Femoral Neuropathy in Relation to Diabetes Mellitus

Report of 17 Cases

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Neurologic lesions have been described in all parts of the nervous system in cases of diabetes mellitus but these abnormalities prove to be predominantly peripheral neuropathy or neuronopathy.[1] While almost any peripheral nerve such as the sciatic, ulnar or median may be involved in diabetic neuropathy, the femoral nerve appears to be affected most frequently in our experience, yet femoral neuropathy is scarcely mentioned in the literature. One reason for the apparent infrequency of this neuropathy may be a lack of familiarity with the condition and consequent failure to suspect its presence.

CLINICAL SYNDROME OF FEMORAL NEUROPATHY

This report deals with the syndrome of femoral neuropathy which was diagnosed in 17 cases, in 16 of which diabetes mellitus was present. There were three outstanding manifestations among these patients: pain, muscular weakness and absence of the patellar reflex. In addition to this triad, a history of paresthesia is obtained frequently.

Pain is the most frequent complaint of patients with femoral neuropathy, and was present in 15 of the 17 cases in this series. The pain may be either spontaneous or provoked by the femoral nerve stretch test (see below). Usually the pain is very severe and sharp; it may be shooting or boring in type or it may have a burning or aching quality. The pain extends from the hip along the anterior and lateral surface of the thigh into the foot, but sometimes it begins in the sacro-iliac region and radiates down the posterior surface of the thigh and leg to the dorsum of the foot. In some cases, pain may develop gradually, in others episodically. In most cases, the pain is worse at night and interferes with sleep. It may become so severe and intractable that opiates are required, and because of failing appetite, there may be loss of weight.

To confirm the diagnosis, the femoral nerve stretch test is employed. This was first called to my attention by a colleague, Dr. Sigmund Wassermann, who originally described the maneuver in nondiabetic patients.[2] The technic of the test is as follows: The patient is instructed to lie prone. By elevating the straight leg off the table, or by flexing the knee without elevation of the thigh, the femoral nerve is placed under stretch. When the test is positive, the patient complains of exquisite pain along the anterior thigh. This sign, which is actually the Lasègue sign in reverse, was positive in 9 of the 17 cases in this series. Incidentally, the Lasègue sign was negative in all of these patients, thus ruling out the possibility of sciatic neuropathy. Occasionally, it is possible to elicit pain by direct pressure on the femoral nerve, which is located laterally to the femoral artery. This sign was positive in four cases.

Muscle weakness and/or atrophy was observed in 11 cases. In the early stage of femoral neuropathy, the patient may complain of clumsiness in walking, principally in going downstairs. Later, the leg or knee may buckle and cause him to fall without warning. In order to avoid falls, some patients resort to the use of a cane. In the more advanced stages, the affected leg may become so weak that the patient is forced to remain in bed. Although the motor weakness in this condition is primarily subjective, occasionally moderate atrophy of the quadriceps muscles, the calf or even the buttocks may be demonstrable.

Absence of the patellar reflex was noted in 12 cases. In view of the intimate relationship to the function of the femoral nerve, this is not unexpected. In testing this reflex it is preferable to have the patient sit on a chair or stool, both feet planted firmly on the floor. In a normal response, contraction of the quadriceps femoris muscle is readily felt upon strik-

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ing the patellar tendon with a hammer.

In the face of the high incidence of absent achilles reflexes among diabetic patients,[1] I believe this finding has no special significance in patients with femoral neuropathy.

Paresthesia was described by 9 patients. Paresthesia is a common symptom in many peripheral neuropathies, and femoral neuropathy is no exception in this respect. The area of distribution of the paresthesia is identical with that of the pain, namely, the anterior and lateral surfaces of the thigh radiating down the leg into the foot. Numbness is a common complaint of most patients, but in some the paresthesia has a tingling quality and produces a sensation in the feet akin to that produced by electricity; in others the paresthesia has a sharp, burning character. Hyperesthesia may be a predominant complaint, and it may even be aggravated by such a slight stimulus as the contact with the bedclothes or pajamas. It is worthy of mention that the paresthesia of femoral neuropathy does not disappear as rapidly as the pain. For example, in case 16 (a physician) a feeling of coldness and tingling in the right foot persisted for five months after the severe pain had subsided, and was unaffected by the administration of vitamin B_{12}; four months after the paresthesias finally disappeared, they recurred for a brief period during a mild attack of influenza.

