

mouth and pharynx. This attack gradually subsided without confining the patient to bed.

May 10: The patient was feeling unusually well in the morning, but was taken in the afternoon with a sudden pain about the heart, radiating down the left arm, during which the cyanosis was increased.

June 1: Pain and tenderness suddenly developed in the calf of the right leg, resembling the results of a severe sprain, but there was no history of injury or unusual exertion to account for it. The soreness, which was acute, yielded but slowly to rest and applications of heat, liniments, clay dressings, etc. The patient was confined to bed for a week, and then was only able to get about with crutches for another week. There was no local swelling, redness or other signs of inflammation. This attack may have no relation to the general trouble, but may be related to the muscular cramps reported by some observers. Another special symptom was an annoying pruritus which came after bathing. This affected the legs especially and lasted fifteen to thirty minutes after each hydrotherapeutic application. The sensation was described as a pricking or stinging so severe as to make scratching almost irresistible. There was no eruption or redness of the skin. This symptom had been noticed at times for the past five years.

Treatment.—The treatment was chiefly symptomatic, tonic and eliminative. The constipation was met by a laxative diet, stimulating applications of hydrotherapy over the liver and abdomen, massage, mechanical vibrations and electricity. After the first two weeks the bowels moved daily without any form of cathartic. The intestinal autointoxication was met by an antiseptic, low-proteid dietary, with charcoal and lactic acid cultures, etc. The cephalic congestion was counteracted by derivative applications of heat to the feet and legs with cold to the head. General eliminative and tonic measures, such as the electric light bath, salt glow, sprays and douches, especially over the liver and spleen, were used daily, together with cold baths and an outdoor régime to increase oxidation. During the seventh week, just after the attack of soreness in the leg, the patient was put on potassium iodid rather experimentally. This was gradually increased from 30 drops *per diem* to 60 and was continued over a period of eight weeks, being discontinued two weeks before the patient left. No special effect was noted from this, unless it was a temporary lowering of the blood pressure, which during this period was recorded successively at 140, 135 and 120 mm. The reading, taken one week after the potassium iodid was stopped, however, showed the pressure to be again 145. These fluctuations may have been due to some other cause, but if due to the iodid it suggests that the result came from some effect on the heart or the caliber of the blood vessels rather than to any change in the condition of the arterial walls, since the fluctuation was too rapid for actual tissue change. The *x-ray* was applied to the spleen during the last two weeks of treatment, the patient receiving in all six applications. No results were observed during this short period. During his entire illness the patient was able to sleep well. The appetite was generally good, the patient eating on an average 2,000 calories per day. The weight was normal and constant.

Later Course.—During treatment the patient made a very decided gain, especially in his subjective symptoms. There was no further exacerbations of the symptoms after the second week. His strength and endurance gradually increased, and the patient reported a very marked improvement in his mental activities, the feeling of "grogginess" having disappeared, together with the giddiness which had been troublesome. The general cyanotic appearance of the face and hands was no longer marked, and even the ears, lips and nose had faded to nearly normal color. The dilated venules about the nose were still quite noticeable, however. As already noted, there had been temporary reductions in the number of red cells and in the blood pressure; but the last test showed the hemoglobin to be still in excess of 120 per cent., the red cells 140 per cent., white cells 175 per cent. and blood pressure 125 mm. While the patient felt so much improved physically and mentally that he went home on August 19 to give his business affairs some much needed attention, it is probable the relief was only

temporary and his trouble will be gradually progressive. We shall, therefore, endeavor to follow the further history of the case through his home physician.

To summarize the characteristic features of this case on which the diagnosis was based, we have cyanosis, headache, vertigo, general weakness (in this case more mental than physical) marked increase of hemoglobin and red cells and enlargement of the spleen. In this case the enlargement of the spleen was very moderate, being absent at the first examination or so slight as to be overlooked. Dyspnea was not complained of in this case.

IDIOPATHIC EPILEPSY COMPLICATED BY MOTOR APHASIA AND DIPLEGIA, WITH NECROPSY.*

WILLIAMS B. CADWALADER, M.D.

Pathologist to the Philadelphia Orthopedic Hospital and Infirmary
for Nervous Diseases,
PHILADELPHIA.

Muscular debility following repeated severe epileptic convulsions may be so marked as to give the impression of true paralysis. It is probably analogous to that described by Todd under the name of "epileptic hemiplegia."

In one of his lectures Todd says:

A patient has a fit, distinctly of the epileptic kind; he comes out of it paralyzed in one-half of the body; generally that side which has been more convulsed than the other or which has alone been convulsed; but the paralysis may occur when both sides have been convulsed equally. The paralytic stage remains for a longer or shorter time, varying from a few minutes or a few hours to three or four days or even much longer. It then goes off, or improves until the next fit, when a train of phenomena precisely similar recurs with like result.

