

AN ENQUIRY INTO THE PRODUCTION OF PERIPHERAL NEURITIS
BY PRESSURE DUE TO ACTION OR POSTURE.

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I N T R O D U C T I O N .

Interruption of the conductivity of a peripheral nerve by pressure, due to action or posture, is not an uncommon occurrence, but is a condition for which medical advice is relatively seldom sought.

In the majority of cases, so transient is the condition and so familiar is it to the sufferer, that if spoken of at all, it is lightly referred to as, "numbness in the fingers on waking," or, "the foot going to sleep." On occasions, however, the condition instead of rapidly passing off, persists for a considerable period of time, and it is then that the patient seeks medical advice, and the practitioner is presented with what may prove to be a puzzling clinical problem. The word "puzzling" is used advisedly since it is often difficult to detect the cause, which has produced in a limb an isolated area of motor or sensory disfunction.

An intriguing point in such a condition is, that although the patient himself is frequently the sole cause of damage to the nerve, yet he may have no knowledge, at the time, or later, of producing upon himself a nerve lesion, and it is only by careful questioning that the cause may be discovered.

It may appear almost incredible that a patient can indeed produce unknowingly upon himself a traumatic neuritis, but a consideration of the mechanism

whereby the integrity of the body is preserved will provide a clearer appreciation of the manner in which such a situation may arise.

The protective mechanism of the body may be divided into two parts, the anatomical or structural, and the physiological. With reference to the former, it can be said that the more delicate structures, which enter into the composition of the body, have been accorded positions in which they lie secure from injury unless subjected to extreme violence.

The brain and spinal cord constitute good examples. They serve vital and highly specialised functions, yet have little or no ability to withstand injury nor to regenerate when injured. The degree of structural protection afforded to them is unrivalled throughout the body. They owe this to the covering of unyielding bone, within which they lie, insulated from shocks by the meninges and cerebro-spinal fluid.

The peripheral nerves, although not provided with such impregnable protection, lie, for the most part, shielded from injury by their surroundings of muscular and fatty tissue.

Now at certain points, on the courses of various peripheral nerves, such insulation by resilient surrounding structures is deficient or completely lacking, and it is at these danger points that the nerves are liable to be injured either by compression or by repeated minor traumata. The insulation may be deficient internally, because the nerve, lying directly

upon the surface of a bone, is liable to be crushed against this hard background, by pressure, transmitted from without.

The terminal fibres of peripheral nerves, owing to the superficial positions, which they frequently occupy, are obviously more exposed to injury than the more deeply embedded proximal trunks. The points, at which the greatest danger of injury is present, are those in which a nerve not only lies superficial, but runs upon the surface of a bone. Typical examples of such superficial neuro-osseous relationships are to be found on the ulnar nerve, as it lies upon the posterior surface of the medial humeral epicondyle, and on the common peroneal nerve, as it winds round the neck of the fibula.

A further potential source of danger occurs at the points where a peripheral nerve pierces the substance of a muscle, or runs between two muscles, the direction of whose pull is other than the direction of the path pursued by the nerve itself. Here the nerve may be compressed and injured during violent or repeated muscular contraction. From these observations it will be realised that there are situations in the body where, as a result of deficient structural protection, nerve injury is liable to occur.

There is still the problem of how injury may be done to a nerve, outwith the knowledge of the patient.

Previous mention has been made of the physiological protective mechanism. It performs its function

by initiating voluntary or automatic movements, as in the "Withdrawal Reflex", which remove the body, or a part of it, away from danger of injury. Movements, following the reception of stimuli by the special sense organs, may be regarded as prophylactic, since they are initiated before injury has been done. In the absence of such movements, and as soon as the painful stimulus reaches the body surface, a protective movement will occur. It is obvious that a patient asleep, or rendered unconscious by injury or narcotics, will not only lack knowledge of the occurrence of nerve injury, but will also fail to perform any protective movement, unless the stimulus be of sufficient strength to reach his conscious mind, or unless the automatic protective reflexes are not too deeply depressed.

In addition to this type, it will be seen from the case records which follow, that ignorance of injury may be due to the patient's attention being distracted by some task or recreation. Again, familiarity with the discomfort produced by some posture or action, may in course of time render the patient oblivious to it.

In a few cases the production of traumatic neuritis appears to have been unaccompanied by pain.

The following selected cases, which I have observed personally, are illustrative of the subject matter of this thesis.

PRESSURE NEURITIS OF THE COMMON PERONEAL NERVE.CASE No. 1.R.G. Aged 37.

Three weeks prior to examination, the patient had suddenly developed weakness in the left ankle. Since then, whilst walking, he had been obliged to raise the left foot higher than normal in order to clear the ground with the toes, which otherwise dragged, and tended to trip him up. Upon the same evening, as the weakness in the ankle developed, he noticed, whilst in his bath, that the upper surface of the left foot and outer side of the left leg were numb.

The following day he had sought medical advice, and was undergoing treatment, consisting of intermittent Faradism to the paralysed muscles, for a period of fifteen minutes three times a week. He had already regained some degree of movement, and the numb feeling was present only in a small area of the skin overlying the base of his first and second toes.

Questioned as to his actions, immediately before he noticed the weakness in the ankle, he stated that he had spent some twenty minutes in a crouching position in front of a fire, to which he held a newspaper in an attempt to make it burn more brightly. Having accomplished this, he had sat for over two hours reading, during which time, as far as he can remember, he did not cross one leg over the other, a position which he seldom adopted.

The first indication, which he had that anything was amiss, occurred, when, having finished reading, he removed the soft slippers, which he had been wearing, and put on his shoes, with the intention of going for a walk. His attention was at once drawn to the "slapping" noise which his shoe made on the ground, and he then found that his left ankle had become weak. The numbness of the foot, as previously mentioned, he did not notice until later the same evening.

On examination, the power of dorsiflexion of the left foot and toes and of inversion and eversion of the foot was found to be present, though the movements were both weak and diminished in range. Diminution of sensation, to touch pain and temperature, was present over an area some two inches in length, extending from the metatarso-phalangeal joint of the first and second toes proximally upon the dorsum of the left foot.

The muscles affected were the tibialis anterior, the extensors digitorum longus and hallucis and the three peronei. There was no alteration in the electrical reactions.

Pressure, over the proximal part of the lateral border of the popliteal fossa, revealed some tenderness at the point where the common perineal nerve emerges from the medial side of the biceps tendon, and courses over the posterior surface of the lateral head of gastrocnemius.

Seven weeks later the power had completely returned to all muscles and there was no longer any sensory disturbance present, making the total duration of his disability ten weeks.

CASE No. 2J.McG. Aged 47.

Two days prior to my seeing him, the patient had developed a weakness in his right ankle. He knew the time of onset of the condition accurately, and could account for the action, which he considered to be the direct and immediate cause of the condition.