Elevation of the cerebrospinal fluid protein was found in three cases. The values were 97 to 158 mg. per 100 cc. In one case, the Pandy and gum mastic tests were positive. However, the spinal fluid was not examined routinely. Elevated proteins are consistent with severe peripheral neuropathy or neuronopathy.[1]

Concomitant neurologic manifestations were observed in 5 cases. The widespread nature of the neurologic disorder in diabetic patients with femoral neuropathy was shown by the involvement of other peripheral, cranial and autonomic nerves in these cases (cases 1, 4, 7, 14 and 16). Sometimes, the peripheral nerves in the shoulders and upper extremities were involved. The trigeminal and ocular were the only cranial nerves affected in this group. Autonomic nervous system disturbances were manifest in only one case (case 7); here there was diarrhea, gastric retention and absence of sweating.

Absence of the achilles reflex was noted in 8 cases. The significance of this finding in eight patients with femoral neuropathy has already been mentioned.

Objective sensory findings are infrequent in patients with femoral neuropathy. Slight hypesthesia in the distribution of the femoral nerve was noted in three cases. Position sense is usually unimpaired in this condition. Because diminished vibratory sensation is such a frequent finding in diabetic patients as a whole, its impairment in four patients with femoral neuropathy in the present series must be considered coincidental. It may be stated in passing that the diagnostic import of this finding, especially in older diabetic patients, is greatly depreciated by the high incidence in older nondiabetic individuals.[1]

Disturbances of the autonomic nervous system are not prominent in association with femoral neuropathy. A sweating test may be employed, since loss of the sweating response is an indication of degeneration of sympathetic fibres somewhere along their course in the peripheral nerves. While sweating tests were not performed routinely, in one case loss of sweating was demonstrated below the knees and in the palms of the hands. In this case, chronic diarrhea and gastric retention were further evidence of autonomic dysfunction.

RELATION TO DIABETIC STATUS

Sixteen of the 17 patients with femoral neuropathy were diabetic and only one nondiabetic. Thirteen patients had lack of control by the accepted clinical criteria, since polyuria, polyphagia, weight loss, weakness and fatigability were present. In the majority of cases, marked glycosuria was present. Hepatomegaly, another indication of poor diabetic control in our experience,[3] was found in 7 of the 17 cases. A close relationship between femoral neuropathy and poor diabetic control can be inferred from these findings.

DIFFERENTIAL DIAGNOSIS

Femoral neuropathy is a true peripheral neuropathy or neuronopathy. Herniated intervertebral disc had been suspected in several cases. The consideration of "diabetic tabes" in some cases is not surprising. Actually, there are several features of femoral neuropathy suggestive of tabes, namely, severe lancinating pain, ataxia, areflexia and, in some cases, cerebrospinal fluid changes. In my opinion, femoral neuropathy may be the underlying basis in many cases designated as diabetic "tabes" or "pseudotabes."

COURSE

With control of the diabetes by diet and insulin, the neurologic complaints of patients with femoral neuropathy usually disappeared within three months. Whereas the severe pain subsided soon after regulation of the diabetes, the residual paresthesia receded more slowly

as stated above; for example, in case 16, in which there was extremely severe pain, it practically disappeared three days after insulin administration, whereas the paresthesias, and a feeling of coldness in the foot of the affected side persisted five months longer. In every case, the pain subsided within two weeks after diabetic treatment had been instituted. Improvement of motor function usually began a few days after diabetic therapy was started, and normal muscle strength was regained rapidly, generally within three months. Recovery of the patellar tendon reflex depends upon the severity of the nerve damage. In one case, the knee jerks returned to normal within six weeks after instituting diabetic treatment. With neglect of treatment and consequent deterioration of the diabetic control, a tendency to recurrence of the neuropathy may be noted (see case 17).