He then goes on to describe eleven cases, and suggests that it is probably caused by exhaustion of the cerebral centers.

As this condition does not seem to be very common, the following case should be of interest. I wish to take this opportunity of thanking Dr. J. K. Mitchell for the privilege of studying and reporting this case, and also Dr. Spiller for verifying my pathologic examinations.

Patient.—A. R., female, aged 14, was admitted to the Orthopedic Hospital and Infirmary for Nervous Diseases, Jan. 22, 1908. Her parents and four brothers were well. The patient was born at full term, after normal labor, weight 7¼ pounds. She developed normally and was perfectly healthy; walked and talked at 1 year; was very intelligent and did well at school. She had measles in May, 1900, and made a good recovery.

Onset of Disease.—During July of this year the patient's family noticed that she was getting nervous, and at times very excitable; she was then sent to the seashore, and while there was very much frightened by seeing a friend in an epileptic convulsion. She returned home in September and seemed well. While she was eating, her arm was struck by one of her brothers, so that the spoon was sharply pressed against the muscles of the right side of the mouth; then there suddenly began a clonic spasm at the point of injury, which gradually extended till all the muscles of the right face were involved. This attack was confined to the right face and lasted only one or two minutes, without loss of consciousness. Three similar attacks occurred at intervals of a month, always apparently brought on by contact of a spoon at the angle of the mouth. During the last attack the right arm was violently

*From the Laboratory of Neuropathology of the University of Pennsylvania.

extended and rotated outward and consciousness was partially lost. Under treatment the patient was free from attacks until August, 1902, when there was a series of six typical nocturnal epileptic convulsions, involving chiefly the right side of the body. For the next year and a half there were a few right-sided convulsions with loss of consciousness. In 1904 attacks became more frequent and more general, the two sides of the body being affected equally, followed by difficulty in talking and with weakness and incoordination of the extremities which lasted a few hours at the time. In July, 1904, after a severe general convulsion, with loss of consciousness, the patient became totally aphasic. From this time on there were many major and minor attacks with increasing weakness of the extremities and incoordination, so that the patient was confined to her bed.

Physical Examination.—The patient lay in bed with a vacant expression, the mouth half open, saliva dribbling from the right side. The pupils were equal and reacted to light and accommodation. Ocular movements seemed normal in all directions. The tongue could be partially protruded, but with difficulty. There seemed to be difficulty in swallowing. The patient could not speak but could make sounds, and understood clearly both written and spoken language. The facial muscles were not paralyzed but contracted sluggishly. Both upper extremities were very weak, but not totally paralyzed. The forearm was partially flexed on the arm and the hand on the wrist, on account of the spasmodic contracture of the flexor muscles, but this could be partially overcome by manipulation. All voluntary movements were restricted, and slowly and laboriously performed with marked incoordination. The tendon reflexes were equal and greatly increased. The muscles of the neck and back were weak. Spasmodic contracture was quite marked in the adductor muscles of the thighs and extensors of the feet. Movements were restricted, weak and incoordinate. Tendon reflexes were equal and much increased. There was a distinct Babinski reflex and ankle clonus on both sides. Sensation for touch and pain was everywhere less than normal. Dr. Langdon reported that the pupils were equal in size and reacted promptly to light and accommodation. The ocular movements were full and equal in all directions. The fundi were normal. Vision was normal. Color fields could not be obtained satisfactorily. Dr. Wood reported that the fauces were congested. Digital examination showed a small adenoid in the vault, no relaxation of palate, and apparent loss of sensation. No evidence could be found of any mass or growth above the larynx which would obstruct respiration. Laryngeal examination was impossible on account of the mental condition of the patient.

Course of Disease.—During the following six weeks, in spite of larger doses of bromid, there were one hundred and thirty-two severe general convulsions, with loss of consciousness. During the interparoxysmal stage, from time to time, the contractures were not so marked and there was slight increase of muscular power, but on the whole there was very little change. Finally the patient developed a severe follicular tonsillitis and died two weeks later of what appeared to be clinically a general streptococic infection.

Autopsy.—This was performed two hours after death. The brain and spinal cord only were obtained. On removal the brain was moderately soft and the vessels were intensely congested. There was a small amount of straw-colored fluid beneath the meninges. The convulsions were well formed. On section the ventricles were not distended. There was no localized lesion apparent to the naked eye. The macroscopic appearance of the cord showed nothing abnormal.