His statement was to the effect that, whilst repairing a faulty waste pump in the coal mine, where he was employed, he had been obliged to work in a confined space, in which it was impossible to alter his position for a period of almost an hour. His position, whilst carrying out the repair, was one frequently assumed by colliers, and necessitated squatting with one knee flexed to a right angle, the sole of the foot in contact with the ground, whilst the other knee was fully flexed, the heel raised clear of the ground and in contact with the buttock - virtually sitting on one heel. As soon as his task was completed, and he straightened up and started to walk, he found that he was lame, and that owing to weakness of his right ankle he was obliged to step high in order to clear the ground with his right toes.

He stated that he had been a martyr to indigestion for many years, and of late had been vomiting frequently and losing weight. He was, in fact, awaiting admission to hospital to undergo operation for the relief of a long-standing pyloric stenosis.

When asked to assume the position he had occupied, whilst working on the pump, it was observed that his right knee was the one which he fully flexed.

On examination, the patient was found to be a tall thin man, emaciated, and showing considerable pallor of his mucous membranes (R.B.C. 3,200,000. Hb 60%). Palpation of the right common peroneal nerve, as it emerged into the popliteal fossa from the medial side of the biceps tendon, produced pain. No thickening of the nerve could be felt. He had foot-drop on the right side, and the movements of dorsiflexion of the foot and first phalanges of the toes, and of inversion and eversion of the foot, were absent. There was no loss of electrical reaction in the muscles involved. A small area of diminished sensibility to light touch was present at the base of the first and second toes. Treatment consisted of the application of a right-angled posterior splint, to be worn at night to correct the dropped foot, and of daily massage to the paralysed muscles. Power of dorsiflexion of the foot was first observed in four weeks' time. The other movements, previously lost, all returned in a further fortnight. Use of the splint was discontinued five weeks from the onset of the condition.

CASE NO. 3.R.T. Aged 51.

The patient came for treatment, complaining of weakness in the left ankle, which caused his foot to drag as he walked.

His history of the onset of the condition was as follows. The previous evening he had worked in his garden laying an edging of bricks along a path, and on standing up, after completing a short section, he found that his ankle was affected in the manner of which he complained. He had never previously experienced any form of paralysis and was alarmed, fearing the occurrence of a minor "shock."

When asked to assume the position he had adopted, when working in his garden, he crouched down with his left knee fully flexed, the heel raised off the ground and in contact with his left buttock, whilst his right knee was flexed to a right angle, the foot resting flat upon the ground.

On examination he was seen to be a tall, thin man, in perfect health, save for a weakness of the anterior tibial group of muscles, producing complete inability to dorsiflex the left foot and toes. Slight eversion and inversion of the foot could however be performed. There was a small area of skin at the base of his first and second toes, in which the appreciation of light touch was diminished.

On palpation the left common peroneal nerve was found to be tender at the point where it crossed the dorsal surface of the head of the fibula.

All muscles responded well to Faradism. The patient was reassured that he had not suffered from a stroke, and it was explained to him that by adopting a crouching position, whilst working, he had compressed a nerve sufficiently to give a temporary degree of paralysis in the muscles, which it supplied.

He was treated by applications of intermittent Faradism to the anterior tibial group of muscles three times a week.

Voluntary movement of dorsiflexion of the foot reappeared at the end of six weeks. Treatment was discontinued two weeks later. When seen after a further two months, there were no signs of motor or sensory loss.

CASE No. 4.Miss E.W. Aged 17.

Four weeks prior to being seen for the first time the patient, previously a domestic servant, had obtained employment on a farm, and was set to work lifting potatoes. This entailed kneeling upon the ground for six to eight hours per day, whilst digging up the potatoes with a short handled fork.

Being unaccustomed to such work she found it fatiguing, and so had to rest frequently by sitting back on her heels and straightening her aching back.

At the end of her third day's work she found, while walking home, that her right foot felt numb and weak. She remembers that one of her fellow workers enquired as to the cause of her lameness, to which she answered, that her foot had "gone to sleep."

The following morning the numbness and weakness had disappeared, but after another day's work, both feet had "gone to sleep", and the next morning her right ankle, although no longer numb was weak, compelling her, as she walked, to raise her knee high to avoid catching her toes on the ground.

After four additional days, working upon her knees, both ankles were so weak and the feet so numb, that it was only with considerable difficulty, that she could walk at all. She consulted her doctor, who certified her as unfit for work, and kept her in bed for a fortnight, before sending her to hospital for investigation.

Examination showed her to be a small stout girl. She suffered from bilateral foot-drop, it being impossible for her to perform the movement of dorsiflexion of the feet, extension of the toes at the metatarso-phalangeal joints, or inversion or eversion of the feet. On both sides an area of anaesthesia, to light touch pain and temperature, was present over a narrow band, extending from the lower end of the outer surface of the tibia along the middle of the dorsal aspect of the foot to the base of the first and second toes. On either side of these bands of anaesthesia, were present areas half to one inch in width of diminished sensory acuity.

There was no alteration in the electrical reactions of any of the anterior tibial group of muscles save in the right tibialis anterior, in which the response to Galvanism was sluggish.

Tender areas were found on palpation of both the common peroneal nerves, where they emerged from beneath the biceps to run across the dorsal surface of the lateral head of gastrocnemius.

The patient was unable to attend hospital for a course of physio-therapy, nor could she obtain such treatment where she lived in the country. She was consequently fitted with a spring toe-brace to either boot and encouraged to go about as usual.

When seen again after an interval of three weeks, the sensory loss had completely disappeared. She was also able to extend slightly the metatarso-phalan-

geal joints, and with the aid of light pressure could dorsiflex the feet. A limited amount of inversion could be performed with the left foot.

In a further four weeks' time the power had returned to her ankles to an extent, sufficient to render unnecessary the further use of supports.

When seen again four weeks later, that is thirteen weeks from the date on which she had ceased working on the farm, all movements had returned to normal both as to range and strength.

CASE No. 5.Mrs J.B. Aged 29.

The patient was admitted to hospital, in order to undergo the operation of radical excision of a pilo-nidal sinus, which had caused her discomfort during the greater part of her life.

During the operation, which necessitated a more extensive dissection of the tissues overlying the posterior surface of the sacrum than was anticipated, the patient lay face downwards upon the table. The end panel was then lowered sufficiently to give flexion of approximately 140° at the lumbo-sacral joint, and to render the operative field more accessible. The edge of the panel reached only to three inches above the patient's knees, with the result that the weight of her legs and feet produced extreme extension of both knee joints.

The operation, as previously mentioned, took longer than was anticipated, the patient being on the table with the knees hyperextended for over forty minutes. On recovering from the anaesthetic she immediately complained of intense aching pains behind both knees, and of a weak feeling in both feet. The slightest movement of either knee-joint caused severe pain. It was found on examination that she was suffering from bilateral foot-drop. No loss of cutaneous sensibility in either legs or feet could be found on repeated examination, nor did she complain of any subjective sensory phenomena in the dis-

tribution of the common peroneal nerves. Treatment consisted of placing a soft bolster beneath the middle of both thighs, which kept the knees slightly flexed, and the popliteal fossae clear of the mattress. A firm pillow was placed at the feet to maintain the ankles at an angle of flexion of 90° .

