schema indicate that this lesion cannot have been in the internal ear, but must have involved the vestibular pathways at the cortical or subcortical level. The disorders of the body-schema, which impaired awareness of the size and shape of the body as a whole, the relationship between its parts, and of the whole body to gravity, are of an unusual kind, but are those particularly likely to result from a disorder of the vestibular contribution to the body-schema (Schilder, 1935). There is evidence for cortical representation of the vestibular system in the temporo-parietal region, and this lies near the parietal area, which, in the right cerebral hemisphere at least, is concerned with the representation of the body-schema. The visual symptoms, none of which is very common, are all known to occur after a lesion in the occipital lobe (Brain, 1947; Critchley, 1949). The apparent tilting of objects, a form of metamorphopsia, is not due to an abnormal position of the eyes, as Hunter thought, but to a disorganization of the cortical integration of awareness of visual space, a process in which both vestibular and ocular factors play a part, the latter undoubtedly including unconscious afferent impulses from the proprioceptors of the eye muscles. The appearance of objects as either abnormally small or abnormally distant can also be the result of disordered awareness of visual space, since judgments of the size of objects and their distance are reciprocally related.

Hyperaesthesia to sensory stimuli, while not uncommon in respect of painful stimuli—for example, in the thalamic syndrome—rarely occurs in the case of sight and hearing as a result of a cerebral lesion. One of my patients (Brain, 1947), who had a vascular lesion in the right parieto-occipital region, in addition to suffering from visual hallucinations, micropsia and macropsia, and disorders of the body-schema, complained that sounds appeared much louder than normal. Moreover, she experienced synaesthetic hallucinations, the sound of a car backfiring causing her to see brilliant colours in the left (hemianopic) half of the visual fields. Such symptoms, produced by organic lesions of the parieto-occipital region, resemble some of the symptoms of mescaline intoxication. We do not know how sensory activity can thus irradiate from one sense to another, nor how a general hyperaesthesia of hearing, sight, smell, and taste is caused. Loss of topographical memory is commonly due to a lesion in the occipital lobe, usually in the left cerebral hemisphere, but sometimes in the right.

It is highly probable that the sensory attacks accompanied by pain and hyperalgesia or hyperpathia in the left upper limb were a variety of sensory epilepsy, for Home noted that they always began in the same way, and records that on one occasion Hunter lost consciousness at the height of the attack and had no recollection of it afterwards. They suggest a lesion of the right parietal lobe extending rather deeply into the neighbourhood of the optic thalamus. Similar attacks were experienced by a patient of mine who had a glioma in this region (Henson, 1949).

D'Arcy Power (1931) has attributed John Hunter's symptoms to cardio-aortic and cerebral syphilis resulting from his self-inoculation with syphilis in 1767. The dilatation of the aorta, amounting to an "incipient aneurysm," certainly suggests syphilis, and the description of the intimal surface is consistent with a syphilitic aortitis which had gone on to severe atheroma. The patches of pericarditis might have been due to underlying infarction of the myocardium. But the naked-eye appearances of both his coronary and his cerebral vessels suggest advanced atheroma. It is surprising that no naked-eye changes were detected in the brain. D'Arcy Power points out that the anatomists who carried out the post-mortem examination would have been unlikely to overlook a gross lesion such as a gumma, and infers that the lesions must have been due to endarteritis of the smaller vessels. Whether this or atheroma was the cause, the resulting areas of ischaemic softening must have passed undetected. When we reflect that for the last 17 years of his life John Hunter suffered from progressive disease of the brain, and for the last eight years from epilepsy, in addition to angina, we may agree with D'Arcy Power that it is "marvellous that he could accomplish so much, crippled as he was mentally and physically."

I am grateful to Professor Dorothy Russell for kindly discussing the pathology with me.

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COMPRESSIVE LESIONS OF THE EXTERNAL POPLITEAL (COMMON PERONEAL) NERVE

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Most adults are familiar with the transient condition of "the foot going to sleep." If a hurried examination is made when this occurs it will be found that the temporary foot-drop results from weakness of those muscles supplied by the external popliteal nerve only. It is a commonplace observation that this event results from such things as prolonged kneeling or, more frequently, crossing one leg over the other while sitting. It would seem certain that it follows temporary ischaemia of the nerve, resulting from compression, and complete recovery in a matter of seconds is the general rule. The student or practitioner is not, however, so familiar with more prolonged states of external popliteal paralysis, and he is perhaps more likely to be puzzled by this fairly common condition than he would be by such disorders as tabes dorsalis, syringomyelia, or myasthenia gravis, all of which are rare.