TREATMENT

In my experience, the only effective treatment of diabetic neuropathies, including femoral neuropathy, is adequate management of the diabetes. A nutritious diet and a dosage of insulin adjusted to control glycosuria and hyperglycemia are essential.

In these cases, many different therapies for the relief of pain had previously been attempted elsewhere. Lumbar sympathectomy failed to alleviate the pain in two cases; in one of these a cordotomy was finally performed. Although the pain was relieved by this procedure, the patient unfortunately developed paraplegia after the operation and died from an ascending urinary tract infection. Other measures, designed to relieve pain by improving the peripheral blood flow, were uniformly unsuccessful; these included priscoline, nicotinic acid and, in one case, sodium chloride. The numerous vitamin preparations, particularly thiamin chloride and vitamin B_{12}, which have been strongly advocated by some authors, were consistently ineffectual. Of interest in this regard is case 16, in which thiamin chloride had been used daily for several years, during the time the neuropathy developed; the severe pain was alleviated promptly after treatment with insulin was begun, but paresthesias continued in spite of treatment with a 12-day course of Vitamin B_{12} in 50 μg. doses. In case 14 the administration of procaine, 2 per cent intravenously, failed to relieve the pain. Following two injections of BAL (25 mg. and 50 mg. on successive days) there was a slight diminution of the extreme hyperesthesia which, except for moderate dysesthesia in the fingers, disappeared entirely following the next two doses (100 mg.). In all other cases, after diabetic management was instituted, improvement was so prompt that no other therapy was even considered.

CASE REPORTS

Case 1. A 55-year-old male was admitted to hospital July 31, 1950, because of severe pain in the legs of several years duration, loss of weight from 190 lb. in 1945 to 118 lb., and symptoms of an acute urinary tract infection.

He had had diabetes mellitus for four years but had neglected treatment. In March 1950, very severe, boring pain developed, extending from the hips to the feet. After this, both legs became so weak that he was unable to lift them off the ground to take a step. The diagnosis was diabetic neuropathy.

He entered another hospital in May 1950. A left lumbar sympathectomy was performed without relief of pain, so that on June 19, 1950, a cordotomy was done. Although this relieved his pain, he was unable to walk again and an automatic bladder developed. The examination showed that the achilles and patellar reflexes were absent in both lower extremities. The position sense was normal. A positive Babinski sign was present on the right side and the abdominal reflexes were absent bilaterally. With the exception of elevated proteins, the cerebrospinal fluid was normal. In the opinion of the neurologic consultant, the weakness of the lower extremities and the cord bladder were sequels of the cordotomy operation. In the early stages of the neuropathy the patient had experienced paresthesias in the right hand. The course was steadily downhill and he died Oct. 16, 1950, as a result of generalized peritonitis secondary to a ruptured abscess of the prostate.

Case 2. A 54-year-old man was admitted to hospital March 17, 1951. A diabetic glucose tolerance curve had been obtained during a previous admission. The diagnosis of hemochromatosis was suspected on a second admission in September 1949. After discharge he began to notice increasing fatigue, especially during the day, nocturia (6-8 times), polyuria (every hour), and his urine was sticky. He drank a quart of milk, a large quantity of fruit juice and 6 to 8 glasses of water daily, and dropped in weight from 165 to 148 lb. in less than six months. In November 1949, he noted intermittent numbness over the lateral aspect of the right leg which occasionally had a sharp, burning character. In February 1950, his leg buckled and he fell down. On this admission both knee jerks were absent for the first time, but there were no sensory abnormalities. There were more than 100 gm. of glucose in the 24-hour urine.

During the next three months, with control of the diabetes by diet and insulin, the neurologic complaints disappeared completely. When he was readmitted in March 1951, the patellar reflexes had returned to normal.