The pains at the back of the knees soon diminished in intensity, and at the end of three days little discomfort on movement was complained of, save on full extension of the knee joints.

Daily massage to the paralysed muscles namely; tibialis anterior, flexors digitorum & hallucis longus, and the peronei was begun on the fourth day following operation. The patient was allowed up on the seventeenth day, there being at that time no signs of returning power in the anterior tibial group of muscles.

Slight voluntary dorsiflexion of the left foot could be performed at the end of five weeks. By this time the patient was up all day with light right-angled aluminium splints, applied posteriorly, to correct the dropped feet.

She was discharged from hospital in a further six days, by which time the right foot also could be voluntarily dorsiflexed, returning twice weekly for massage, and continuing to wear the aluminium splints. Two weeks later strength had so far returned to the anterior tibial group as to render unnecessary further wearing of the splints.

CASE No. 6.I.G. Aged 54.

This patient was seen for the first time five weeks before he died of inoperable gastric carcinoma. He was emaciated in the extreme, and had the cachectic pallor characteristic of advanced malignant disease. The amount of weight, which he had lost since the onset of his illness, was not known. Eighteen months before, according to his own statement, he was a robust, healthy man, from which it was concluded, that a very considerable amount of wasting must have occurred.

Examination disclosed a hard, palpable mass in the epigastrium. The lower edge of the liver, firm and irregular, could be palpated with ease two fingers' breadth below the right costal margin. Percussion showed free fluid to be present in the peritoneal cavity, but no actual fullness was apparent in either flank. (R.B.C. 3,100,000 per cu.mm. H.B. 55%. C.I. .9. W.B.C. 14,500 per cu.mm.)

Special attention was directed to those parts of the limbs supplied by the longer and more exposed peripheral nerve. Apart from the general aesthenia, no abnormality could be detected in either upper, nor in the right lower extremity. The left foot, however, was "dropped", all voluntary power of dorsiflexion, eversion, and inversion being lost. A small area of diminished cutaneous sensibility was found over the anterior surface of the lower end of the left tibia, and on the dorsum of the left foot. In this area points could be demonstrated of complete anaelgesia.

As previously mentioned, the patient was in an advanced state of emaciation. He was confined to bed, and was so weak that the effort of changing his position was too much for him. He lay consequently, for long periods, on the flat of his back, with his legs extended.

The bed which he occupied had a sagging mattress, with the result that his legs, owing to the loss of muscle tone and laxity of ligaments at the knee-joints, followed the contour of the bed, his knees adopting a mild degree of genu recurvatum. Palpation of the common peroneal nerves, on the posterior surface of the head of either fibula, revealed tenderness especially in the left.

A soft bolster was placed across the bed over which the knees were flexed. A pillow was also placed at the feet to maintain the ankles at an angle of 90° , and correct the foot-drop.

After a fortnight no improvement was manifest. I did not see the patient again, but his doctor informs me that up to the time of his death, two weeks later, the condition of the left leg and foot remained unchanged.

It is to be noted that no paralysis developed in the distribution of the right common peroneal nerve.

CASE No. 7.

Miss A.S. Aged 68.

The patient was admitted to hospital, suffering from an appendix abscess, which could actually be seen bulging in the right iliac fossa.

She was a frail little woman, and at the time of admission was seriously ill (T. 100.4 P. 114 R. 24). Vomiting had occurred on three occasions within the previous six hours.

An incision was immediately made into the abscess, under light gas, oxygen and ether anaesthesia; a copious amount of foul smelling pus evacuated, and a rubber dam drain inserted. The wound drained well, and at the end of 12 hours the temperature had returned to normal. Her progress was satisfactory, but the wound as might be expected in so elderly a subject, suffering from such a condition, was slow in healing.

When allowed up for the first time seven weeks after the operation, she was very unsteady on her legs, and said that her feet felt numb and dead.

On examination, she was found to have bilateral foot-drop with numbness on the dorsa of both feet, extending upwards on to the front of the legs, limited to the common peroneal distribution.

Her legs were thin and wasted, the popliteal concavities being obliterated, and the heads of the fibulae projecting prominently into them when the knees were extended. Palpation of the common peroneal nerves, in this situation, revealed tenderness at the point where they lay on the dorsa of the

heads of the fibulae.

As it was concluded that this unfortunate development had been occasioned by pressure upon the common peroneal nerves between the heads of the fibulae and the mattress, while the patient lay in bed, the following treatment was carried out:- A bolster was placed across the bed beneath the middle of both thighs. This maintained flexion at the knees, and not only relieved tension on the affected nerves, but also kept the popliteal fossae clear of the bed, thus avoiding further nerve compression. A firm pillow was placed at the foot of the bed to maintain dorsi-flexion of the ankles to a right angle. Daily massage of her legs was instituted, and passive movements regularly carried out by the nursing staff. At the end of a fortnight she complained of prickly feelings on the dorsa of her feet. The numbness was then found to have shrunk to an area, situated on the dorsa of the feet, at the base of the first and second toes.

In eight weeks, definite voluntary movements of dorsi-flexion could be performed with either foot, and with a little assistance she could walk a few steps. From then on her progress was rapid, and at the time of her discharge from hospital she could walk well with the aid of a stick, although the anterior tibial groups were still weak.

A letter received from the patient fourteen months later, stated that she was in excellent health, and that her ankles were quite strong again.

CASE No. 8.

A.E. Aged 61.

The patient is a lorry driver by profession. Ten weeks prior to being seen he found, that his right ankle had become weak. The manner, in which his attention was drawn to this disability, is worthy of note.

He had started up his lorry as usual and was driving it out of the garage when, on wishing to reduce speed, in order to negotiate a corner, he found that his foot "would not come off the accelerator pedal." With some presence of mind, he drew back his leg by bending the knee, and so regained control of his vehicle. He got out and tried to move his right foot about, but found that it hung down limply, although he was still able to raise himself upon his right toes.

He continued driving, making the necessary movements from the knee, and using the hand throttle instead of the foot as much as possible. He had no pain or numbness in his right foot or leg.

The same evening he consulted his doctor, who recommended him to a neurologist, by whom the condition was diagnosed as traumatic neuritis of the common peroneal nerve.

On palpation the common peroneal nerve was tender, and could be felt as a firm fibrous cord, as it lay upon the outer side of the leg immediately inferior to the head of the right fibula. The left common peroneal nerve was a more slender

structure and neither indurated nor tender. Treatment consisted of applying a thick cotton-wool pad over the outer side of the right leg below the knee, in order to prevent further traumatisation of the common peroneal nerve. Massage and Faradism to the paralysed muscles was also instituted three times a week.

On being questioned, the patient remembered, that whilst driving, he habitually steadied his right leg by resting it against the top of the gear lever, which on his lorry, consisted of a round wooden ball. He also stated, that there was considerable vibration of the gear lever, whilst the engine was running. The day previous to the onset of the paralysis he had driven an unusually long distance of over eighty miles. The day had been cold and he had suffered discomfort from draughts, coming up through the floor of the driver's cabin.

