ISCHAEMIC LATERAL POPLITEAL NERVE PALSY

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The importance of vascular factors in the production of peripheral nerve affections has been considered by various authors in articles relating both to clinical and to experimental aspects of the problem. Experimental work and the information arising from cases of tourniquet paralysis have led to discussions on the relative parts played by direct neural compression and ischaemia. Thus, Lewis *et al.* (1931) concluded that ischaemia was of prime importance in producing neural symptoms, and that sensory manifestations in an ischaemic limb were not dependent upon direct nerve compression. Richards (1951) discussed tourniquet paralysis in the upper limbs, and concluded that ischaemia and direct nerve pressure both play some part in this type of paralysis.

Discussions on the common clinical problem of foot-drop usually include a reference to the vulnerability of the lateral popliteal nerve both to direct pressure and to indirect effects of pressure upon its blood vessels. Woltman (1929), in reviewing 29 cases of foot-drop, decided that weight loss followed by local compression of the lateral popliteal nerve in crossing the legs played an important part in the production of the lateral popliteal paralysis, suggesting that arteriosclerosis might also be a contributory factor. Garland and Moorhouse (1952) also emphasized the importance of mechanical factors, and confessed themselves at a loss to explain four examples in which the paralysis was said to have developed suddenly whilst walking. Sunderland (1945) stressed the point that the superficial and unprotected position of the nutrient artery to this nerve makes this vessel particularly susceptible to pressure, with the result that an ischaemic neuropathy may develop. In the literature there are, however, few accounts of the occurrence of frank vascular disturbances in association with foot-drop, although Roberts (1948), in an article dealing with experimental ischaemia of the sciatic nerve in dogs, alluded to a patient with bacterial endocarditis who developed a left lateral popliteal nerve palsy, and in whom post-mortem examination revealed thrombosis of the vasa nervorum in the centre of the affected nerve; whilst Mafson (1952), in reviewing a large series of cases of obliterative arthritis, discovered eight cases of lateral popliteal nerve palsy. The cases described below are all examples of foot-drop associated with a definite local vascular affection, and serve to illustrate the special vulnerability of the lateral popliteal nerve to ischaemia.

Case 1

A woman aged 51 was seen on February 15, 1952. Six weeks earlier she had developed spasms of pain in the right popliteal space, shooting down into the ankle and foot. After four or five days there was a slight swelling of the foot and ankle. A week later still there was numbness of her right toes and of the distal half of her foot. Four weeks after the initial pains her right foot became very floppy, and it was this symptom which troubled her most at the time of examination. Abnormalities were confined

to the right leg and comprised slight oedema from the knee, right foot colder than the left, and absence of right foot pulses. Both femoral and the left foot pulses were easily palpable. There was complete loss of power in the peronei and anterior tibial groups; all other muscles were normal. Reflexes were normal. Sensation to pinprick was diminished over the first and second toes only, to a point just proximal to the heads of the metatarsals. All investigations were normal, apart from electrical stimulation and electromyography, which indicated a lower motor neurone lesion in the lateral popliteal group, the anterior tibials being rather more affected than the peronei.

The patient was given a toe-raising spring but no other treatment. The affected muscles began to improve, and by April 24 (about four months after the onset) the peronei were almost normal and there was appreciable power in the dorsiflexors, though sensation was still impaired, the foot still cold, and the posterior tibial pulse not palpable. The improvement has continued, and on March 19, 1953, she had a normal gait and full power in the dorsiflexors of the foot. She still had "pins and needles" in the area of initial sensory loss, and the right foot was still colder than the left.

Case 2

A woman aged 44, a known case of rheumatic heart disease with mitral stenosis, suddenly developed a moderate aching pain in the right groin on July 19, 1951. Thirty-six hours later she was awakened by a severe cramp-like pain in the right calf. On getting out of bed her right foot was very weak. She massaged her leg, with some comfort, but four hours later she was reawakened by an even worse pain. She noticed now that the leg had "no colour in it," was very weak both below and above the knee, and felt numb and tingling to a point above the knee. On July 21, forty-eight hours after her first pain, she was seen in hospital, when there was evidence of popliteal thrombosis. Neurological signs comprised weakness of all the muscles of the right leg below the knee, absent ankle-jerk, and an impairment of sensation in the leg. Paravertebral lumbar sympathetic block was performed (Mr. S. Rose) and tocopherol acetate, 1 g. daily, tolazoline hydrochloride, 50 mg. t.i.d., and "cyclospasmol," 100 mg. t.i.d., were given. Within six days the movements of the leg were good, and sensation was almost back to normal. By August 11 she was able to return home "fully mobile." She continued well until February 3, 1952, when for forty-eight hours she had "pins and needles" in the right leg from the knee downwards.

