

gait with short steps. The patient had hemiplegia with some mental changes. At autopsy no gross changes were found in the cerebral vessels. For that reason Dr. Spiller was kind enough to let Dr. Byrnes have the brain to study in more detail. About 200 sections were made of the brain. Nowhere was there even in the cortical vessels any gross changes. There was, however, a moderate round cell infiltration, particularly about the left chiasm, about the anterior surface, and about the cerebellum. The round cell infiltration suggested luetic infection. He had marked nephritis. The question arises whether a condition simulating arteriosclerosis can be due to a toxic state. Dr. Byrnes saw a case in Washington, in which was a nervous condition supposed to be caused by infection. There was no history of lues, but the patient had chronic appendicitis. Strange to say after appendectomy the condition entirely disappeared. Undoubtedly toxic states can produce these definite changes. Other cases have been described by neurologists.

FAMILIAL MYOCLONUS

By John H. W. Rhein, M.D.

The patients were two brothers, in whose family two other members, a sister and a brother, were similarly affected. The patients were 37 and 39 years of age, respectively. In the case of Marion, aged 39, the symptoms began at 12 years of age and in Robert, aged 37, they began at 12 years of age. In both cases the disease began with a tremor of the right hand extending to the right leg and head and then to the left arm and leg. Robert has not been able to walk for several months on account of the violence of the movement when he attempts to walk and on account of some weakness not true paralysis in the legs. The movements are the same in both cases and consist of to and fro movements of the arm and hand, rotary movements of the head, the head turning to the right, the muscles of the trunk causing jerking back of the shoulders and a rotary movement of the trunk. The legs are affected to a less degree, more in Robert than in Marion. The tremor practically affected the entire musculature but was more apparent in the right arm, neck muscles, trunk and left arm. The movements are mild during rest and become very greatly exaggerated upon emotional disturbances and upon voluntary effort. In the case of Robert there had been contractures in the knee joint which were broken up under ether and did not return. The tendon of the quadriceps femoris was probably cut also.

There was no spasticity of the knees although there was some slight rigidity apparent at times in the case of Marion. There were no contractures except of the tendon Achilles on one side, the right in the case of Robert and the left in the case of Marion. The knee jerks were large and equal on both sides in the case of Robert and slight and equal in the case of Marion. There was no Babinski phenomenon or ankle clonus in either case. There was no nystagmus or extra-ocular paralysis and the pupils responded normally. There were no sensory disturbances.

In the case of Robert the tongue was pushed slightly to the left and was the seat of a tremor. The jaw muscles were affected in both cases. Both calf muscles were atrophied in the case of Marion and the left thigh and leg in the case of Robert. The mental condition of these patients was good. There was some difficulty in speech, consisting of a jerky articulation. There was no true dysarthria or dysphasia. There was no dysmetria, dyssynergia, or adiadochokinesis.

The family history is as follows: The maternal grandfather died of apoplexy and the maternal grandmother of cirrhosis of the liver and senility. The paternal grandparents died of unknown causes in middle

life. One maternal aunt died at childbirth and one maternal aunt and two uncles were living and well.

There were no paternal aunts or uncles. Their father died of apoplexy and their mother of dropsy, having had intermittent attacks of melancholia. There is no history of nervous disease in the mother's or father's family.

One brother died at birth and another of diphtheria. There were another brother and one sister who were affected with the same disease.

The diagnosis in these cases is not clear. At first sight a diagnosis of paramyoclonus multiplex was suggested, but in this disease voluntary acts quiet the spasm and in these cases the reverse is true.

Unverricht has described a familial form of this disease associated with epilepsy. There was an absence of any history of the latter in these cases.

The absence of hypotonia, dyssynergia, dysmetria and adiadochokinesis take these cases out of the category of those described by Hunt under the title of dyssynergia cerebellaris progressiva. These cases above described resemble to a certain extent progressive lenticular degeneration or Westphal's pseudosclerosis. The absence of pronounced contractures and spasticity, and the duration of the disease is against the diagnosis of the former; while the lacking of dementia which is looked upon as characteristic of pseudosclerosis by many is against the diagnosis of the latter.

The cause of the symptoms in these cases is extrapyramidal as there were no exaggerated reflexes and the Babinski phenomenon was absent. It is not improbable that the lenticular nuclei are the seat of the lesion in these cases.