Case 3. A 63-year-old white male was admitted to Crile (Veterans Administration) Hospital April 10, 1951. A diagnosis of diabetes was made in 1944, but he remained aglycosuric on diet alone until September 1949, when he was placed on insulin in another hospital. In October 1949, he was transferred to Crile Hospital for a transurethral resection. During three months in the hospital, the diabetes was poorly controlled. Four months after discharge, March 1950, intractable, burning pain developed in the soles of both feet, and he re-entered Crile Hospital for these complaints. Vibratory sensation was diminished in both soles, and both achilles reflexes were absent. The other tendon reflexes were present. A diagnosis of "peripheral neuritis" was made. While in the hospital, the burning pain frequently became so severe as to necessitate the administration of papaverine or demerol; the pain was not relieved by priscoline or nicotinic acid. After discharge a bilateral sympathectomy was done at another hospital without relief of pain. Postoperatively there was further weight loss leading to malnutrition, and the patient complained bitterly of pain along the anterior thighs extending into the feet. All the tendon reflexes were unobtainable and vibratory sensation was diminished in the feet and legs. The peripheral circulation was unimpaired. The diagnoses were (1) femoral neuropathy, (2) uncontrolled diabetes, and (3) malnutrition.

Case 4. A 53-year-old colored male was admitted to Crile (Veterans Administration) Hospital March 20, 1951, for the fifth time. He had been a known diabetic since 1931, and had taken insulin from 1940 until he was no longer financially able to purchase it. Soon this was followed by polydipsia, polyuria and nocturia, and he was admitted to Crile Hospital for the first time in March 1946. The right knee jerk was absent and the right ankle jerk diminished. He was discharged on a diet and insulin. He was readmitted Feb. 24, 1947, complaining of a peculiar sensation in the left side of the face and left arm "as though he were going to have a stroke." There was hypesthesia to pinprick in these areas. The fractional urines showed the diabetes to be poorly regulated. During the interval before the next admission, he did not follow his diet but apparently got along fairly well until January 1949. At this time there was stiffness of the left hip, of such severity that he had to stop work, and shooting pains and paresthesias in the right foot unrelated to exercise. There was marked weakness in the right leg which greatly interfered with walking. The right knee and ankle jerks were diminished compared with the left, which were quite lively. The diabetes was controlled by diet alone. On the next admission, Dec. 5, 1950, though moderate glycosuria was present, he had regained 12 lb. The right achilles and patellar reflexes could not be obtained. He became aglycosuric on a reduction diet. On the succeeding admission, March 6, 1951, he complained of coldness over the lateral aspect of the right thigh, right leg and foot, and over the lower half of the left leg and foot. In addition to the previously recorded absent patellar and achilles reflexes, there was impaired vibratory sensation over the entire right lower extremity, and hypesthesia to touch and pinprick over the right lower leg and foot. Position sense was normal. There was localized tenderness over the right femoral nerve and exquisite pain on stretching the femoral nerve by flexion of the knee. The diagnoses were (1) diabetes mellitus with right femoral neuropathy, (2) obesity, and (3) proximal and peripheral atherosclerosis.

Case 5. A 63-year-old white male was admitted to Crile Hospital Oct. 13, 1949. He complained of sharp, burning and tingling pain, of 10 to 12 years' duration, over the anterior and lateral surface of both thighs, radiating down the legs and producing an electricity-like sensation in the feet. The legs became so weak that he was forced to walk with a cane. A diagnosis of neuropathy had been made on a previous admission (October 1947). He had been unable to work since 1943 on account of the leg difficulty. The principal physical findings were an enlarged liver and the neurologic manifestations. The latter consisted of tenderness to pressure over the anterior surface of the thighs, especially over both femoral nerves, more marked on the right. Stretching the femoral nerves elicited pain, and there was moderate tenderness over the achilles tendon and moderate weakness of the leg muscles. There were no other objective neurologic findings. The diagnoses were (1) obesity, (2) marked dietary inadequacy with enlarged, fatty liver, (3) peripheral, mainly femoral, neuropathy, and (4) proximal atherosclerosis.