On examination the patient was found to be a thin little man. Both radial and brachial arteries were thickened. His B.P. was $\frac{170}{110}$. He was able to make slight voluntary movements of dorsiflexion and of inversion and eversion of his right foot. The range of these movements was small, and the power slight. No sensory loss could be discovered on his right leg or foot.

When seen four weeks later the strength and range of movement had increased considerably. In a further four weeks the movements were so good, that

massage and electrical treatment were discontinued. He was advised to continue wearing the pad on the outer side of his leg when driving.

PRESSURE NEURITIS OF THE RADIAL NERVE.CASE No. 9L.F. Aged 50.

Eight weeks prior to being seen, the patient, whose occupation is that of a house painter, fell from the ladder on which he was standing, and sustained a fracture of his left os calcias. He was removed to hospital, where he was detained for seventeen days. At the end of that time, he was discharged with his left leg and foot encased in plaster of paris, and advised to move about with the aid of a pair of crutches.

At the end of a fortnight, during which time he made full use of the crutches, he found that his left arm, forearm and hand were becoming weak. He was also troubled by a feeling of "pins and needles" on the back of the forearm and thumb. When questioned, he recalled that this sensation usually occurred after he had been using the crutches.

The weakness increased and gradually the prickly sensation disappeared, giving place to a feeling of numbness. He was still able to grip the bar of the crutch, and continued to walk about as before.

On examination, the patient was found to be a short, stout man. He was considerably over weight and had small atrophic muscles. There was a complete flaccid paralysis of the extensors of the forearm, carpus and first phalanges of the fingers. There was an area of anaesthesia to light touch, pain, and temperature on the dorsum of the first phalanx

of the thumb and index finger, and on an area running proximally therefrom for some two inches onto the dorsum of the forearm. None of the paralysed muscles showed alteration of response to Faradism.

He was advised to discontinue the use of the crutches. A "cock-up" splint was applied to correct the dropped wrist, the arm was supported in a sling, and intermittent Faradism and massage were given three times a week.

Movements of extension at the elbow could be performed in four weeks, by which time only a small area of diminished cutaneous sensibility remained on the dorsum of the first phalanx of the thumb. Slight movements could be performed by the extensors of the carpus and fingers at the end of a further fortnight, when all sensory disturbance had vanished. Contraction of the brachio-radialis could be felt for the first time at the end of a further week, slight movements of supination also being permissible. After a further week's treatment, making nine weeks in all, the patient was discharged, all voluntary movements having returned.

CASE No. 10J.D. Aged 36.

This patient was seen, whilst attending the Massage Department, of the Royal Infirmary, Edinburgh, where he was undergoing treatment for paralysis of all muscles, supplied by the left radial nerve.

His history is as follows. He was involved in a mining accident, sustaining a compound fracture of both bones of the left leg. Apart from some bruising of the right leg he received no other injury.

He was immediately removed to hospital, where the fracture was reduced. The wound remained healthy, alignment of the ends of the bone was good, and at the end of three weeks he was allowed up on crutches, with his leg and foot in a plaster of paris case. After a further week in hospital he was allowed to go home, and encouraged to go about as much as possible on his crutches.

A few days after his return home he found that his left arm and hand were becoming weak, and he had from time to time tingling sensations on the outer side of his left forearm and hand. He took little notice of this and finding himself adept on his crutches, attended a football match as a spectator, which necessitated standing, no doubt leaning on his crutches, for over two hours. When the time came to leave the ground, he found that his left arm was practically powerless, and it was only with considerable difficulty that he walked back to his house.

He reported to hospital the next day, and was at once referred to the Massage Department as a "crutch" radial palsy. I saw him two days later, when examination revealed the following facts.

The patient was of medium size. His muscles were small for a man, following the arduous calling of a collier, and - doubtless owing to his enforced idleness during the previous six weeks - of poor tone.

There was complete loss of power of voluntary movement in the left triceps and aconeous and of all muscles in the left forearm, supplied by the radial nerve. Over a small area on the radial border of the left hand, including the proximal phalanges of the thumb and first finger, there was impairment of sensibility to light touch, pain and temperature.

Treatment consisted of supporting the arm in a sling, the dropped wrist being maintained in a dorsiflexed position by a "cock-up" splint. Passive movements, massage and intermittent Faradism were given daily to the paralysed muscles. The patient was also encouraged to remove the sling and splint and carry out passive movements, on his own, night and morning.

The first sign of recovery appeared at the end of three weeks, when the extensors of the wrist could be felt to contract voluntarily, although no movement of the wrist was perceptible. At the end of a further week slight voluntary movements at the elbow, wrist and knuckles were present.

Progress from then on was rapid, and recovery, to the extent of being able to perform movements with all the previously paralysed muscles, took place in a further fortnight - seven weeks in all from the onset of the complete radial palsy.

CASE No. 11.B.L. Aged 43.

During the course of his employment as a furniture remover, the patient had occasion to carry upon his back, down three long flights of stairs, a heavy wooden packing case. The manner in which he performed this task was to support the box on his shoulders, with his body bent forwards, and to steady it there by gripping the front of the upper edge with both hands.

When he had descended half-way, he realised that the weight of the box was too much for him, yet by a great effort he was able to maintain his grip, until he reached the foot of the stairs, and obtained assistance in lowering the box to the ground. He dared not drop it, lest he broke the china, which it contained.

As soon as he was freed from his burden, he found that his right wrist had become weak, and that his hand hung down limply. He was unable to straighten either the wrist or the fingers at the knuckle joints.

He had suffered the misfortune three weeks previously, of cutting the little finger of his right hand. The wound had become septic, and at the time of the accident to his wrist, the inner side of his hand was red and swollen, occasioning him considerable pain. The major part of the grip with his right hand was consequently confined to the thumb and first two fingers.

On examination, two days after the onset of the paralysis, the patient was seen to be a small, under-nourished man. His muscles were small but firm. The wrist and metacarpo-phalangeal joints were flexed, and he was unable to extend them. The flexion was limp and easily reduced. The grip of the right hand was weaker than that of the left, but became stronger, when the wrist was passively dorsi-flexed. Full flexion of the fingers at the interphalangeal joints was possible after they had been fully flexed passively at the metacarpo-phalangeal joints. Abduction and extension of the thumb were lost, the patient being unable fully to flex the fingers, until the thumb had been passively abducted out of their way. On flexing the forearm against resistance, the brachio-radialis was not felt to contract. There was no loss of power in the triceps. Supination was carried out merely by the biceps during flexion of the arm. There was no loss of power of pronation.

Sensibility to light touch, pain and temperature was impaired over the dorsum of the proximal phalanx of the thumb and index finger, and over the dorsum of the distal end of the left radius. Pressure over the radial nerve towards the outer side of the dorsum of the right arm caused pain, and for that reason sufficient pressure could not be exerted in order to palpate the nerve.