Dr. William G. Spiller said he thought, with Dr. Cadwalader, that these cases should be placed in the pseudosclerosis class. The pseudosclerosis is a condition concerning which we are learning much. It seems to be a lenticular degeneration with changes in the cortex from autopsies obtained. Dr. Rhein spoke of absence of mental disturbance. It is true in most of the cases of pseudosclerosis there has been mental disturbance. Dr. Spiller thought those who state that there must be mental disturbance in pseudosclerosis are going further than facts justify. Recent work has demonstrated that pigmentation of the cornea and of the liver is a part of pseudosclerosis. He did not know whether Dr. Rhein found anything of that kind in his cases. Dr. Spiller said the cases of pseudosclerosis he had reported at a previous meeting were in one family.

Dr. Charles K. Mills said the case presented looked in many respects like one of some form of lenticular disease. It would be remembered, however, that the speaker believed we have a cortico-striate or strio-cortical apparatus concerned with tonicity and it seemed to him that a cortical sclerosis peculiarly situated might, as might also a lenticular sclerosis, give the symptomatology exhibited by the patient who has no sensory symptoms and he believed no marked motor paralysis. A peculiar tremor seems to be the most striking phenomenon in the case, without abnormal reflexes. We might have a lenticular or cortical affection without any marked mental reduction, or at least not any more decided than is present in some cases of lenticular disease.

Dr. Cadwalader said that he had presented the first case just shown by Dr. Rhein before this Society in December, 1912, and it was recorded in the proceedings under the title of "Pseudosclerosis." Dr. Cadwalader referred at that time to certain similarities which it bore to Wilson's progressive lenticular degeneration.

In October of 1914 he had reported this case together with another in the Journal of the American Medical Association as one of Wilson's

lenticular degeneration, and he still believes that it belongs to this general group.

The difference between pseudosclerosis and lenticular degeneration on clinical grounds is by no means clear. Dr. Cadwalader could not agree with Dr. Rhein in regard to his statement that his patients did not have spasticity. It is by no means marked, but is, in Dr. Cadwalader's opinion, perfectly distinct. The term "spasticity" perhaps is not a good one; "rigidity" might be better.

Strümpell has pointed out that the degree of spasticity is greater in Wilson's disease than in pseudosclerosis, and considers this one of the distinguishing features. Cases have been reported with autopsy by German authors, in which lesions similar to those of pseudosclerosis were found in the cerebellum, cerebral cortex and different parts of the basal ganglia. In one of these cases the alterations of the neuroglia tissue were more marked in the lenticular nucleus than in other parts of the brain. It may be that spasticity is more pronounced when the alterations are greater in this region. It seemed to Dr. Cadwalader that Wilson's progressive lenticular degeneration and pseudosclerosis must be grouped together and considered as modified types of the same general disease. It is true that this point of view may appear to be somewhat premature, nevertheless, recent investigations would seem to indicate that this will ultimately prove to be correct.

Dr. Charles K. Mills said he would like to say an additional word regarding the term tonicities which was called out by what Dr. Cadwalader had said about the remarks of Dr. Rhein. The cases of Wilson's disease, so-called, after all only represent one type of acute or subacute disease of the lenticula associated with disease of the liver. Most of the symptoms present are due to aberrant muscular tonicity. The tremor in one of these cases seemed to Dr. Mills—unless it is simply an asynergy of cerebellar origin and of course he did not think it was this—the peculiarity of speech in another of the cases and most of the symptoms presented belonged with symptoms which come under the general head of aberrant muscular tonicity. We confine our discussions and descriptions too much to hypertonicity as shown in a spastic or rigid musculature.

Dr. Rhein stated that he did not look upon his cases as being typical ones of pseudosclerosis, as in his cases there was no dementia, which was characteristic of these cases, nor marked spastic condition of the muscles. In Dr. Rhein's cases there was little or no spasticity. There was at times apparent resistance at the knee joint in one of the cases, which he looked upon as the result of the muscular contractions due to the tremor.

MULTIPLE SARCOMA OF BRAIN

By John H. W. Rhein, M.D.

Dr. Rhein exhibited the brain which was the seat of a multiple sarcoma. The patient was admitted to one of the state hospitals for the insane, having been found unconscious along the roadside. His condition at the time of his admission was one of partial amnesia. He was talkative but unable to give his name. He had a partial insight into his condition. He stated that he could not get the words but believed that sooner or later that faculty would come to him. When shown an object and told to name it he would say, "I cannot for the world tell you, but I think it will come sooner or later."

The patient was 56 years old and the history otherwise is lacking except that there were inequality of the pupils and slight impairment of motion of the right leg. The brain was referred to Dr. Rhein by Pro-