Case 6. A 55-year-old white male was admitted to Crile Hospital Jan. 9, 1951. A diagnosis of diabetes was made at another Veterans Administration Hospital in 1946. At that time he developed polyuria, polydipsia, polyphagia and loss in weight from 145-150 lb. to 112 lb. He refused to take insulin and merely curtailed the sugar-content of the diet. In November 1950, he gradually

developed pain beginning in the right sacro-iliac region and radiating down the posterior surface of the right thigh and leg to the dorsum of the foot. The pain was episodic and kept him awake at night; it was relieved by exercise and heat. After weakness of the leg appeared, he consulted a private medical clinic where glycosuria, hyperglycemia and acetonuria were found. On admission to Crile Hospital he had four-plus glycosuria, one-plus acetonuria and liver enlargement. There was wasting of the muscles of the buttocks, and he complained of pain along the posterior aspect of the right leg radiating down to the heel upon bending over. On physical examination, the Lasègue sign was negative, but with reverse extension of the thigh and flexion of the knee there was marked pain over the anterior surface of both thighs and moderate tenderness at the site of the femoral nerve in the inguinal region. Vibratory sensation was impaired in both lower extremities up to the knees. The right achilles reflex was present but the left achilles and both patellar reflexes were absent. The cerebrospinal fluid protein was 97.6 mg. Another consultant considered the possibility of a herniated disc and attributed the "neuritis" to arteriosclerosis. *Conclusion*: This patient had poorly regulated diabetes over a period of six to seven years, during which a bilateral femoral neuropathy developed. The findings cannot possibly be attributable to vascular disease in view of a normal peripheral circulation. Upon regulation of the diabetes the severe pain subsided promptly, leaving only residual paresthesias in the feet.

Case 7. A 29-year-old white male, with a history of diabetes since 1943, was admitted to Crile Hospital Oct. 25, 1950, with diarrhea. On a previous admission to Crile Hospital in 1947 for acidosis, he was discharged on a diet and insulin. In 1949 the patient had a dull, aching pain over the anterolateral aspect of both thighs. He was hospitalized at another Veterans Administration Hospital in Dec. 1949, for diarrhea. In April 1950, he complained of a burning sensation in the skin of the thighs, aggravated by contact with the bedclothes, and there was some clumsiness on walking, principally down steps. On physical examination no tendon reflexes could be elicited in the lower extremities and there was some pain on stretching the left femoral nerve. A sweating test revealed absent sweating below the knees and in the palms of the hands. The neurologic consultant reported generalized hyporeflexia. During five months of hospitalization, the diabetes was well regulated and the paresthesias, formerly present over the anterior surfaces of both thighs, disappeared. Whereas even the touch of his pajamas had been unbearable previously, now the only residual was a slight aching in the legs. Incidentally, a story was obtained of impotence, having its onset in conjunction with the diarrhea. In addition, there was marked gastric retention. When he was readmitted March 17, 1951, there was extreme dryness of the hands and feet and marked erythema of the feet which was attributed to autonomic involvement. There was generalized areflexia. *Conclusion*: Diabetic (femoral) neuropathy with neurogenic diarrhea, gastric retention and impotence.

Case 8. A 60-year-old white male was admitted to Crile (Veterans Administration) Hospital Sept. 6, 1951, with a history of onset of diabetes in 1946 (weight loss, polyuria, polydipsia and polyphagia). He was advised by a private physician to restrict the carbohydrates in the diet. In July 1951, the patient began to feel weak and easily fatigued. The glycosuria, which formerly had varied from zero to two-plus, increased to four-plus. He lost 15 lb. in weight during the next month, and this was accompanied by polyuria, polydipsia and polyphagia. On Aug. 1, 1951, the patient experienced aching pains in the legs and sharp pains in the feet. Besides, there were sharp, shooting pains along the lateral and posterior surfaces of the thighs, occurring both during the day and at night, although originally the pain had been worse at night. On physical examination, there were no significant findings with the exception of excruciating pain over the anterior thighs on stretching the femoral nerves. The diabetes was uncontrolled at the time of admission. Diagnoses: (1) Uncontrolled diabetes with bilateral femoral neuropathy, (2) proximal atherosclerosis, (3) senile emphysema, (4) fever of undetermined origin.