Should the student wish to amplify his knowledge of the subject he will find little help from British medical writings; there have been remarkably few papers devoted to this subject, and references in current English works appear to be either inaccurate or inadequate. For example, according to Walshe (1952) the aetiology is usually unknown and the paralysis is often preceded by pain; recovery is slow and frequently incomplete, and the condition may result from a "neuritis" in association with diabetes or typhoid fever. According to Brain (1951) the external popliteal nerve may suffer from "interstitial neuritis," and the peronei are usually more gravely affected than the anterior tibial group of muscles. Purdon Martin and Elkington (1950), however, state that paralysis is usually severe, all the muscles being equally affected, subjective sensory changes being usually absent. Purves-Stewart and Worster-Drought (1952) state that the earliest symptom is pain in the relevant area of distribution. Most of these authors make some reference to compression by garters or tight bandages, though Worster-Drought and Sargent (1952) make no mention of pressure as a cause of external popliteal palsy. Gowers (1899) regarded most palsies of single peripheral nerves as being the result of "interstitial neuritis," though he thought that isolated

"neuritis" occurring during an acute illness probably arose through the agency of unnoticed compression. Finally, Kinnier Wilson (1940) made the remarkable statement, "Doubtless some examples of so-called idiopathic peroneal paralysis have a rheumatic origin. Local neoplastic processes should also be mentioned."

In this paper we are concerned only with examples of uncomplicated external popliteal paralysis, and will make no reference to such a palsy resulting from gross trauma or involvement of the nerve in leprosy or polyarteritis nodosa, though in fact we have never seen an example of either of these latter conditions; nor are we concerned with any lesion of the sciatic nerve above its point of bifurcation. Finally, we shall not discuss foot-drop resulting from any central nervous lesion. Thus limiting the field, we believe that external popliteal palsy probably always arises from compression of the nerve as it winds round the neck of the fibula. In support of this thesis we describe 20 examples that have come to our notice in the past four years. From the scattered sources of this material we have no doubt that the condition is quite common.

Anatomy

The external popliteal nerve is normally derived from the dorsal divisions of the fourth and fifth lumbar and the first and second sacral roots, though variations in the root value occur. Incorporated in the sciatic nerve, it passes out of the pelvis and descends in the posterior part of the thigh. The sciatic nerve normally divides into the external and internal popliteal nerves at the upper level of the distal third of the thigh (the proximal angle of the popliteal space), though the division is variable and may occur at any point below the plexus. It runs downwards and outwards through the popliteal space, following the tendon of the biceps femoris and passing over the outer head of the gastrocnemius to the back of the head of the fibula (Fig. 1, A). From here it winds obliquely forwards round the neck of the fibula, disappearing deep to the origin of the peroneus longus. Where it is in relation to the head and neck of the

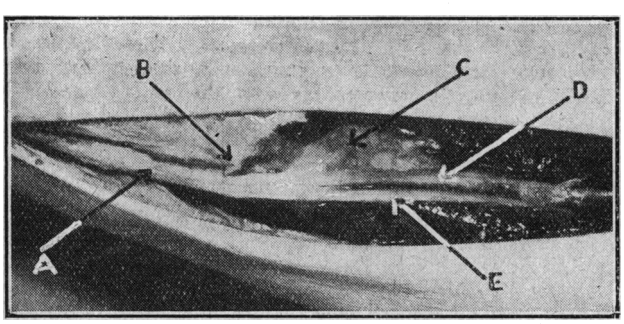

FIG. 1.—A, External popliteal nerve. B, Neck of fibula. C, Recurrent articular branch. D, Anterior tibial nerve. E, Musculocutaneous nerve.

fibula (Fig. 1, B) the nerve is quite superficial and can easily be palpated and rolled against the bone in most subjects. It gives a muscular branch to the short head of the biceps, and there are three articular branches to the knee-joint; the most distal of these, the recurrent articular branch (Fig. 1, C), also supplies part of the tibialis anticus muscle and the superior tibio-fibular joint. There are two cutaneous branches—the lateral cutaneous nerve of the calf, which supplies a variable area of the skin of the proximal two-thirds of the lateral side of the calf, and the sural communicating nerve, which joins the sural nerve in the middle of the calf.

The nerve then divides into anterior tibial (Fig. 1, D) and musculocutaneous (Fig. 1, E) branches. The former supplies the tibialis anticus, extensor hallucis longus, extensor digitorum communis, and peroneus tertius, with branches to the ankle and inferior tibio-fibular joints. It then subdivides to supply the extensor digitorum brevis, the first, second, and third dorsal interossei, some of the toe-joints, and the skin of the adjacent sides of the great and second toes. The musculo-cutaneous nerve supplies the peroneus longus and peroneus brevis, with cutaneous branches to the skin of the distal third of the anterior surface of the leg, the lateral side of the ankle, the dorsum of the foot, and some of the skin of the toes.