On readmission to hospital, examination showed definite weakness of the anterior tibial and lateral popliteal muscles. A week later a new pain in the right calf was accompanied by the signs of popliteal occlusion, including some swelling of the leg. She rapidly developed a frank right foot-drop with impairment of sensation to pin and cotton-wool over the lower part of the leg and dorsum of the foot. A further paravertebral block on March 3 relieved much of her pain, but little improvement was noted in her neurological signs, and so she was referred to the department of neurology, where the positive findings on March 28 were: well-established mitral stenosis; absence of right pulses at and below the knee; slight wasting of right quadriceps with some fibrillation; extreme wasting of right anterior tibial and peroneal muscles with complete loss of power; a little thinning of the calf muscles, but power excellent; right ankle-jerk absent; sensory loss to pin and cotton-wool in the distribution both of the musculocutaneous and anterior tibial nerves.

The further administration of the vasodilator drugs, and the application of the toe-raising spring, enabled the patient to resume walking. When last seen on July 21, 1952, she still had the partial popliteal palsy, but was able to walk with the aid of the foot spring.

Case 3

A man aged 51, after two years of angina of effort, developed the symptoms and signs of myocardial infarction.

Six days later, whilst in bed, he had a severe pain in the right popliteal space. As the pain diminished he noticed that his right leg was numb and cold below the knee. No pulses could be felt at the ankle. Severe weakness of the dorsiflexors of the foot was noted immediately, but only slight affection of the posterior tibial muscles. Anticoagulant therapy was given, but at the end of five weeks, when he began to walk about, the degree of foot-drop was severe, and was accompanied both by pain in the calf and by oedema of the foot and leg. This led to his examination in the neurological department on January 15, 1952. Positive findings were: obvious thickening and sclerosis of peripheral vessels, but peripheral pulses palpable; slight oedema of right foot and ankle; considerable wasting of the right anterior tibial and peroneal muscles with complete loss of power in the anterior tibial and severe loss in the peronei; posterior tibial group good power with a little wasting; right ankle-jerk absent. There was loss of sensation to pinprick and cotton-wool over the lower and anterior part of the leg and the dorsum of the foot. Electromyography and electrical stimulation revealed a lower motor neurone lesion in the group of muscles supplied by the lateral popliteal nerve.

During his stay in hospital his muscle power improved a little, but shortly after discharge a further coronary attack proved fatal.

Case 4

A woman aged 58, who had complained of angina of effort relieved by glyceryl trinitrate for about four years, developed leucoplakia, for which vulvectomy was carried out. Some five weeks after the operation, and shortly before being discharged, she developed a sudden cramp in the left foot, spreading up the back of the leg as far as the knee. This pain continued for two weeks, and she noticed a swelling of the leg as high as the knee. She rested at home, and gradually the swelling subsided. On trying to walk, however, she discovered that her foot was very weak. About one week later a severe pain developed in the left antecubital fossa, striking down the centre of the arm and the radial two fingers and the thumb. She massaged both leg and arm herself, and it was not until three months after this onset of her trouble that she was examined and found to have "coldness of the limbs." Three months later still, muscle-wasting was noted in the hand and sensory loss of an indeterminate character. She was referred for a neurological opinion about 12 months after the beginning of the symptoms. By this time there had been a little improvement in her condition, and she was able to walk fairly well, but this improvement in walking resulted in the development of intermittent claudication in the left leg.

Examination revealed pronounced tortuosity and thickening of the peripheral vessels; left posterior tibial pulse not palpable; wasting of the left thenar eminence; loss of sensation to pinprick and cotton-wool in the distribution of the left median nerve; weakness of the evertors and dorsiflexors of the left foot, with a patchy loss to pinprick and cotton-wool in this area. The left ankle-jerk was absent. All investigations were normal with the exception of electrical testing. Electromyography showed lower motor neurone affection of the muscles supplied by the lateral popliteal and the left median nerves. Electrical stimulation showed partial reaction of degeneration. (The response to electrical stimulation of the medial popliteal group was normal.)

Case 5

A woman aged 43 was seen on November 4, 1952, when she complained of a floppy right foot and pain in the right leg. Five weeks earlier she had developed a pain in her right calf on walking, and 48 hours later had noticed the extreme floppiness of the right foot. Examination revealed that the pulses were not palpable in either foot, though both femoral pulses were easily felt. The right foot, much colder than the left, was rather blue in colour and slightly oedematous. There was a severe foot-drop, with some impairment of power of the plantar flexion. The right ankle-jerk was

absent. A little weakness was detectable in the muscles of the thigh, but the main affection was in the lateral popliteal group. There was a patch of anaesthesia about 3 in. (7.5 cm.) in diameter on the outer side of the leg surrounding the external malleolus.