Case 9. A 72-year-old colored male was admitted to the Cuyahoga County Nursing Home for the second time Jan. 9, 1951. In 1931, he was admitted to a local general hospital in impending diabetic coma and, after a short period of treatment with insulin and a diet, regained his strength and returned to work. In February 1940, he began to lose weight gradually and was hospitalized because of pains in the knees. Despite an increase in insulin dosage, the pains in the legs became excruciating and a progressive weakness in the legs virtually prevented his walking. Upon admission to the Nursing Home, shortly afterwards, he had lost 35 lb. from his optimum weight and complained bitterly of severe burning pain bilaterally extending from the right sacro-iliac region along the lateral aspect of the thigh. The pain was particularly severe at night. There was a noticeable

weakness of the right leg with some atrophy (½ in. at mid-calf and mid-thigh). There was diminished sensation to pinprick beginning in the right sacro-iliac region and extending down along the leg and foot. Vibratory sensation was diminished in both lower extremities and the achilles and patellar reflexes were absent bilaterally. The provisional diagnoses were: (1) diabetes mellitus, (2) generalized arteriosclerosis, and (3) probable diabetes tabes. The cerebrospinal fluid protein was elevated to 158 mg. and there was a positive Pandy and a gum mastic of 2443210000. Prior to admission to the Nursing Home, the patient had been given large doses of thiamin chloride and sodium chloride for relief of pain. Four months after treatment of the diabetes with diet, insulin and a course of thiamin chloride, 50 mg. intravenously, he was able to move the right leg somewhat better and the pain and burning were less severe than before. He had gained weight and appeared much better.

Case 10. A 68-year-old white male, a private patient, was examined Feb. 18, 1950. In 1949 he had developed an increased appetite with a great desire to eat "sweets," polydipsia, nocturia and a moderate weight loss. He consulted a physician, who prescribed a diet in which sugar and starches were omitted. One month before consulting me, an aching pain developed over the anterior surface of his left thigh, and this was accompanied by a less severe pain over the lower spine and weakness of the left leg, which gave way occasionally. The patient was underweight and presented hepatomegaly. The left patellar reflex was absent, the right patellar and both achilles reflexes normal. There was marked tenderness and pain upon acute flexion of the knee (Wassermann's sign). Diagnoses: (1) diabetes mellitus, poorly regulated, with femoral neuropathy, (2) diabetic retinopathy, (3) proximal atherosclerosis, (4) hypertrophic arthritis, (5) underweight.

Case 11. A 61-year-old white male was examined as a private patient Nov. 7, 1952. He gave a history of polydipsia, polyuria with nocturia (times two to three), and a 10 lb. weight loss over the preceding two years. In recent months he had experienced pain and paresthesia over the anterior surface of both thighs. Five weeks previously he was hospitalized because of a rectal abscess and found to be in keto-acidosis. Although all the reflexes were obtained, pain was elicited bilaterally over the anterior thighs by means of the reverse Lasègue sign. Diagnoses: (1) diabetes mellitus with femoral neuropathy, (2) senile emphysema, and (3) peripheral atherosclerosis.

Case 12. A 48-year-old pharmacist was examined Aug. 21, 1952, complaining of a "limping" feeling over the anterior and outer surface of the left thigh. The diabetes had been discovered eight years previously in the course of a life insurance examination. He was given a diet which he did not follow. In 1949 he consulted me for the first time because of nocturia, frequency and polydipsia. Although he reduced the intake of starches voluntarily, nevertheless 15 gm. of glucose were excreted in the 24-hour urine. A moderate glycosuria persisted until the most recent examination, when 90 gm. of glucose were found in the 24-hour urine. Diagnoses: (1) diabetes, obese type, with femoral neuropathy, (2) varicose veins.