It will be seen that where the nerve lies on the fibula it must be unusually vulnerable to pressure from outside. A similar state of affairs is seen where the ulnar nerve lies in the ulnar groove, and it is not without significance that these, the only two large nerves normally palpable, are much the most common sites of peripheral nerve palsy, excluding the results of gross injury.

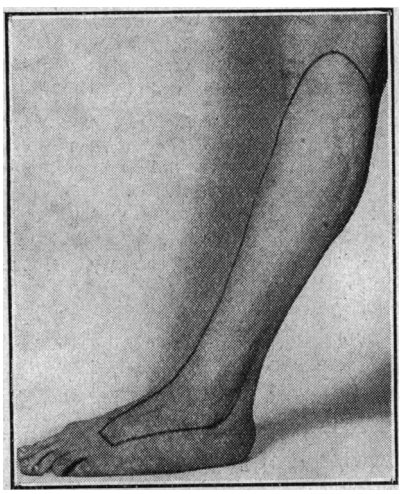

FIG. 2.—Typical area of external popliteal anaesthesia (Case 17).

A complete lesion of the nerve at this point results in paralysis of dorsiflexion and eversion of the foot and paralysis of extension (dorsiflexion) of the toes. The patient has to raise the leg to clear the toe when walking, and tends to bring the foot down heavily; he can stand on his toes but not on his heel. There is an area of cutaneous sensory loss extending over the greater part of the antero-lateral aspect of the leg below the knee and on to the dorsum of the foot (Fig. 2). There is no weakness of plantar flexion at the ankle or toes, nor of inversion of the foot, and the knee- and ankle-jerks are normal.

In all the patients under review the lesion must have occurred above the division of the nerve into its two terminal branches (Fig. 1), and we have not seen an unequivocal isolated paralysis of either of these branches; nor is there any evidence of involvement of the biceps, so that on anatomical grounds the situation of the lesion must be restricted to narrow limits—namely, the point at which the nerve, just proximal to or at its point of main division, is in contact with the fibula.

Case Reports

In the case reports the following abbreviations will be used: T.A. (tibialis anticus), P. (peronei), E.H.L. (extensor hallucis longus), E.D.C. (extensor digitorum communis). The standard M.R.C. method of recording weakness is used; the main defect of this system is that "4" means that a muscle is able to contract against gravity and against "resistance," but the resistance is not defined, and this degree of weakness does, in fact, cover a very wide range.

Case 1.—A man aged 43 was first seen on December 12, 1950. Ten days previously he was squatting on his haunches for several hours, and on getting up had a left foot-drop. All external popliteal muscles were totally paralysed, with a typical area of hypalgesia. He was given a toe-raising spring and recovered completely in six months. He gave a history of three previous attacks in the same foot, each of which recovered in a few days, and subsequently had an attack in the right foot, which recovered (not examined by us) in two months. No abnormality was found on re-examination on July 14, 1952.

Case 2.—A woman aged 37 ten days previously had been squatting or kneeling while cutting the edges of the lawn, and on getting up found she had developed numbness of the right foot and a limp. Examination on September 1, 1950, showed T.A. and E.H.L. 4+, P. and E.D.C. 5, with no sensory loss. The patient normally crosses the right leg over the left when sitting, and is familiar with "the foot going to sleep." There was complete recovery in one month.

Case 3.—A man aged 29 developed right foot-drop suddenly in January, 1949, following an unusual job in which he was kneeling with his buttocks resting on his heels for about 45 minutes. At first there was numbness on the dorsum of the foot. Recovery occurred very slowly in about one year. When examined by us on July 26, 1952, he had no neurological abnormality. He had genu varum with 1¾ in. (4.4 cm.) of separation.

Case 4.—A man aged 25 while walking 13 days previously had felt "pins and needles" on the dorsum of the left foot, which lasted a few minutes, and was followed by numbness; he noticed weakness a few hours later. On the previous afternoon he had been kneeling and squatting in an unusual position for several hours. When seen on July 31, 1952, the disability was stationary. Examination showed T.A., E.H.L., and E.D.C. 2, P. 4 with very vague hypalgesia on the dorsum of the foot.