Treatment consisted of the application of a "cock-up" splint, with massage and intermittent Faradism to the paralysed muscles for periods of ten

minutes each, three times a week.

Complete recovery took place in thirteen weeks, extension of the wrist being the first movement to return. This was first seen at the end of five weeks. The sensory disturbance had disappeared completely at the end of four weeks. The last movement, to recover fully, was extension of the index finger.

CASE No. 12.A.F. Aged 34.

The patient, a muscular man of excellent physique, gave a history of having been involved in an accident during the course of his employment, as a result of which, he had been rendered unconscious by a blow on the head, and had lost the power of extending his right wrist and knuckles.

Details of the accident were as follows. The patient was employed as a labourer in a saw-mill, to which logs were slid down a chute from the roadway above. The chute was some thirty feet in length, and fitted with rough wooden sides, varying in height from six inches to two feet. From time to time a log became wedged crosswise in its descent, obstructing the passage of those coming behind, thus causing a jam, which had to be cleared by hand.

On the day of the accident, the last few logs of a load being slid down the chute became fixed in the manner described, and the patient had descended the chute from above in order to clear the obstruction. He raised the ends of the uppermost logs one by one, and then with a strong push tipped them over those lying in front. He had up-ended a heavy log, and was about to tip it over the obstruction, when, his foot slipping, he fell with the log on top of him, receiving from it a blow on the head, which rendered him unconscious.

He was unable accurately to state for what length of time he lay, but was emphatic that it was for a period of not less than ten minutes. The noise, created by the engine and saws, drowned the sound of his fall, which would have brought a fellow worker to his aid.

On recovering consciousness he found himself lying face downwards upon the shute, and was immediately aware of an aching pain in his right arm, just above the elbow, where it rested across the raised edge, the remainder of his arm hanging down over the side. He succeeded in climbing up to the roadway where, after vomiting, he again lost consciousness for a short time. He was transferred to hospital and was detained for observation for four days.

Reference to his hospital record shows, that on admission he was suffering from a lacerated contusion above the left eye, with gross extravation of blood into the tissues surrounding the orbit. No clinical nor radiological signs of fracture of the skull were present. Paralysis of all forearm muscles, supplied by the right radial nerve, was present. There is no mention of sensory loss.

The patient made no complaint of pain in the right arm at the point, where it had rested across the raised edge of the shute. In view of the radial nerve lesion the right arm was X-rayed, but no injury to bone was present.

As the patient developed no immediate after-effects of his head injury, he was discharged from hospital in four days, the dropped wrist being supported by a "cock-up" splint. He was instructed to remove the splint three times a day, and to carry out passive movements of his wrist and fingers, and to massage his forearm muscles.

He reported a fortnight after discharge from hospital, when slight power of dorsiflexion and supination of the wrist were seen to have returned. The fingers still remained, if unsupported, limply flexed at the metacarpo-phalangeal joints.

He did not again return to hospital, but when interviewed, stated that his hand soon functioned again, and that he gave up wearing the splint and returned to work six weeks after the accident. He stated also that within three months of the accident his hand was as strong as ever. He has had no subsequent return of the radial palsy.

CASE No. 13J.B. Aged 52.

The patient states that on New Year's Day he awoke to find that he had slept the night in a chair, in which, to the best of his knowledge, he had rested for five or six hours.

The previous evening he had participated in the convivialities of a Scottish hogmanay night with the result that his sleep had been deep and untroubled. On his own admission, his condition was such, that in all probability he did not alter his position from the moment of falling asleep until he awoke. The chair, in which he had passed the night, had high arm-rests devoid of padding, and over its left arm his own left arm had hung, while he slept.

At first he felt sore, stiff, and cold, the inner side of his left arm above the elbow aching considerably. He stretched himself and rubbed the aching part of his left arm, when he found that his left wrist was weak and hung down uselessly. He was unable to straighten it, nor could he extend his fingers. He massaged his arm and wrist hoping to remove what he assumed to be a temporary stiffness, but without avail. Later he tried other remedies, such as alternately plunging his hand into hot and cold water, without achieving any better result.

He continued his work as a motor mechanic for three days, but, finding that the weakness of the left wrist caused him considerable inconvenience, and be-

coming worried by the condition which, by its persistence, showed itself to be graver than he had at first allowed himself to believe, he came for medical advice.

On examination he was found to be a thin, wiry man, whose general health was good. Routine examination of the C.N.S. revealed no abnormality, save a paralysis of the muscles of the left forearm supplied by the radial nerve. Pressure over the radial nerve, on the inner side of the left arm three inches above the elbow, revealed considerable tenderness. There was no loss of power of the left brachio-radialis muscle, but there was complete loss of voluntary motor power in all muscles of the forearm, supplied by the left radial nerve. No loss of response to Faradism was present in any of the paralysed muscles. No alteration of sensation could be detected in the forearm or hand, but, on being questioned, the patient recalled that, at the time when he was massaging his wrist and hand immediately after the discovery of the palsy, he had experienced the sensation of "pins and needles" down the outer side of his forearm and hand, which passed off quickly.

Owing to his hours of employment, he could not attend hospital for treatment. He was consequently fitted with a light aluminium "cock-up" splint which, whilst correcting the dropped wrist, enabled him to perform limited movements with the fingers and thumb. He was advised to remove the splint night and morning,

and with his other hand carry out full passive movements of supination and extension of the wrist and extension of the fingers and thumb on the left side.

At the end of five weeks, some slight improvement was apparent, a limited amount of dorsiflexion being possible. The recovery of all movements, to such an extent as to render superfluous further wearing of the splint, occurred after a further three weeks - eight weeks in all from the onset of the paralysis.

CASE No. 14.J.F. Aged 29.

This patient came for treatment, complaining that his right wrist was useless and "fell away", when he attempted to use it. Examination showed him to be suffering from wrist-drop, and that an area of hypoaesthesia was present on the dorsum of the first phalanx of his right thumb, extending proximally for a distance of three inches on to the skin overlying the lateral border of his right radius. When asked to flex his elbow against resistance, the right brachio-radialis was felt to contract.

He volunteered the following statement: Two days previously he had imbibed an excessive amount of alcohol, and, whilst making his way home, had the misfortune to fall down a flight of steps leading to the basement of a house. Beyond a few bruises he did not injure himself seriously, but the fright, combined with the effects of the alcohol, rendered him so confused that the proprietor of the house, who had been attracted by the noise, dissuaded him from continuing his journey immediately, and kindly invited him inside to rest.

The patient spent two hours in the house, sleeping in an armchair. On waking he found that his right arm had been hanging over the side of the chair, and he experienced a numb feeling on the back of his hand and forearm. In attempting to raise himself, he found that his wrist had become weak.

Treatment was instituted at once, consisting of intermittent Faradism and massage, the wrist being supported in a position of dorsiflexion by means of a "cock-up" splint. At the end of ten days he complained of a sensation of "pins and needles" in the area, which previously had been numb. This wore off in a matter of seven days, normal cutaneous sensibility being restored.

