CONCLUSIONS

1. Surgically considered, the operation of cutting the anterolateral column without doing serious damage to the rest of the spinal cord is perfectly feasible and not difficult.

2. Therapeutically considered, the almost complete freedom from pain⁷ produced by this surgical intervention met the indications presented in the case here reported, and in many other sufferers a similar intervention, I feel sure, will give similar relief.

3. Physiologically considered, section (complete?) of the anterolateral column produces (a) loss of pain sense on the opposite side of the body; (b) a disturbance in thermal sense which suggests that the fibers for heat and cold are disassociated in the cord; (c) slight disturbance of deep pressure sense and slightest disturbance in touch sense, both being impaired without producing any disturbance of sense of position.

48 West Seventy-Fourth Street.

SPASTIC PARAPLEGIA WITH THE CUTANEOUS REFLEX OF DEFENSE OCCURRING IN POTT'S DISEASE

EDWARD MURRAY AUER, M.D.

Voluntary Assistant in Neurology in the Hospital of the University of Michigan
ANN ARBOR

Spastic paraplegia is resultant on any impairment of conductivity of the chief motor tracts in the spinal cord. This impairment of conductivity may be due either to intramedullary changes, as, for example, primary degeneration of the pyramidal tracts, or to extramedullary causes, such as a tumor pressing on the spinal cord. These conditions and their treatment are so different that an early diagnosis is essential for effective treatment and prognosis.

In 1899, Babinski¹ described a form of spastic paraplegia due to an organic lesion but without degeneration of the pyramidal tract. The chief characteristic of this form of spastic paraplegia was a contracture of the limbs in flexion, the tendon reflexes being usually not exaggerated and often diminished, while the cutaneous reflexes, on the other hand, were so markedly increased that irritation of the skin on the paralyzed side would cause defensive movements of rapid withdrawal in flexion of the limb. Again, in 1911, Babinski² called attention to the difference between this condition of spastic paraplegia, with contraction in flexion and continuous exaggeration of the cutaneous reflex of defense, and the usual type, with contraction of the limb in extension and continuous exaggeration of the tendon reflexes.

A case with these clinical symptoms was reported by Claude³ which, at necropsy, proved to be a case of sarcomatous meningitis. Another case, reported by Souques,⁴ was diagnosed multiple sclerosis; there was no necropsy. Gelma⁵ reports a similar case of contracture in flexion and exaggerated cutaneous reflex of defense. The occurrence of this cutaneous reflex of defense in

Friedreich's ataxia was noted by Babinski, Vincent and Jarkowski.⁶ Lian and Rolland⁷ reported a case of spastic paraplegia of "the cutaneous reflex type of Babinski" occurring in Pott's disease. Sicard and Gutmann⁸ described such a case, with operation and cure. One must not overlook the fact that paralysis in Pott's disease is not always accompanied by demonstrable changes in the vertebrae, for not infrequently there is a paraplegia in Pott's disease without deformity of the spine caused by the pressure on the cord of an extradural exudate. According to Cadwalader,⁹ this occurs in 73 per cent. of cases.

I have to thank Dr. Carl D. Camp for permission to report the following two cases seen in his service in the hospital of the University of Michigan.

CASE 1.—*History*.—W. S., a farmer, aged 28, entered the hospital complaining of great difficulty in walking and of pain, tremor and stiffness in the lower limbs. His family history and previous medical history were negative. The patient had had the usual children's diseases. He denied venereal disease. In January, 1911, the patient was struck by a log and thrown to the ground; the log rolled completely over the right side and pinched his head quite badly. Except for a few severe bruises he had no ensuing symptoms at the time of his injury. In February, 1912, the patient fell on the ice, striking his side and back. During the month of March he had occasional sudden, sharp pains in his side and back. In May, the patient first noticed a "lump on his spine" and about the second week in July his legs began to feel weak and numb and the muscles twitched. His limbs gradually became weaker and stiff until at the time of his admission he could barely walk and complained that his limbs felt numb and cold.

Examination.—The physical examination of the patient was negative except for a marked deformity about the third, fourth and fifth thoracic vertebrae. The urine and blood examinations were negative. The neural examination of the head and upper extremities was negative. There was no atrophy or deformity of either lower limb. Sense of pain, touch, temperature, motion and position was normal in both limbs. The umbilical reflex was not obtained and the cremasteric reflex was absent on the left side. The knee-jerk was slightly increased on the left side and very markedly so on the right. The Achilles reflex was increased on the left side; there was ankle-clonus on the right side. Plantar irritation caused extension of the great toe on the right side and flexion on the left. The cutaneous reflex of defense activated by a pin-prick was very forceful on the right side; there was none obtained on the left side. There was no paralysis of the limbs. All movements were possible but were weak and slow, more especially on the right side. There was pain on jarring the spine and on pressure over the deformity. The spinal fluid showed a slightly increased albumin content but was otherwise negative.

