFE said that the current was not selective, but that now six years after the accident, much 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

DR. JUNIUS W. STEPHENSON in a plea for the more fundamental instruction of medical students in the practical aspects of syphilology, gave a brief classification along lines suitable for complete and rapid instruction. In attacking the nervous system, Dr. Stephenson pointed out, lues 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 depends largely upon the individual's knowledge of the anatomy and physiology of the nervous system. The successful treatment of syphilis, accordingly depends upon proper discrimination as to what anatomical physiological 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 forty years should be considered luetic 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, syphilis of the optic nerve, are briefly outlined, following which Dr. Stephenson surveys the branch of syphilis involving the spinal cord and touching upon the principal diagnostic points and certain special tests and signs that he has found of value. The entire field of syphilitic disease is rapidly summarized and the earmarks of the various types set forth for rapid and easy assimilation.

DR. 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

MISS HANNAH M. CREASEY [by invitation] stated that it was her impression that speech specialists should endeavor to gain the co-operation of neurologists if the profession of speech correction were to assume the dignity and gain the respect that it deserved. In Miss Creasey's 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 defines 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 pathological 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 have been removed, 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, and the nature of the fear is fear of people, of speaking before people. On entering school the stutterer carries his fear over from his home environment to anyone in authority, anyone he thinks might be hostile and unsympathetic. In the best of environments, even, *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 makeup 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 Creasey's treatment follows along the lines laid down for other psychoneuroses. The work is largely a matter of character build-

ing, reorganization of the emotional life. To counteract the attributes of fear, cowardice, and stubbornness, environmental trends are re-organized 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 individual, and letting him know that the doctor understands his peculiar difficulties will go far towards altering his attitude towards others. Thus with friendly cooperation of teachers and schoolmates, Miss Creasey has found that the 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 stuck by an inability to get over the initial consonant sound to the vowel. The psychic fear causes an overinnervation in the occlusive, consonantal 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 consonantal 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.

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 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 very lengthy and trying. The permanency of the cure cannot be guaranteed.

DR. JELLIFFE agreed with Miss Creasey'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 type of result obtained lie in the nature of the beginning of the neurosis. The compulsive type, beginning in the autoerotic period is treated with extreme difficulty, especially when the muscle erotic is

involved. Such patients are prone to relapse and require much time. Miss Creasey'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 the neurologist. Neurology needs more help from intelligent laymen. 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 CREASEY in closing the discussion 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, schools are advisable. Team work was the most necessary to combat the time required and the difficulty of the cure.