She was given tolazoline, 50 mg. thrice daily, and since then has shown considerable improvement in the affected muscles. The foot-drop in particular was much improved, and her gait was normal, but she continued to have coldness and swelling of the affected leg.

Case 6

A man aged 63 suddenly developed pain, exacerbated by walking, in his left calf. Twenty-four hours later his left foot became floppy, and he noticed numbness of the dorsal aspect of the outer three toes. After two weeks the condition began to improve, and when seen on December 19, 1952, he felt that it was almost normal. Examination showed: left foot colder than right; femoral pulses palpable; left foot pulses and right posterior tibial pulses absent; power in peronei and anterior tibials of the left foot reduced; posterior calf muscles normal; left ankle-jerk absent; no demonstrable sensory loss.

Further investigation could not be carried out, as a few days later he developed a sudden substernal pain and died at home.

Case 7

A woman aged 70 had experienced intermittent aching pains at night in the left calf since 1951. In July, 1952, she noticed that even in the warm weather her left foot and lower part of the leg seemed unnaturally cold. Three months later she developed polyuria and polydipsia, and began to lose weight. In May, 1953, these general symptoms brought her to hospital, where a diagnosis of diabetes was made. In June she developed "floppiness" of the left foot and was sent to the neurological department, where physical examination revealed that, apart from bilateral cataracts, abnormalities were confined to the left leg. There was a moderate paresis of the anterior tibial and peroneal groups of muscles. The left ankle-jerk was diminished and there was impairment of sensation to pinprick and cotton-wool in an area some 2 in. (5 cm.) wide and 5 in. (12.5 cm.) in length surrounding the lateral malleolus. The left foot pulses were not palpable and the foot was cold.

She was provided with a suitable light toe-raising spring, and at the end of two months had recovered considerable power in the dorsiflexors of the left ankle. The foot was still cold, although the pulses could then be palpated. She has continued to improve.

Case 8

A man aged 54 had a somewhat complicated medical history, mostly irrelevant to the present problem. Briefly, he was admitted to hospital in May, 1950, with lightning pains, Argyll Robertson pupils, and absent tendon jerks. His blood W.R. was positive, and C.S.F. analysis showed 49 cells, 50 mg. of protein, Lange 0112110000. The C.S.F. W.R. was also positive. Treatment with 14 mega units of penicillin in three weeks produced some improvement in the lightning pains, and allowed his C.S.F. chemistry to return to normal, although the W.R. remained positive in blood and C.S.F. two years after treatment. In March, 1952, he was admitted to another hospital for repair of a perforated duodenal ulcer (not a gastric crisis!), and during the subsequent treatment was given intravenous fluids into his right saphenous vein at the ankle (one week). After the drip was stopped his foot was swollen and he could not move his foot up or down. Two weeks later he was allowed up, and found that he could stand on his right leg but that the foot flopped about.

He was transferred to the neurological department four weeks after the operation. Examination of the right leg revealed the external popliteal palsy, with a patch of anaesthesia and analgesia over the outer side of the leg and dorsum of the foot. The ankle-jerk was absent. His

foot pulses could not be palpated on the right side, although they were easily palpable on the left. Electromyography showed a severe but incomplete lesion of the lateral popliteal nerve.

Massage and exercises were given, and a foot spring was provided. When seen four months later his condition was very much improved; although he still had some weakness of the peronei, he was otherwise recovered, and was able to walk normally. Eleven months after the paralysis his muscle power was normal, but he complained bitterly of his cold feet and said the right was worse in this respect than the left. His pulses could then (February 19, 1953) be palpated normally.

Case 9

A woman aged 67 was admitted to a mental hospital on March 31, 1952, on account of involutional melancholia with paranoid trends (including a fear of being poisoned). During the course of treatment she was given an intravenous injection into the left saphenous vein, the leg being bound to a wooden back splint for a period of 48 hours. At the end of that time she developed some infection of the vein, and within the next two days a swelling of the left leg to a level above the knee developed. The observers are certain that at this time there was no evidence of foot-drop and that during the extreme activity which ensued the patient was moving all parts of all her limbs naturally and violently. By April 24 the swelling had almost subsided. She was sufficiently recovered mentally to be allowed up, but when she tried to walk on April 28 she had a definite left foot-drop. Immediate examination revealed, in addition to the paralysis with loss of power in the anterior tibials and peronei, an area of hypoaesthesia in the anterior tibial and musculocutaneous areas. The posterior tibial pulse on the left side could not be palpated, nor could the dorsalis pedis artery. On May 2 these findings were unchanged, though the area of sensory loss was much less extensive. The tendon reflexes were brisk and equal.