Case 5.—A male Parsee aged 19 developed right foot-drop suddenly on June 14, 1952, on the third day of strawberry-picking. He was wearing gumboots and tended to sit with his left leg fully extended and his right leg fully flexed at the knee. He was immediately aware of numbness, but only on the dorsum of the foot. The gumboot acted as a back-splint and he continued to work another one and a half days. On the fifth day he was admitted (undiagnosed) to hospital for a week, and wore a back-splint for two months. Improvement was first noticed after about a week, and continued slowly. First examined by us on September 13, 1952: E.H.L. and E.D.C. 3, T.A. and P. 4+, with hypalgesia over the whole distribution of the nerve.

Comment on Cases 1–5.—There would seem to be no doubt that in this group four out of five developed a unilateral palsy while kneeling, and in three the particular posture was unusual for the patient. A different story was told in Case 4, this patient insisting that his symptoms developed on the day following prolonged and unaccustomed kneeling.

Case 6.—A man aged 56 developed a fractured femur on July 3, 1947, which was treated by splinting. When the splints were removed he had a right foot-drop. This began to recover slowly and he returned to work on March 1, 1948. He was first seen by us on August 30, 1948, we found E.H.L. and E.D.C. 1, T.A. 2, P. 5, with no sensory loss. On January 14, 1949, he was still wearing a toe-raising spring; examination then showed no change, and there was still no change on July 5, 1952.

Case 7.—A man aged 48 had been grossly overweight, and in the previous year had reduced from 26 to 16 stones (165 to 101.5 kg.). Two months previously he had developed skin sepsis, for which he was wearing a bandage below the left knee. This had been applied particularly tightly for three days, when he suddenly (five weeks previously) developed left foot-drop while walking, subsequent to prolonged standing. When first seen on December 17, 1951, we found E.H.L. and E.D.C. 2, T.A. 3, P. 4+, with no sensory changes. Recovery began quickly and was complete in three months. He had no abnormality on June 10, 1952.

Case 8.—A woman aged 48 sprained her right ankle on September 19, 1950, and her leg was strapped almost to the knee. Foot-drop developed suddenly on the fourth day of strapping. She attended an orthopaedic department two months later and was given a toe-raising spring. About this time her palsy began to improve. Examination on May 24, 1952, showed E.H.L. 2, T.A. and E.D.C. 3, P. 4. There were slight subjective and objective sensory changes in the relevant area.

Case 9.—A boy aged 15 was admitted to hospital on December 20, 1951, with osteomyelitis of the left ischium, and on December 31 was put on an abduction frame, with strapping round both legs from the knees down. Left foot-drop was first detected on the fourth day. Examination was of necessity incomplete, but only muscles supplied by the external popliteal nerve were involved. When seen on June 10, 1952, the foot had recovered apart from the big toe; recovery started in May and was continuing. On examination: E.H.L. 0, T.A., P., and E.D.C. 4+, with slight hypalgesia.

Comment on Cases 6–9.—There seems to be no doubt that in all four cases the palsy resulted from strapping applied below the knee. The precise time of onset is unknown in two, but in the others it is clear that the foot-drop developed suddenly after several days. Another important observation is that two of these patients show incomplete recovery after very long periods, and it is most unlikely that there will be any further recovery.

Case 10.—A woman aged 56 had left foot-drop in February, 1952, after sitting with her legs crossed. She improved after two months. Examination on July 5, 1952: P. 1, E.H.L. and E.D.C. 2, T.A. 3, with no sensory loss. The patient usually crosses her left leg over the right.

Case 11.—A man aged 46 developed right foot-drop in March, 1951, and one month later was admitted to another hospital for physiotherapy. There was never any sensory loss. He began to improve after four months, and recovered completely in six months. When seen by us on August 2, 1952, there was no abnormality. He normally crosses his right leg over the left.

Case 12.—A woman aged 43 had a long drive in a new car three days previously, and went to sleep with her left leg crossed over the right. On waking two hours later her left leg felt dead and there was difficulty in walking. On examination on December 11, 1949: T.A., P., E.H.L., and E.D.C. were all 4+, with a typical area of slight hypalgesia. Complete recovery occurred in two weeks.

Comment on Cases 10–12.—Case 12 appears to be a straightforward example of external popliteal palsy following crossing the legs. Cases 10 and 11 were unable to give very clear accounts of themselves, though both normally crossed the affected leg over the other. A fourth example is seen in the left leg in Case 15. Attention to crossing the legs as a frequent cause of foot-drop was drawn by Woltman (1929), and again, especially in relation to occupation, by Nagler and Rangell (1947).