Return of voluntary movement was first apparent in the extensors of the carpus at the end of twenty-eight days. Power of extension of the digits at the metacarpo-phalangeal joints returned in a further ten days.

The patient was discharged six weeks after the onset of the paralysis, at which time all voluntary movements had been restored, but were diminished both in strength and range.

CASE No. 15W.D. Aged 34.

The patient, who was employed as a mechanic in a woollen mill, was involved in an accident during the course of his employment, as a result of which he received numerous lacerated puncture wounds of his right arm accompanied by a dropping of the right wrist and fingers.

Details of the accident are as follows: He was lubricating the bearings of the machine, into which the raw wool is fed at the beginning of the manufacturing process. The machine resembles an ordinary domestic mangle, save that the upper roller is armed with spikes.

The machine was running at the time, and, having raised the safety guard, a procedure which he admits was not devoid of risk, his hand slipped between the rollers, and immediately his forearm and arm were drawn into the machine. His shoulder and trunk prevented his arm being drawn in further than the junction of its upper and lower halves, and the machine, being driven by a belt, came to rest owing to the belt slipping. With commendable presence of mind he managed to slip the belt off the driving drum with his free hand and thus stopped the tugging on his arm. He was however, still held a prisoner between the rollers, which exerted considerable pressure on his arm.

Liberation of his arm by rotation of the rollers in the reverse direction would have produced further lacerations from the spikes. It was necessary

therefore, partially to dismantle the machine in order to raise the upper roller clear of his arm. This manoeuvre took over twenty minutes, by which time, he states, he had lost all feeling in his forearm and hand. On being liberated his arm, distal to the point where it had been caught, was numb and powerless.

Medical attention was directed primarily to the lacerated puncture wounds, fourteen in number, situated on the dorsum of his right hand and forearm and on the outer side of the distal half of his arm. The wounds healed satisfactorily, his arm being immobilised in a splint for ten days.

On attempting to move his arm at the end of that time, he found that the wrist and fingers hung down limply and that he was unable to straighten them. On examination, paralysis of the extensor group of muscles of the right forearm supplied by the radial nerve was present, the right brachio-radialis also being paralysed.

The question which then arose was, had the radial nerve been injured merely by pressure, or had it been severed by one of the spikes, which had so extensively lacerated his arm? At the end of a fortnight all muscles continued to respond well to Faradism, and it was concluded that solution of the continuity of the nerve had not taken place.

A "cock-up" splint was applied, and massage and Faradism were started a fortnight after the accident, which was as soon as the wounds would permit.

In six week's time after the accident weak Dorsiflexion of the wrist was possible. In a further fortnight slight movements of extension at the metacarpophalangeal joints were also possible. Three months after the accident power had so far returned to the arm, that the patient was able to resume work.

CASE No. 16.

Miss I. McG. Aged 19.

The patient, a medical student, sought advice on account of weakness in the fingers and thumb of her right hand. Her attention had first been drawn to the condition whilst playing tennis four months previously, when she experienced difficulty in playing the back-hand stroke, owing to weakness in extending her wrist.

Two months prior to coming for advice she had taken up rowing as a pastime, since when, she had observed a progressive weakness in the fingers of her right hand. At first she was unable to extend the fifth finger, then the fourth, and finally, after a longer outing in the boat than usual, she was unable to extend any of the fingers or the thumb. At no time had she experienced numbness or tingling in the fingers or thumb.

On examination it was found, that active extension of the fingers at the metacarpo-phalangeal joints and extension and abduction of the thumb, were weak and limited in range. On active extension of the wrist the extensor carpi ulnaris was not felt to contract with the same degree of firmness as its neighbour on the opposite side. No muscle wasting was present, the right forearm being actually 2 cm. greater in circumference than the left at the junction of its upper and middle thirds. All muscles responded well to Faradism. No tenderness could be discovered on palpation of the dorsal interosseous

branch of the radial nerve, by which all the muscles, showing partial paralysis, were supplied.

She was advised to cease rowing, which from the history appeared to be the cause of the palsy, restricted to the dorsal interosseous nerve.

When seen six weeks later, power of extension had returned completely to the thumb and first two fingers; the fourth and fifth finger still showed some weakness of extension. In a further two months all muscles, previously weakened, had regained their normal strength.

PRESSURE NEURITIS OF THE MEDIAN NERVE.CASE No. 17.Mrs A. G. Aged 48.

The patient, a healthy middle-aged woman, was involved in a motoring accident, as a result of which she suffered from total paralysis and sensory loss in the distribution of the right median nerve.

The manner, in which she was injured, is as follows. She was a passenger in a small open charabanc, which, following a collision with a private car, left the road and running into a ditch, overturned. The final movement of overturning, as the charabanc settled upside down, was slow. The driver and passengers were thrown clear, escaping with minor injuries, with the exception of the patient, who was pinned beneath the vehicle - the edge of its body resting across the middle of her right arm. Owing to the deliberate manner, in which the charabanc finally came to rest, a steady pressure rather than a sudden impact was applied to her arm, but was sufficient to hold her so firmly that she was unable to escape. She lay parallel with the side of the overturned charabanc, her arm at right angles to her body, the palm of her hand facing upwards.

Fortunately, the ground on which she lay was soft, so that her arm sank into it, easing a little of the pressure, which notwithstanding was very considerable.

Attempts to free her arm, by excavation of the ground beneath it were unsuccessful, and she was

held a prisoner for over half an hour before sufficient help to raise the charabanc could be obtained.

She states that so sudden was the occurrence, that at first she felt no pain, but as time went on she experienced considerable pain, not only where her arm was held, but also down the front of her forearm and hand. Later the pain in the forearm and hand gave place to numbness. When released her arm and hand, distal to the site of injury, were swollen, bluish-red in colour, and numb. The swelling soon disappeared from her forearm and hand.

She was removed to hospital, where she was found to be suffering from a severe contusion two inches in breadth, situated on the inner side of the middle of her right arm. No injury to bone could be demonstrated radiologically. A total paralysis in the distribution of the median nerve distal to the site of injury, accompanied by typical sensory loss, was present.

At the end of a week when the bruising had started to subside, a course of massage and Faradism to the paralysed muscles was started. At that time the following muscles were completely paralysed, the two pronators, flexores carpi radialis, digitorum sublimis and the radial half of profundus. The radial lumbricals, opponens, adductor brevis and outer head of flexor brevis hallucis, also showed loss of motor power. All muscles responded briskly to Faradism. After five weeks's treatment the electrical reactions

remained unaltered, and in view of the long disability usually associated with injuries of the median nerve, a most satisfying degree of recovery was present. Movements of pronation of the forearm were possible though weak and limited in range. When the wrist was flexed actively, the flexor carpi radialis could be felt to contract, although its tendon could not be felt to stand out with the same firmness as that of its neighbour in the opposite arm. Flexion of the fingers was well performed, except for the movement of flexion of the first two fingers at the metacarpophalangeal joints. Adduction of the thumb, flexion of its terminal phalanx, and opposition of it to the other fingers could not yet be performed. There was still diminution of appreciation to light touch on the volar surface of the terminal phalanges of the first two fingers, cutaneous sensation being otherwise normal.