Case 13. A 71-year-old white male was referred for examination April 5, 1951. Five years previously (1946) glycosuria had been discovered during a routine physical examination and he was placed on a diet by the examining physician. While on this diet, his weight dropped from 190 lb. to his present weight of 164 lb. Two weeks previously an aching pain developed in the left leg which was much worse at night. Later, his son noted that his knee buckled frequently. Two days before consulting me, a herpetic eruption appeared over the left anterior thigh. The deep tendon reflexes were very lively but the left patellar reflex was absent. The blood sugar was 200 mg. per cent and a diabetic glucose tolerance test was obtained. The neuropathy gradually subsided during the next three months. When he was re-examined July 14, 1952, the left patellar reflex still could not be elicited. Diagnoses: (1) diabetes mellitus, borderline, with femoral neuropathy and herpes zoster, (2) proximal atherosclerosis, (3) benign prostatic hypertrophy and hydrocele, left.

Case 14. A 40-year-old white male, examined as a private patient March 28, 1951, had had diabetes for 19 years. On Aug. 15, 1950, he awakened with a feeling of numbness in the fingers and toes of the left side and paresthesias over the left side of the face. Two months later the right great toe was affected similarly. In the four-month period prior to my examination, the paresthesias in the hand and foot became unbearable and he complained also of impotence and loss of libido. The patient presented evidence of severe retinopathy, hepatomegaly and occlusive vascular disease. All the tendon reflexes were lively except for absence of both achilles reflexes. There was moderate tenderness over the femoral nerves with the reverse Lasègue sign, and considerable glycosuria was found in the 24-hour urine. The patient was hospitalized for study, and a peripheral vascular con-

sultant attributed the pain to neuropathy. This conclusion was confirmed by a neurologic consultant. The patient obtained no relief from the administration of vitamin B_{12}. Following two injections of 25 and 50 mg. of BAL, there was slight diminution of the extreme hyperesthesia, which disappeared entirely following two 100 mg. doses of BAL. Some dysesthesia in the fingers still remained. Shortly afterwards the patient expired suddenly and, because no autopsy permit could be obtained, the cause of death was undetermined. Diagnoses: (1) poorly regulated diabetes with retinopathy and neuropathy, (2) peripheral atherosclerosis.

Case 15. An 18-year-old white female, with recently discovered diabetes mellitus, was examined May 29, 1952. In the preceding two to three months, she experienced frequency of urination (8-10 times daily and 2-3 times at night), occasional urinary incontinence and severe thirst. Six weeks previously the left ankle became weak and turned readily; the leg felt numb up to the level of the knee. The principal physical findings were an enlarged liver and absence of both patellar reflexes. The Wassermann sign, that is, pain on stretching both femoral nerves, was positive. Two days after the administration of insulin, the urinary symptoms disappeared. The neurologic symptoms gradually subsided within one month, and six weeks afterwards the patellar reflexes could be obtained by re-enforcement.

Case 16. A 61-year-old white physician has been under observation since 1948. After a severe attack of mumps in 1926, the patient experienced recurrent attacks of cranial nerve paralyses (facial, oculomotor and trochlear). The diabetes was discovered through a routine urinalysis and had always been asymptomatic, although four-plus glycosuria had been present frequently after large meals. In September 1949, a diabetic glucose tolerance curve was obtained and all the deep tendon reflexes were normal, though hypoactive. Following a hard fall in March 1950, pain developed over the right lateral thigh. Shortly afterwards, he experienced paresthesias in the right foot as though he were walking on a carpet. In May 1950, a neuritic pain and hyperesthesia appeared in the left arm and the pain in the leg became so severe that the patient was unable to eat properly and lost 10-12 lb. in weight. On physical examination (June 7, 1950) he presented an enlarged liver and the deep tendon reflexes were absent in the left arm and right leg. There was no femoral or sciatic tenderness. The neurologic findings were confirmed by a neurologist who also found tenderness in the left arm on pressure. The 24-hour urine contained 36 gm. of glucose. Three days after the administration of insulin was begun, the pain had practically disappeared and the liver size receded to within normal limits. The paresthesias, and a feeling of coldness, persisted for a period of five months until November 1950. It is of interest that the patient had been taking thiamin chloride, 100 mg. daily, for many years, up to and including the time of development of the neuropathy. Also, the paresthesias were unaffected by a 12-day course of vitamin B_{12} in 50 μg. doses. In March 1951, the paresthesias recurred slightly during a mild bout of influenza. He was hospitalized for two weeks in April 1951, with a right trigeminal and ocular neuropathy characterized by severe pain in the eyeball and face persisting for six to seven weeks. There were accompanying eye muscle paralysis and diplopia, from which he recovered completely after two months. When re-examined Sept. 3, 1952, the patellar and achilles reflexes were absent bilaterally.