Case 13.—A miner aged 24 five weeks previously developed right foot-drop with numbness over the relevant area, after working at the coal-face in a kneeling position and wearing knee-pads. He had been at this particular work for two years, and continued at the same work after the palsy developed. On March 28, 1949, we found T.A. 0, P., E.H.L. and E.D.C. 1, with a typical area of hypalgesia. He was fitted with a toe-raising spring, and on May 9, 1949, had made a complete recovery.

Case 14.—A miner aged 32 had been at the coal-face for only eight weeks. Three weeks previously he bought a new pair of knee-pads, and after wearing these for two days began to drag the left toes. He gave up work on the fourth day because he was unable to dorsiflex the foot, and had an area of external popliteal numbness. Recovery began in two weeks and was complete, without treatment, in six weeks. When examined on May 24, 1952, there was no abnormality.

Case 15.—A floor-layer aged 24, who spends all his working hours kneeling (Fig. 3), six weeks previously had noticed loss of control of the right foot, and two weeks previously loss of control of the left foot. He is satisfied that the right foot-drop developed while at work and the left during a train journey when he was sitting with the left leg

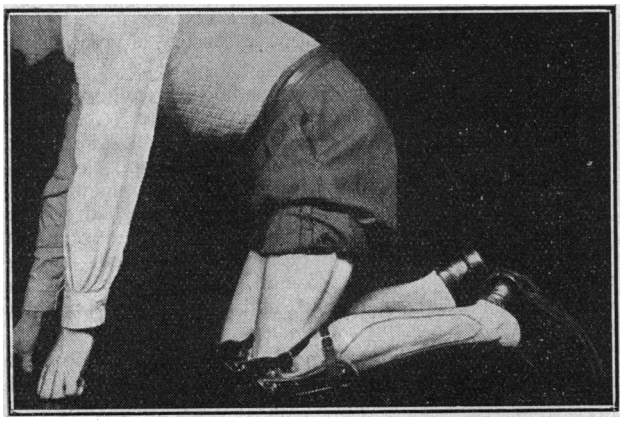

FIG. 3.—Knee-pad showing the position of the straps and external popliteal anaesthesia.

crossed over the right. Examination on March 27, 1952, showed: Right, T.A. 1, P. 2, E.H.L. 3, E.D.C. 5; left, T.A. 1, P. and E.H.L. 2, E.D.C. 5, with a clearly defined and typical area of hypalgesia on each leg (Fig. 3). He discarded his knee-pads but continued to work. The right foot recovered in ten weeks and the left in five weeks. Complete recovery was confirmed on August 2, 1952.

Comment on Cases 13–15.—It would seem probable that in Cases 13 and 14, and in the right leg in Case 15, an important factor was compression of the nerve by the lower strap of the knee-pads, but whether it played a part in the production of left foot-drop in Case 15 is not known. Knee-pads are perhaps worn most frequently by coal-miners, and Dr. John Rogan, Chief Medical Officer to the National Coal Board, has kindly provided us with the following information. The incidence of the wearing of knee-pads varies considerably from 60% of all underground workers to 15% of face-workers; this is largely determined by the depth of the seam, all face-workers wearing knee-pads when the seam is 3 feet (0.9 metre) or less, and very few when more than 4 feet (1.2 metres). The life of the knee-pads varies from three months to two years. External popliteal palsies do not seem to have been recorded by the Board's medical officers. We would point out, however, that our own Cases 13 and 14 were coal-miners whose disability seems to have escaped their medical officers. No doubt multiple factors are involved in the production of foot-drop by knee-pads, including the type, newness, and tightness of the strap, the thickness of the trousers, and the length of time spent in one position. We are not aware of any previous reference to knee-pads as an aetiological factor in this condition, and it is the kind of factor which is easily overlooked.

Case 16.—A woman aged 23 had a prolonged febrile illness with gross loss of weight early in 1950, and in April had a left nephrectomy, lying on her right side on the operating table. On getting out of bed some weeks later she had a right foot-drop. When first seen by us on December 7, 1950, we found E.H.L. 2, T.A. 4, E.D.C. and P. 5, with slight external popliteal hypalgesia. The patient was wearing a toe-raising spring. Recovery was complete in about one year, and there was no weakness when we saw her on May 24, 1951.

Comment on Case 16.—This seems to be an example of compression of the nerve on a hard surface during operation, in a patient who had lost a good deal of weight, though the possibility of subsequent compression while lying in bed on her right side cannot be excluded.