Microscopic Examination.—With the Weigert method the pyramidal tracts of the spinal cord were slightly paler than normal, but there was not a distinct degeneration of the fibers; with the hemalum and fuchsin method there was no evidence anywhere of inflammation. The blood vessels and pia appeared normal. With the Nissl method there was a moderate chromatolysis of the ganglion cells of the anterior horns. The medulla oblongata, pons and basal ganglia, studied by the Weigert, Nissl, hemalum and fuchsin methods, showed nothing abnormal except for a few scattered minute capillary hemor-

rhages. Portions of the cortex taken from the motor convulsions of both sides, parietal lobes, Broca's area, frontal and occipital lobes, studied by the same methods presented occasionally a few scattered capillary hemorrhages. The pia here and there showed a slight accumulation of small round cells. No characteristic pathologic change was found and there was no evidence of hypertrophic sclerosis.

I consider that the pathologic examination should be looked on as entirely negative, as the slight changes which were found could easily be accounted for by an infection which in this case was the immediate cause of death.

In many respects this case is unique. Paresis of the extremities and of the muscles of speech was at first slight, but gradually became more profound as the convulsions increased in frequency, till finally there was almost total paralysis of all four extremities with flexure contracture and incoordination and total motor aphasia. When the patient attempted to stand, her legs gave way and the head hung forward. The general appearance was not unlike that in infantile pseudobulbar palsy. All over the body there seemed to be a diminution of pain sense; by some authors this has been attributed to the slowing of associated activities which exists after an attack. Increase of tendon reflexes, with ankle clonus and Babinski reflex, shortly after an attack, has been described by Jackson,¹ Beevor,² Vorkastner³ and Gowers.⁴

During the first few weeks that the patient was under observation it was frequently noted that when the nurse attempted to administer food or medicine, as soon as a spoon was brought in contact with the muscles of the right side of the mouth there immediately began a clonic spasm of the right side of the face, which spread quickly till the whole body was equally convulsed, accompanied with loss of consciousness. Later, after the patient had taken much bromid, the convulsions were confined to the face and consciousness was partially preserved. Finally, after some weeks, direct contact at this area was no longer followed by convulsions. It would seem as if this might fairly be considered an example of that rare variety of epilepsy associated with what has been termed the "epileptogenic zone." Motor irritation over a moderately well-defined area seemed to bring on a convulsion.

An interesting case, presenting somewhat the same phenomena, has been described by Clark,⁵ in which motor or sensory irritation over the biceps muscle was followed by a convulsion. Motor aphasia, partial or complete, following epileptic seizures, although uncommon, has frequently been described and is probably due to a condition involving the cerebral centers concerned in articulation, which is similar in character to that which causes the paralysis of the extremities. Cases presenting more or less complete paralysis of one or more extremities following idiopathic epileptic seizures, have been reported by Féré,⁶ Voisin, Dutil,⁷ Echeverria,⁸ Hughlings Jackson,¹ Gowers,⁴ Pierce Clark⁵ and others.⁹

Although a great variety of lesions have been found in the brains of epileptics, no constant causal pathologic

1. Med. Times and Gazette, February, 1881.

2. Quoted by Gowers.

3. Vorkastner: Diseases of the Nervous System, 1908: Epilepsy.

4. Epilepsy and other Chronic Convulsive Diseases, London, 1881.

5. Arch. Neurol. and Psycho-pathology, 1899, II.

6. Compt. rend. Soc. de biol., 1896.

7. Rev. de méd., 1883, p. 161.

8. Epilepsy, 1870.

9. Todd: Clinical Lectures on the Nervous System, 1856, lecture xiv, Epileptic Hemiplegia.

condition has yet been demonstrated. Todd believed that paralysis following a fit was due to exhaustion of the brain from excessive action; this view has been accepted by Hughlings Jackson and others. Clark, who has had abundant opportunities of studying this subject, says: "It is not necessary to invoke any other state than exhaustion to explain the temporary paralysis in epilepsy."

In the preceding case the persistence of paralysis probably depended on the rapidity with which the fits followed one on another. Negative pathologic findings would be additional evidence in favor of exhaustion, as the probable cause of paralysis.

1710 Locust Street.

CANNABIN TANNATE (MERCK).*

S. A. MATTHEWS, M.D.
CHICAGO.

In response to a request of the Secretary of the Council on Pharmacy and Chemistry, I have recently investigated the preparation listed by Merck & Co. as cannabin tannate.

The name of this preparation would naturally lead one to suppose that it is a chemical union (salt) of tannic acid and some basic principle contained in *Cannabis indica*. Whatever the chemical constitution of the substance may be, it evidently contains tannic acid as one of its constituents. This is indicated by the dark blue color imparted to its watery solution on the addition of a few drops of a ferric chlorid solution.