In a further four weeks movement had returned to all intrinsic muscles of the hand, and no sensory upset was present. When seen again four months after the accident there was still present some slight residual weakness.

CASE No. 18.

J.B. Aged 35.

The patient was sent for treatment on account of a numbness in the tips of the first three fingers of his right hand. He had first noticed the numbness six weeks previously, and stated that it seemed to be slowly spreading upwards into his hand.

Examination showed loss of cutaneous sensibility to light touch, pain and temperature on the volar surfaces of the terminal phalanges of the first three fingers. There was no loss of motor power in the forearm, hand or fingers. Routine examination revealed no evidence of other abnormality in the nervous system beyond this limited area of hypo-aesthesia in the distribution of the right median nerve.

Interrogation elicited the following facts: The patient was employed as a lorry driver, and he habitually drove with his right hand high on the wheel, his wrist resting across a spoke. He never wore gloves, and during the cold weather of the previous two months, his hands had frequently become so numbed, that he temporarily lost all feeling in them.

It was explained to him that the probably cause of his condition was injury to the small nerves running to his finger, produced by the constant pressure on his wrist of the vibrating wheel spoke, whilst his hands and wrists were cold.

He was advised to wear thick leather gloves for warmth and protection, and whilst driving to hold the wheel in a manner, which would avoid pressure on his wrist.

He reported four months later, when no sensory loss could be discovered in his finger tips. He said, that acting upon the advice, which he had received, he had soon lost the numbness in the fingers.

Further discussion drew from him the information, that about three weeks after wearing the gloves he had experienced occasional tinglings in his fingers, but which, as far as he could remember, had only lasted a day or two.

CASE No. 19Mrs M.H. Aged 57.

The patient came for treatment on account of an injury to her right wrist, which she had sustained in attempting to save herself, as she fell down a short flight of steps.

On examination she was found to be suffering from a typical Colles fracture of both bones of her right forearm. The fracture having been reduced under light anaesthesia, a Carr's and a dorsal splint were applied. An X-Ray showed good position of the fragments.

The following day the patient complained of a tingling sensation on the outer side of the palm of her right hand, and in the first two fingers. No congestion, indicative of circulatory interference, was present in the fingers, and it was considered inadvisable to alter the position of the splints, as to do so might upset the alignment of the bones. On the fifth day, when the splints were removed, the patient stated that the tips of her first two fingers felt dead, and that she still had tingling in the proximal parts of her fingers and outer side of the palm of her right hand.

Examination revealed the fact that total anaesthesia to light touch, pain and temperature was present on the volar surfaces of the terminal phalanges of the index and second fingers. Diminution of sensation was present over the volar surfaces of the proximal phalanges of the same two fingers, and ex-

tended for a short distance on to the outer side of the palm.

Special care was taken in applying the plaster bandages to avoid pressure upon the superficial branches of the median nerve at the wrist.

Recovery took place with a most satisfying degree of rapidity. Tingling was present in the tips of the first two fingers at the end of a week, persisting for about ten days, when it gradually subsided, normal sensation being restored. Synchronous with the onset of paraesthesia, the scratch of a needle could be felt, on the previously analgestic finger tips.

At the end of three weeks from the removal of the Carr's splint no areas of abnormal cutaneous sensibility could be found on the right hand or fingers.



PRESSURE NEURITIS OF THE ULNAR NERVE.CASE No. 20.J.P. Aged 51.

The patient, a heavy obese man, was admitted to hospital suffering from an extensive cellulitis of the right side of the neck, the site of multiple shrapnel wounds received during the war. He was dangerously ill, being in a semi-comatose condition, his temperature swinging between 102° and 104°F. As no immediate surgical interference was indicated, conservative measures were employed, consisting of the application of mag. sulph. and glycerine fomentations to the affected part, three-hourly.

On the morning of his fourth day in hospital there was definite evidence of pus formation, and an abscess, which was beginning to point near the middle of the anterior border of the right sterno-mastoid, was opened, a considerable quantity of pus evacuated and a drain inserted. Drainage of the wound was satisfactory. In twenty-four hours the temperature had returned to normal, and the patient had so far recovered as to be able to feed himself. He complained then of a numbness on the inner border of both hands, accompanied by a feeling of stiffness in the ring and little finger of either hand.

Examination showed diminution of the appreciation of light touch, pain, and temperature on the ulnar borders of both hands, and on both little fingers. The grip with the thumb and first two fingers of both hands was good. There was slight weakness in the power of flexion of both ring and little

fingers. Palpation along the course of both ulnar nerves revealed tender points, situated half an inch distal to the medial humeral epicondyles.

As previously mentioned, during the first four days in hospital the patient was seriously ill and lay inert, oblivious to his surroundings. For the greater part of the time he lay flat on his back unable to move his head, owing to the painful and swollen condition of his neck, with his hands loosely clasped across his abdomen, and his elbows resting upon the bed on either side. His arms were well covered with muscle and fat, and heavy.

The conclusion arrived at was, that owing to his elbows being firmly pressed into the mattress by the weight of his arms for long periods at a time, both ulnar nerves had been compressed to an extent sufficient to interfere with their normal conductivity.

Thick woollen pads were applied to his elbows to protect the ulnar nerves against further trauma, and the nature and cause of the condition having been explained to the patient, his co-operation was enlisted to avoid further pressure upon his elbows. A Balkan frame was placed over his bed with the chain and handle within easy reach, so that he might move himself in bed without having to lever himself up on his elbows.

At the end of twenty-four hours he complained of a sensation of "pins and needles" in the inner border of both hands. This rapidly passed off and normal cutaneous sensation was restored. The

"stiffness" complained of in his fingers also disappeared, though less rapidly, four days having elapsed before all objective and subjective signs completely disappeared.

CASE No. 21.Miss M.McM. Aged 37.

This patient sought medical attention on account of a tingling sensation, which she experienced on the inner border of her right hand and in her right ring and little fingers. She also complained of some loss of power in the grasp of her right hand, which weakness was especially marked in the two innermost fingers.

These phenomena had been present for upwards of three weeks, and at first she had paid little attention to them. They persisted however, and becoming daily more troublesome -the tingling at times giving place to numbness and the grip becoming weaker -forced themselves upon her attention in a manner, sufficient to give rise to alarm and to cause her to seek medical advice. Her health had always been robust and she was unable to name any cause, which might account for the condition of her right hand.

A routine physical examination, with special attention directed towards the central nervous system, revealed no abnormality beyond a weakening of those muscles supplied by the right ulnar nerve, and a diminution in the appreciation of light touch along the inner border of her right hand, little finger, and to a lesser degree, on the inner border of her ring finger. Thus, when both wrists were flexed against resistance, the tendon of the right flexor carpi ulnaris was not felt to stand out with the same firmness as that of its neighbour on the opposite side.