Case 17.—A woman aged 62 had developed a left external popliteal palsy five weeks previously. Examination on July 7, 1952, showed E.H.L. 0, E.D.C. 2, P. 3, T.A. 4, with a typical area of hypalgesia (Fig. 2). Her blood pressure was 155/105. After two weeks she was fitted with a toe-raising spring, and thinks that recovery began about this time. When seen again on August 9, 1952, the foot was still recovering: E.H.L. 2, T.A. and E.D.C. 4, P. 5. The hypalgesia had entirely disappeared. On September 1, 1952, she had virtually recovered.

Comment on Case 17.—The aetiological factors are uncertain. The patient never crosses her knees when sitting. She is certain that the foot-drop developed suddenly after she had left work and walked some 200 yards (180 metres). Her work on an electric dish-washer involves standing for long periods, lifting racks containing crockery from left to right, and at the same time turning through an angle of 90 degrees, then passing the trays still further to the right. At this stage her left leg is close to any one of three large iron levers; the distance from the ground to the point at which the external popliteal nerve crosses the fibula is about 16 in. (40 cm.); each lever is fixed 14 in. (35 cm.) off the ground, is about 6 in. (15 cm.) long, and the distal end is never less than 17 in. (43 cm.) from the ground. It is therefore possible that the nerve was compressed by one of these levers, though the patient is not aware of this having occurred.

Case 18.—A man aged 65, a mild diabetic of four to five months' duration, whose glycosuria was controlled by dieting and who had recently lost a little weight, suddenly noticed, about five weeks previously, dragging of the left foot after walking about 100 yards (90 metres) and, almost immediately, numbness on the dorsum of the foot. Examination on June 14, 1952, showed E.H.L. 0, T.A. and P. 3, E.D.C. 4, with hypalgesia over the antero-lateral aspect of the leg and hyperalgesia over the dorsum of the foot. His blood pressure was 170/110. The palsy began to improve after two months, and when seen again on August 9, 1952, he had recovered completely.

Comment on Case 18.—Here again the aetiology is uncertain. The patient is a clerk who sits all day at his work and frequently crosses his legs, but he is certain that he crosses the right over the left. He is also certain that foot-drop appeared while walking and was not present on waking that morning. He usually sleeps on his right side, on a medium-hard mattress, and does not wear garters or suspenders.

Case 19.—A youth aged 18 noticed right foot-drop immediately on getting out of bed about a month previously. Weakness was maximal from the onset and there was no numbness or pain. He thought that recovery had begun after three weeks and was continuing. Examination on August 9, 1952: T.A., E.H.L., and E.D.C. 1, P. 3, with no sensory loss.

Comment on Case 19.—This patient is certain that the weakness was present on waking, and says he always sleeps on his left side on a soft mattress. His work as an engine cleaner involves sitting and standing, but no kneeling. When sitting he usually crosses the right leg over the left, but he is not familiar with "the foot going to sleep." He never wears garters or suspenders, and there had been no recent loss of weight.

Discussion

Twenty examples of external popliteal palsy have been seen in 19 individuals. These are the only examples we have been able to examine, and are in no other way selected. Of the 19, 14 were males; there has been previous reference to a preponderance of males (Woltman, 1929). The right and left sides were each involved 10 times. At the time of onset the oldest patient was 65 and the youngest 15. In all these patients the nerve was very easily palpable as it crossed the fibula. In contrast to statements quoted in the introduction, there was a complete denial of pain at any stage, as was also noted by Woltman (1929). It is also clear from this series that the degree of involvement and the rate of recovery of affected muscles is by no means constant; apart from four examples, in all of which the difference was slight, the extensor hallucis longus was the most severely affected muscle; the tibialis anticus was less severely involved than the extensor hallucis longus in all but these

four. The peronei were more affected than the other muscles only once, whereas in three the peronei were normal when other muscles were severely paralysed.

Although it has been impossible to make frequent examinations, it seems clear that the extensor hallucis longus tends to be the most severely affected muscle and to be the last to recover. This can be correlated with the well-known observation that sciatica resulting from root compression by a disk protrusion often results in weakness of the extensor hallucis longus as an isolated palsy. In only 7 out of the 20 had the patient noted any form of paraesthesiae, though in 13 objective sensory changes were discovered; all these showed varying degrees of impairment of pain sensation, but in none was there complete analgesia. It is therefore clear that in this group of patients the incidence of damage is much greater in the motor than in the sensory fibres. Denny-Brown and Brenner (1944) point out that the late involvement of pain sense from direct pressure on nerves had been noted as long ago as 1895 by Bastien and Vulpian; they also note that tourniquet paralysis is often purely motor.