Treatment.—August 25, the patient was put to bed and a Buck's extension was applied to both limbs. August 27, the knee-jerk on the left side was about normal; that on the right side was still increased. The Achilles jerk on the right side was still exaggerated but there was no ankle-clonus. The Babinski reflex was still present on plantar irritation of the right foot. There was no cutaneous reflex of defense obtained.

CASE 2.—*History*.—J. L., an iron-molder, aged 38, was brought to the University Hospital, Aug. 20, 1912, because of paralysis of both lower limbs. His family history was negative. He had had the usual children's diseases and had malarial fever in 1902. In 1907, his left kidney was removed; the cause for its removal was unknown. He gave no history of trauma and denied venereal disease. The present trouble began in August, 1911, when the patient was taken with severe pains across the lower part of his back. Shortly after that he noticed that his right leg was becoming weak and there was a feeling of numbness down the front side. In

7. All pains in extremity had ceased. Whether a higher section would have abolished the pains that developed at a higher level along the spine, further experience must demonstrate.

1. Babinski: Bull. et mém. Soc. méd. d. hôp. de Paris, 1899, p. 342.

2. Babinski: Rev. neurol., Jan. 12, 1911.

3. Claude: Rev. neurol., Feb. 2, 1911, p. 249.

4. Souques: Rev. neurol., March 2, 1911, p. 376.

5. Gelma: Rev. franç. de méd. et de chir., 1912, No. 5.

6. Babinski, Vincent and Jarkowski: Rev. neurol., 1912, xl, 463.

7. Lian and Rolland: Rev. neurol., June 30, 1912, p. 843.

8. Sicard and Gutmann: Rev. neurol., 1912, lli, 833.

9. Cadwalader: Am. Jour. Med. Sc., April, 1911.

September, he noticed the same condition in the left leg. This feeling of weakness and stiffness, more marked in the right leg, gradually increased until the patient could walk only with the aid of a crutch and cane. All during this time he had sharp pains down both limbs. In February, 1912, the right leg gave out entirely and he could barely use his left leg because of its weakness and the severity of the pain in it. Since February, 1912, he has been confined to his bed. He had retention of urine and now has to be catheterized. He is constipated.

Examination.—The patient was greatly emaciated. The examination of the thorax revealed nothing abnormal. There was a slight deformity of the spine at about the fourth lumbar vertebra and in the sacral region. He had large bed-sores over the tuberosity of both ischii. Examination of the urine showed a few white cells and a few granular casts but was otherwise negative. Blood examination showed 3,580,000 red blood-corpuscles, 8,000 white blood-corpuscles and 65 per cent. hemoglobin. Blood-pressure was 190.

The following observations were made by Dr. Camp: "Neural examination of the head and upper extremities is negative. Both lower limbs are greatly atrophied, the right thigh measuring 11 and the left 11½ inches in circumference.

Diagram showing areas of anesthesia. Upper heavy line, upper limit of anesthesia to heat; lower heavy line, upper limit of anesthesia to cold; dotted line, upper limit of analgesia.

The calves of both legs measure 8 inches. Both limbs are slightly flexed at the thigh and at the knee. Except for a slight flexion and adduction of the thigh, the limbs are completely paralyzed. There is complete loss of sensation in the left leg and on the left side of the abdomen extending up to about 2 inches below the umbilicus and on the back to about the level of the fourth lumbar vertebra (see illustration). Both sides of the scrotum are anesthetic to all forms of stimuli. The line of anesthesia does not exactly correspond to the upper limit of analgesia or thermesthesia. The umbilical reflex is not obtained. The cremasteric reflex is present on the right side, absent on the left. The cutaneous reflex of defense is obtained in both limbs but it is more intense in the left. The right knee-jerk is greatly diminished and the left is absent. The Achilles jerk is not obtained on either side. Plantar irritation causes no movement in either foot. To the left of the coccyx there is an area of preserved sensation to all forms of stimuli. The anal reflex is present. The patient complains of severe pain in the left limb and of slight pain in the right. He has retention of urine and his bowels are constipated. The spinal fluid had a slightly yellow color and coagulated spontaneously. There was no increase in the cell content.