As sold by Merck it is a greenish-brown powder, slightly soluble in water, slowly soluble in alkalies, and possesses a bitter and highly astringent taste. In fact, when applied to a mucous surface it acts much like tannic acid. On account of its bitter astringent taste, tardy solubility, and astringent action on mucous membranes, it is a very unpalatable preparation. Further, its astringent action leads one to doubt whether it can be absorbed from the alimentary tract otherwise than very slowly.

The symptoms most characteristic of *cannabis indica* intoxication are as follows: first, an apparent stimulation of the psychic functions which has given rise to the more or less romantic literature on this phase of its action; second, a condition of quietness, lack of response to external surroundings and finally sleep. On account of the latter effect the drug has enjoyed a considerable reputation as a hypnotic. Dogs are said to be very susceptible to the influence of the drug, showing a decided narcotic effect. "After a primary ataxia, excitement and nausea and vomiting, the animal tends to fall into a deep and prolonged sleep during which the sensation of pain is much diminished, while the reflexes persist."

An attempt was made to see if any of the symptoms characteristic of *cannabis indica* could be elicited in dogs by the administration of the preparation under consideration.

EXPERIMENT 1.—Subject, a dog, weight 6 kg. Five grams of cannabin tannate were shaken up in 200 c.c. of water rendered alkaline by the addition of sodium carbonate (only a partial solution resulted). Forty c.c. containing one gram of the drug, were administered by means of a stomach-tube (stomach previously empty) every two hours for four doses. Fifteen minutes after the second dose the animal began to show

nausea and soon began to vomit, the vomited matter apparently containing all the drug previously administered. The two subsequent doses were promptly vomited. Aside from the nausea and vomiting, the dog showed none of the other symptoms characteristic of *cannabis indica*—no ataxia, no psychic stimulation or depression, and no tendency to sleep.

EXPERIMENT 2.—Subject, a dog, weight 8 kg. Five grams of the drug in 100 c.c. of alkaline water were administered by means of a stomach-tube (stomach previously empty). Thirty minutes afterward the animal showed nausea and began to vomit. Apparently everything was vomited up. All subsequent doses were promptly vomited. In this experiment the animal showed no other symptoms characteristic of *cannabis indica* intoxication—no ataxia, no mental depression and no sleep.

EXPERIMENT 3.—This experiment was carried out on myself as a subject and consisted in the taking by mouth of four doses, each one gram, every two hours. Aside from a very astringent feeling extending from mouth to stomach and some nausea, no other symptoms became manifest. The discomfort in the stomach seemed to be due, in part at least, to the astringent action of the tannic acid.

By way of comment on the results of these experiments, it might be noted in the first place that the only symptom in any way characteristic of *cannabis indica* poisoning exhibited by the dogs was the nausea and vomiting. Judging from the vomited matter one would be led to think that very little of the drug was absorbed from the alimentary tract. This is what might be expected. Tannic acid alone administered by mouth imparts a like astringent feeling in the stomach and intestines and may result in vomiting and purging. The latter actually occurred in dog 2. If experiments on dogs prove anything at all as to the action of *cannabis indica*, this preparation exerts no demonstrable hypnotic action, and in man no psychic derangement, either in the form of a stimulation or depression.

Hence cannabin tannate as a therapeutic agent not only exhibits no action similar to that of *cannabis indica*, but is practically inert. Even if active in such a form, the preparation possesses so many undesirable physical properties as compared with other preparations of *cannabis indica* as to render it therapeutically superfluous.

CASE REQUIRING IMMEDIATE NEPHRECTOMY.

C. W. ROBERTS, M.D.
DOUGLAS, GA.

The following case requiring immediate operation came under my observation June 28, 1908:

Patient.—J. M. C., aged 46, farmer, weight, 165 pounds, height 5 feet 7 inches.

History.—The patient was born in Georgia, reared on a farm and has always been in good health, being noted as one of the strongest men of the county. About twelve years ago, while he was walking through the woods with an ax on his shoulder, a falling limb from a tree under which he was passing struck the ax driving it forcibly against the right occipital region, producing a fissure fracture of the vault, accompanied by considerable concussion of the brain. After some weeks' confinement to the house, during which time the patient gradually recovered from what at first appeared to be a very serious brain injury, he again assumed the duties of his farm, having made a complete recovery. Operative measures were not indicated, the expectant plan having been pursued. The patient's health at the time of the injury was good. He had been well for many years, except for a slight disturbance of digestion.

Present Injury.—About 5 p. m., June 28, 1908, the patient was riding a bare-backed horse at full speed down a hard lane,

*From the Laboratory of Experimental Therapeutics, University of Chicago.