Every patient showed some degree of recovery, this being complete in 13 and in a recovering phase in 5, leaving 2 who had recovered only in part; it was complete in one patient in two weeks and in another was incomplete after four years. One patient gave a history of recurrent attacks, of which only one came under our care, but the patient, who is an intelligent and observant person, is certain that the three previous palsies all recovered completely in a few days. Unfortunately, no patient was examined within hours of the onset, and only three were seen within the first ten days. Through lack of early observations we are unable to correlate recovery with the degree of paralysis at the onset (though we suspect that paralysis is always complete at first), but it is true to say of most that complete recovery takes several weeks or months. It is interesting that in the only two patients showing incomplete recovery the main aetiological factor was bandaging; in such patients the foot-drop may not be detected for some days after its onset. Although our figures are small they do suggest that this eminently preventable form of foot-drop carries the worst prognosis.

In the past, and to a less extent to this day, it has been customary to invoke a non-specific inflammation as the explanation for isolated peripheral nerve lesions of obscure origin. However, with the exception of leprosy there seems to be no satisfactory proof of infective "neuritis," and indeed the peripheral nerves appear to be remarkably resistant to the onslaught of organisms, perhaps almost more than any other human tissue. Isolated peripheral nerve palsies are well-known events in the rare disorder polyarteritis nodosa (Miller, 1949; Astley and Rangam, 1950), and in these the basic pathology is vascular occlusion. Similar effects have been described by Barker (1938) in thrombo-angiitis obliterans, when permanent derangement of the architecture of nerves may result from vascular occlusion. It is true to say, in general, that there have been few studies of the pathology of isolated peripheral nerves in the human being, excluding the results of gross trauma, because it is usually impracticable to perform biopsies on peripheral nerves, and post-mortem or amputation studies have been notably neglected. Important observations on the results of nerve ischaemia have, however, been made by Lewis *et al.* (1931) and by Denny-Brown and Brenner (1944), and it would seem that the only *proved* causes of damage, whether temporary or permanent, to an isolated peripheral nerve are gross trauma, ischaemia (from various factors), and leprosy. In the patients under review the first and last of these are excluded.

As already stated, it would seem certain that the familiar and transient palsy resulting from crossing the legs is of ischaemic origin. Simple personal observations on this common event show the following features. It is not easy to produce by design, but this can be done, though the latent period in the production of a complete palsy is usually at least ten minutes. The onset of the paralysis is symptomless, and the first subjective evidence of its existence is seen only when the compression is released by moving the uppermost limb. Immediate examination shows complete paralysis of all the muscles innervated by the external popliteal nerve. During this phase there are demonstrable changes in the area of cutaneous distribution, which include faulty localization and an unpleasant over-reaction to painful stimuli. We have not been bold enough to carry out very prolonged investigations of this kind, but we have been unable to produce total analgesia. The tingling which follows release of the temporary compression seems to be due to revascularization of the nerve, and this sensation is also rather difficult to localize. It seems that right-handed people tend to cross the right leg over the left when sitting, and that left-handed people tend to cross the left over the right. The Table shows the position in 100 adults selected only to the extent that 52 were males and 48 were females. Three of the right-handed who crossed left over right said that they were left-footed.

	R. Leg over L.	L. Leg over R.	Total
Right-handed	71	19	90
Left-handed	0	10	10
Total	71	29	100

It may, of course, be suggested that compression results in damage to nerve fibres quite apart from the effects of temporary ischaemia, but so far as we know there are no such proved results of pressure capable of resolution within a few seconds—an observation also made by Denny-Brown and Brenner (1944). At one end of the scale, therefore, is the transient nerve block resulting from temporary ischaemia, and at the other the permanent changes in the structure of the nerve resulting from the vascular occlusion of polyarteritis nodosa and thrombo-angiitis obliterans. Within these limits it would seem reasonable to postulate every degree of damage from the transient and completely reversible to the completely irreversible. However, so far as we know, the type of patient under review never shows a complete and permanent palsy, but some show permanent though partial lesions.

If ischaemia is the essential basis of these palsies, multiple factors have to be considered. In 17 of the 20 examples under review, such mechanical factors as kneeling, bandaging, crossing the legs, the wearing of knee-pads, or lying on a hard surface seem to be established. Widespread atherosclerosis may well be a factor of importance, and vascular hypertension certainly existed in two of the three whose origin was not clear. The significance of diabetes in aetiology is uncertain, but only one of our patients was a diabetic, and he was a man of 65 with vascular hypertension; moreover, in the studies of Woltman and Wilder (1929) and of Rudy and Epstein (1945) on diabetic neuropathy, no reference is made to any example of isolated popliteal or other nerve palsy. We do not believe that diabetes is in itself ever an important factor in this condition. This series does not include an example of the palsy complicating typhoid fever, but we suggest that when it arises it results from the combination of emaciation and compression. In this connexion serious loss of weight, with resulting disappearance of protective fat, was seen in two of our patients. If our view (that ischaemia from prolonged compression is the main factor in this disorder) is correct, it may well explain why we have been unable to trace any example of external popliteal palsy in children (though it might arise from tight bandaging), for the child normally never remains in a fixed position for a sufficiently long time.