Treatment and Course.—Operation was advised and a laminectomy was performed August 29. The skin incision, made in the median line, extended from 1 inch below to 3 inches above the line of anesthesia. The laminae of the third fourth and fifth lumbar vertebrae were removed. Between the fourth and fifth vertebrae there was, slightly to the left side, a mass of hard, yellowish tissue about one-half inch in diameter which, on microscopic examination, proved to be a dense mass of fibrous connective tissue, either fibroma or granulation tissue, probably the latter. This was removed and the canal was found apparently free above and below. The dura was opened and the spinal fluid appeared clear. The cauda equina was seen and seemed small but presented nothing abnormal. The wound was closed without drainage.

August 30: Examination shows practically the same findings as before the operation but no cutaneous reflex of defense reaction is obtained in the left leg.

September 2: The patient can contract the left calf muscles and the left posterior thigh muscles and adduct the thigh. He complains of pain shooting down the left leg. He can slightly extend the left leg but has no other movements in that leg. Tapping the left patellar tendon causes contraction of the left calf muscles and adductors of the thigh on the left side but no extension of the knee. The right knee-jerk is normal or perhaps slightly diminished. The left and right Achilles jerks are normal. Plantar irritation on the right side causes no movement; on the left side there is no movement of the toes but a drawing up of the left leg. Sensation to pain is absent in the left leg but the upper limit of the analgesia is lowered to the groin, and the scrotum on both sides is sensitive to pin-point.

September 10: The patient has pains in the left leg which are nearly as bad as before the operation. There is a catheter in the bladder. He has incontinence of feces and thinks he can move his legs a little better than before the operation. The abdomen being bandaged, the umbilical reflex cannot be taken. There is considerable wasting of both legs above and below the knee, a bilateral foot-drop and some swelling of the feet. Tapping the left knee gives rise to contraction of the flexors of the left knee and the left calf muscles. Tapping the right knee causes a normal knee-jerk. The Achilles reflex is present, equal and normal on both sides. Plantar irritation on the right side causes no movement of the toes, but the patient has a painful sensation; there is no pain on the left side. Deep pressure on the calf muscles on both sides is painful. There is complete loss of pain and tactile sensibility over the left leg except that deep pin-prick on the inner side of the left calf is sometimes felt, though delayed. The patient says that he feels touch and pin-point normally in the right leg. He has normal sense of motion and position of the right foot; this is lost in the left. The upper limit of anesthesia does not extend quite as high as before. The genital organs have normal sensation to pin-point.

The patient left the hospital September 10.

These two cases and those reported by other observers show that the spastic paraplegia accompanied by the cutaneous reflex of defense is often due to a gradually progressing compression myelitis. This phenomenon also occurs in Friedreich's ataxia and in multiple sclerosis; in these diseases we find areas of sclerosis which may involve the pyramidal tract, but the axis cylinder of the nerve fiber may escape destruction and the secondary degeneration be comparatively slight.

In Pott's disease there is an extradural pressure on the spinal cord which may or may not cause a secondary degeneration of the pyramidal tract. If it does not, then the results of relief of the pressure should be a restoration of function.

It is therefore highly important to be able to distinguish clinically between a paraplegia due to degenerative changes in the pyramidal tract and one caused by pressure not producing marked secondary degenerative changes. In the former case treatment is

of little value; in the latter the prompt removal of the compression by surgical or other measures may be followed by a great improvement. If this differentiation of the two types of spastic paraplegia assists us in recognizing the case in which an operation may be attended with good results, it is of great value. Not only is the cutaneous reflex of defense occurring in spastic paraplegia with contraction in flexion one of the earliest signs found, but it may also be present with complete loss of sensation to touch, pain and temperature, abolished reflexes and paralysis of the affected parts. A definite proof that it is produced by pressure is the fact that it disappears on removal of this pressure, either by palliative or by operative means.

In conclusion, I wish to say that this differentiation of the spastic paraplegias can not fail to be of great value in the early recognition of a condition in which early treatment offers gratifying results and in which the usually delayed diagnosis produces such disastrous effects.

1000 East Ann Street.

ONE OF THE POSSIBLE FACTORS IN THE CAUSATION OF PELLAGRA *

ALLAN H. JENNINGS

AND

W. V. KING

WASHINGTON, D. C.