This patient, too, was given a foot spring, but no other treatment. During the next three months she improved steadily and when last seen had only a minimal weakness of the dorsiflexors of the foot.

Discussion

The first seven cases described show a very definite association between the arterial occlusive lesion and the onset of the foot-drop. In Cases 8 and 9 the immobility of the leg required for maintenance of the intravenous therapy may have resulted in a compressive ischaemia of the lateral popliteal nerve of the type suggested by Sunderland (1945), but in both cases the onset of the foot-drop seems to have been separated from the infusion by several days, and in both cases a period of oedema of the leg intervened. Furthermore, in both cases there were the obvious vascular abnormalities and the absence of the peripheral pulses in the affected leg. Case 4 had the interesting median-nerve palsy in addition.

These cases illustrate the susceptibility of the lateral popliteal nerve to ischaemia of whatever origin, and indicate the predominance of motor affection with the relative paucity of sensory signs. The sensory loss was slight, and varied from case to case, but the manifestations of the foot-drop followed a constant pattern.

The question naturally arises regarding the reason for so much affection of the lateral popliteal muscles in contrast to retention of the medial popliteal function in cases in which the occlusion appears to have been of the popliteal artery. It is interesting, however, to note that in six of the nine cases the ankle-jerk was absent for a time at least. The medial popliteal nerve receives a fairly rich blood supply, stemming in part from the gluteal arteries and perforating branches of the femoral artery, with additional nutrient vessels entering at various points in the popliteal space and in the calf. The lateral nerve, on the other hand, receives only one nutrient vessel within the popliteal space

(Sunderland, 1945). Bentley and Schlapp (1931) were able to show, in experiments on the sciatic nerve of the cat, that if the anoxic portion of the nerve exceeds 5 cm. considerable impairment of function of the nerve ensues. An occlusion of the femoral or popliteal artery therefore constitutes a much greater ischaemic threat to the lateral popliteal division than to the medial division, and, in particular, an obstruction at or just above the popliteal space itself will render the lateral nerve ischaemic over a critical length. Bentley and Schlapp also showed in their work that recovery of function is more rapid and complete in a nerve which has been rendered ischaemic than in one which has been subject to direct compression of its fibres. This fact accords well with the good functional prognosis for the local lesions described in our cases.

From the clinical point of view, the main feature is the benign prognosis of the local process, some degree of recovery being the rule and return to full power likely. The two deaths which occurred were attributable to the associated coronary disease. The most satisfactory line of treatment is the provision of the light elastic type of toe-raising spring during the period of paresis. Tolazoline and tocopherol acetate have been used, with a view to improvement in collateral circulation, but it is not possible to be certain about the exact benefits of such drugs.

Summary

Nine cases are described in which a local vascular abnormality, either thrombosis or embolism of the femoral or popliteal arteries, was followed by foot-drop.

Attention is drawn to the vulnerability of the lateral popliteal nerve to ischaemia and to the underlying anatomical factors which may account for this.

Clinically the local condition is shown to be relatively benign and has a good functional prognosis.

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A new electronic clinical thermometer which gives an accurate temperature reading in five to seven seconds—less time than it takes to shake down the mercury in the ordinary type—was demonstrated recently at the Army Medical Service Graduate School, Washington. The electronic thermometer, known as the "Swiftem," has a thermistor (thermally sensitive resistor) attached at the tip of the delicate steel probe which replaces the old-fashioned glass mouth-piece. The recording meter and the mercury cell battery which supplies the power are contained in a plastic case small enough to fit easily into the palm of the hand. The thermometer is operated by a finger button switch and in appearance resembles a typical photographic light meter. Handpiece and thermometer are connected by a transmission cord, but its length does not affect the sensitivity or accuracy of the instrument. Its inventor, Colonel George T. Perkins, Director of the Dental Division of the U.S. Army Medical Service Graduate School, said it would be possible for a nurse to remain at her desk in the ward and take a patient's temperature in another part of the ward. The meter registers degrees of temperature in black figures below normal and red figures above normal. The steel probe is easily detached for sterilization, and tends to eliminate breakage and loss. It is claimed that the "Swiftem" represents the first change in clinical thermometers since the mercury-column type was introduced in 1867. In addition to recording a temperature much more quickly, the electronic one is more accurate in its readings.—*U.S.A. in Review*, June 25.