It is probably of significance that in four examples the paralysis is said to have developed suddenly while walking; we can draw no conclusions, but would call attention to the fact that two of these were elderly people with hypertension. Finally, although the precise factors in three of

the patients are not clear, we would emphasize that in these the clinical picture and duration of the disability fall well within the limits of those whose aetiology is more certain.

Treatment

With regard to treatment, we would make the following observations. Patients should be warned against crossing legs when sitting. There is no justification for the admission of these patients to hospital. A toe-raising spring overcomes the essential disability and even in heavy industry there is no need for the patient to remain off work. Massage, coloured lights, and other forms of passive physiotherapy, not to mention the administration of vitamin B_1, play no part whatever in treatment. The patient should be instructed to carry out active movements of the affected muscles as often as possible when not wearing a spring, and, indeed, the spring itself is necessary only in the event of a severe paralysis of the tibialis anticus.

Summary and Conclusions

Paralysis of the external popliteal nerve, excluding the results of gross trauma, is not uncommon.

Such paralysis generally (if not always) results from local nerve ischaemia, and simple mechanical factors can usually be found; these include kneeling, bandaging, crossing the legs while sitting, lying on a hard surface, and the wearing of knee-pads. Previous loss of weight conduces to this type of damage.

Motor paralysis is often complete, but sensory loss is frequently absent and is never profound. The condition is always painless, and the onset sudden.

Complete recovery is common and partial recovery the rule.

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Messrs. Williams and Wilkins (Baltimore, U.S.A.) announce the forthcoming publication of two new journals. The *Journal of Histochemistry and Cytochemistry* is to commence publication in January. It is to appear bi-monthly at an annual subscription of $7 (£2 10s.), and will contain original papers relating to the development and application of histochemical methods, with occasional review articles covering important aspects of histochemistry. *Applied Microbiology*, an official publication of the Society of American Bacteriologists, will publish papers concerned with the application of microbiology to the fields of food, sanitation, agriculture, antibiptics, and other subjects concerning the use or control of animal and plant disease. The journal will appear bi-monthly, commencing in January, 1953, and the annual subscription is $7.50 (£2 15s.).

CHRISTMAS DISEASE

A CONDITION PREVIOUSLY MISTAKEN FOR HAEMOPHILIA

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Haemophilia is a severe bleeding disease of males with a sex-linked recessive inheritance. Laboratory tests show a prolonged whole-blood clotting-time and deficient conversion of prothrombin to thrombin during clotting. When clinical, genetic, and the usual laboratory features are all present the diagnosis of haemophilia is generally believed to be simple. In some families haemophilia arises suddenly with no previous history of the disease; the clotting-time may not be greatly prolonged. To establish a diagnosis in these less clearly defined cases a new technique was used extensively by Merskey (1950). This test depends on the fact that normal blood added to haemophilic blood in small proportions shortens the clotting-time of haemophilic blood, whereas the addition of haemophilic blood is ineffective. The normal blood contains a substance—the antihaemophilic globulin—which is lacking in haemophilia. Thus to confirm the diagnosis of haemophilia the blood of the patient to be tested is added to the blood of a known haemophilic patient and the shortening of the clotting-time is compared with that obtained with normal blood. If the blood contains the antihaemophilic globulin its addition will shorten the haemophilic clotting-time.

This test has led to the discovery that occasionally a mixture of blood samples from two apparently classical cases of haemophilia has a shorter clotting-time than that of either specimen separately. We have now found seven patients who by ordinary tests would be said to have haemophilia. When a small proportion of the blood or plasma of these patients is added to haemophilic blood or plasma the clotting-time is greatly reduced. Similar instances of this phenomenon were recorded by Pavlovsky (1947), Koller *et al.* (1950), Aggeler *et al.* (1952), Schulman and Smith (1952), and Poole (1952). From these observations it must be concluded that within the general group of patients thought to have haemophilia there are at least two different conditions. To avoid confusion it is essential at this stage to make a restricted definition of the term "haemophilia."

Antihaemophilic globulin can be shown to be essential for the normal formation of blood thromboplastin (Biggs, Douglas, and Macfarlane, 1953). Haemophilia may therefore be defined as a recessive, sex-linked