Case 17. A 60-year-old white male was seen in consultation Jan. 14, 1950, because of a dull, aching pain along the left anterior thigh and leg of three weeks' duration. The pain was severe, aching, worse at night, and there was weakness in the leg. A diagnosis of diabetes had been established in 1940 on the basis of polydipsia, weight loss, polyuria and polyphagia. Insulin was started in 1944. On examination, the patient presented hepatomegaly, marked glycosuria and weakness of the left leg. There was slight hypesthesia and tenderness of the left femoral nerve. Both patellar reflexes were greatly diminished; the achilles were normal. One week after the insulin dose was modified, the leg felt somewhat stronger and there was less pain; after two weeks of treatment the pain disappeared entirely. Three months later the strength in the leg had returned to normal, and two months afterwards both patellar reflexes could be obtained, two-plus on the right, one-plus on the left. In October 1950, there was a slight recurrent soreness and tenderness along the lateral portion of the left thigh, mainly at night. Diagnoses: (1) uncontrolled diabetes mellitus with bilateral femoral neuropathy and retinopathy, (2) severe proximal atherosclerosis, (3) nerve deafness.

SUMMARY

Seventeen cases of femoral neuropathy have been studied; in 16, diabetes was present. The condition is manifested by pain and tenderness in the distribution of the cutaneous branches of the femoral nerve, weakness of the muscles supplied by it and absence of the patellar tendon reflexes and paresthesia.

TABLE 1

Neurologic manifestations in 17 cases of femoral neuropathy

Symptoms and Signs	Number of Cases
Pain	15
Absent patellar reflex	12
Muscle weakness and/or atrophy	11
Paresthesia	9
Positive femoral nerve stretch test	9
Localized femoral nerve tenderness	4
Elevated cerebrospinal fluid protein (not tested routinely)	3

TABLE 2

Concomitant neurologic manifestations in 17 cases of femoral neuropathy

Findings	Number of Cases
Other peripheral nerves involved	5
Absent achilles reflex	8
Diminished vibratory sensation	4
Hypesthesia	3
Autonomic nerves affected Diarrhea, gastric retention, and absent sweating response	1
Achilles tendon hyperesthesia	1

Although the sensory symptoms are always more prominent, there is usually a motor disturbance except in mild cases. One of the early manifestations of femoral neuropathy may be a "giving way of the legs." Moderate wasting of the quadriceps muscle group may be observed but marked atrophy is rare. Absence of the patellar reflex is an important sign; the impairment of this reflex can vary from slight diminution in some patients to complete, irreversible loss.

Thorough control of the diabetes is the most important measure in treatment. It usually results in early subsidence of pain and slow but gradual recovery from the other manifestations.

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Committee to Study Artificial Sweeteners

The Food and Nutrition Board of the National Research Council, at the request of the Food and Drug Administration, has named a committee to study the principles which should govern the use of artificial sweeteners in foods for special dietary purposes.

The primary purpose of the committee will be the development of a statement of general principles and of other factors for consideration in the formulation of a policy on the use of artificial sweeteners in food.

The committee, which held its first meeting in January at Ithaca, N.Y., includes Dr. W. H. Griffith, chairman, professor of physiological chemistry, University of California Medical Center, Los Angeles; Dr. B. S. Clark, president, Institute of Food Technologists; Dr. P. L. Day, professor of biochemistry, University of Arkansas School of Medicine; Dr. Norman Jolliffe, director, bureau of nutrition, New York City Department of Health; and Dr. Charlotte Young, professor of food and nutrition, Cornell University.

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