Among the various phases of the problem presented by pellagra, of its symptomatology alone can we be said to have even moderately full knowledge. Its epidemiologic features and geographic distribution are as yet but imperfectly known, while in spite of the labors of many investigators whose researches have been directed toward the discovery of its etiology, the knowledge thus far gained leaves that important factor still largely within the realm of theory.

Many hypotheses of causation, based on certain aspects of the disease or its environment, have been advanced, but none of these, perhaps, quite accounts for all the phenomena involved.

It is not our purpose to discuss these theories in detail or to attempt to show the truth of any one of them, far less to propound a new one, but rather to point to facts which may have a bearing on the question.

If it be admitted that pellagra is a distinct morbid entity and not a mere symptom-complex, it is evident that the essential factors in its causation and spread must be uniformly active wherever the disease is endemic. In spite of this self-evident fact, the phenomena of the disease present many puzzling and apparently contradictory facts, under the varying conditions of environment and habit of its victims. Yet somewhere among these confusing phenomena must lie the constant element which is the cause of the manifestations of pellagra.

That the disease is due to an infection is an opinion held by a considerable body of investigators, and the arguments in favor of this hypothesis seem to warrant its serious consideration. On this hypothesis and the epidemiology of the disease are based the theory that pellagra is an insect-borne disease. This theory, first formulated by Sambon, has been given prominence dur-

ing recent years and has received the concurrence of able students of the disease.

If the disease is due to an insect-borne infection, a consideration of some entomologic facts which appear to have significance in this connection may be worth while.

As pointed out by Knab,¹ for an insect to be a potential transmitter of human disease, it is necessary that four factors should be active. These factors are the habit of biting man regularly, a more or less close association with him, a certain degree of longevity and, finally, abundance. These biologic characteristics assure the opportunity to bite man, the practical certainty that numerous members of the species will bite him, not once only, but that they will secure successive blood meals and that a considerable number will survive beyond the developmental period of the hypothetical organism involved and again inflict bites.

The qualifications mentioned may vary in their relation to each other, as when great abundance and the persistence of the parasite in the blood of the vertebrate host compensate for a less close association with man, which occurs among the mosquito transmitters of malaria.

Apparent exceptions occur in some of the ticks, which are not usually domestic in any sense, yet convey certain diseases of man. But the power of this group to transmit the germs of disease through their progeny, together with their longevity and the duration of the life-cycle, extend the possible period of infectivity to at least several years, in the case of some species.

The phenomena of pellagrous incidence, as exhibited in America, differ in some important respects from those seen in some parts of Europe, according to authoritative reports.

Sex incidence in the Western hemisphere shows a marked preponderance of female cases.² In some localities these cases bear a relation apparently of more than three females to one male, but always in marked preponderance. This seems not to be true of Europe, where the cases among females, although somewhat more numerous than those of males, are in much smaller preponderance than in America.³ There may be several reasons for this difference in the proportion of the two classes of cases in these countries, but the most obvious is that of habit and occupation. The peasant woman of Europe shares the work of the men of her family in much greater degree than is the case among the women of any class in this country. Whether at work in the fields or about her home, the peasant woman is exposed in much more nearly equal degree to the same conditions and liability to infection, whatever be its source. How this may influence the occurrence of the disease in the two sexes will be referred to later.

According to published statements, the class of the population affected with pellagra is much more sharply defined in Europe than in America. If we may believe these statements, the disease is confined almost exclusively to the strictly rural population and to those in the most abject circumstances in life. Its occurrence among the well-to-do is the exception.⁴ In the United States

1. Knab, Frederick: Unconsidered Factors in Disease Transmission by Blood-Sucking Insects, *Jour. Econ. Ent.*, 1912, v, No. 2.

2. Roberts, S. A.: Pellagra, St. Louis, 1912. Beall, K. H.: *Bull. Tex. State Board Health*, 1911, v, No. 9. *Tenn. State Board Health, Report on 316 Cases of Pellagra*, 1911. Lavinder, C. H.: Reprint from P. H. Rep. 65, 1911. Grimm, R. M.: P. H. Rep., 1911, xxvi, No. 38.

3. Roberts, S. A.: Pellagra, St. Louis, 1912. Babes and Sion: *Die Haulersschien der Pell.*, Innsbruck, 1909.

4. Marie, A.: Pellagra, Transl. by Lavinder and Babcock, The State Co., Columbia, S. C., 1910.

* From the Bureau of Entomology, U. S. Department of Agriculture.

* Paper read at the second triennial meeting of the National Association for the Study of Pellagra, Columbia, S. C., Oct. 3 and 4, 1912.