Flexion of the right ring and little fingers was weak, nor could full flexion be produced whilst the index and middle fingers were extended. The power of gripping a flat piece of paper between the right thumb and index finger was less than that of the left hand, indicative of some slight loss of power in the adductor pollicis and inner head of the flexor pollicis brevis muscles.

Having thus ascertained that an isolated ulnar palsy was present on the right side, that other manifestations of nervous disease were absent, and that the condition had existed for three weeks and was becoming progressively worse, an attempt by means of careful questioning was made in order to discover some cause for the condition. The following facts were elicited. The patient admitted that the tingling and weakness of her right hand were worst in the morning on wakening. She also stated that she usually slept upon her face, her head resting upon the back of her right hand. Her employment was that of a biscuit packer and, in coping with the Christmas rush, her work had been heavier and her hours longer than normal, with the result that she was unusually tired at night and slept heavily. The previous month she had changed her lodgings, and readily volunteered the information, that the bed in which she now slept was harder than that to which she had been accustomed, but being a sound sleeper this discomfort had not inconvenienced her.

From these facts it was concluded, that the condition from which she suffered was that of a mild traumatic neuritis, brought on by pressure on her ulnar nerve between the point of her elbow and the firm mattress during sleep. This was confirmed subsequently when the patient, acting upon advice received, protected the ulnar nerve nightly by means of a thick cotton-wool pad, the sensory manifestation clearing up in under a week, and the palsy inside four weeks.

TOURNIQUET PARALYSIS OF THE UPPER LIMB.CASE No. 22.Miss B.M. Aged 33.

This patient had been troubled for a number of years by a carpal ganglion, situated over the head of the second metacarpal on the dorsum of her left hand. It was unsightly, and from time to time became inflamed and tender. Excision of the ganglion was decided upon, and the operation successfully performed. Prior to starting the dissection a tourniquet was applied to the left arm two inches above the elbow, and remained in position for approximately fifteen minutes.

On recovering from the anaesthetic, the patient found that her left forearm and hand were numb and powerless. The following day she experienced painful tingling sensations in the parts previously numb. This continued for three days, steadily decreasing in intensity, at the end of which time normal sensation was completely restored. She was still unable to make any voluntary movement with her left hand or fingers.

Examination revealed complete flaccid paralysis of all muscles in the forearm and hand, including the brachio-radialis. On the sixth day following the operation, slight voluntary movements could be performed by the flexors of the carpus and digits, supplied by the median nerve, and in a further three days the ulnar palsy, previously present, was seen to be recovering. There yet remained the typical dropped

wrist, which persisted for six weeks after the operation before any signs of improvement were discernable.

Treatment consisted of supporting the dropped wrist on a "cock-up" splint. Massage and intermittent Faradism were given to the paralysed muscles, starting ten days after the operation, and being continued for eight weeks. When seen three months after operation all voluntary muscular power was completely restored.

CASE No. 23.G.S. Aged 16.

The patient, an obese youth with small atonic muscles, was seen whilst attending the Massage Department of the Royal Infirmary, Edinburgh, undergoing treatment for a tourniquet paralysis of his right forearm and hand.

The tourniquet had been applied one inch below the middle of the upper arm, and had remained in position for approximately thirty-five minutes, during which time the head of his right radius was excised. The operation was necessary because the head of the bone had been detached from the shaft in an accident two years previously, and had failed to unite. This had produced limitation of extension of the elbow, and during movements of the elbow there was an audible and painful clicking. Prior to operation the limitation of movement of the elbow was the only disability present, there being no weakness of the wrist or hand.

On recovering from the anaesthetic the patient was aware of a painful tingling sensation in his right forearm and hand. The pain at the site of operation however, overshadowed all other sensations, and as far as he could remember the tingling had disappeared by the following morning.

His arm was immobilised in an extension splint for five days. When the splint was removed he was found to have a total flaccid paralysis of the muscles of the right forearm and hand, including the brachio-

radialis. All muscles responded briskly to Faradism. A course of massage, movements, and electricity was begun, the treatment being given daily for the first three weeks, by which time he was able to perform weak and limited movements of pronation of the forearm, and flexion of the wrist, fingers and thumb. At the end of a month from the date of operation, the patient was discharged from hospital with his wrist and fingers supported on a "cock-up" splint. He reported three times a week for massage treatment.

In a further month, the range and power of movements of flexion in the wrist and fingers, and of pronation had increased considerably, and he was then able slightly to dorsiflex the wrist, and to extend the fingers and thumb voluntarily. The elbow could now be fully extended and without pain or clicking. Massage was continued for a further month.

When seen four months after operation the functional result at the elbow was excellent, and all signs of the tourniquet paralysis had disappeared.

PRESSURE NEURITIS OF THE LONG THORACIC NERVE.CASE No. 24.W.E. Aged 21.

The patient, a tall slender youth, was seen whilst attending the Massage Department of the Royal Infirmary, Edinburgh, for treatment of a paralysis of his right serratus anterior muscle. The paralysis had come on suddenly during a game of rugby football, his attention having been first drawn to it by a weakness in his right shoulder when attempting to raise his arms above his head.

On being questioned he stated that he had no recollection of receiving a blow upon the right side of his neck or shoulder, but, as he played on the left side of the front row of the scrum, he constantly pushed with his shoulders, and especially the right against the shoulders of members of the opposing team. Examination revealed typical winging of the right scapula, when he raised his arms in front of him. There was no bruising, nor tenderness on pressure, to be found on the right side of the neck or shoulder. The serratus anterior responded well to Faradism ten days after the onset of the paralysis.

As it was concluded that the long thoracic nerve had been injured during the course of the game, he was advised to cease playing lest further trauma might occur.

Treatment consisted of intermittent Faradism given three times a week to the paralysed muscle, and the patient was instructed in light exercises calculated to maintain its tone. At the end of six weeks he

could raise the arm with greater ease, the scapula being drawn slightly forwards and upwards, and the "winging" of its vertebral border being less prominent.

Treatment was discontinued at the end of a further month, the disability having practically disappeared. When seen again two months later no weakness of the right serratus anterior was detectable.

CASE No. 25.J.G. Aged 32.

This patient was seen in the Massage Department of the Royal Infirmary, Edinburgh, where he was undergoing treatment for paralysis, of sudden onset, of the right serratus anterior.

On being interrogated as to his actions, prior to the onset of the paralysis, he gave the following history. Whilst on holiday ten days previously in Blackpool, he had spent an evening in a fair ground. He had enjoyed himself greatly, and had spent a considerable amount of time manipulating a "trial of strength" machine. In order to record his strength upon this machine the operator was obliged to grasp a handle, set at right-angles to the outer circumference of a large wheel, and to rotate it with both hands at high speed against resistance.

The patient stated that he was delighted with the success, which he achieved, and exerted himself again and again until quite exhausted. During the remainder of the evening he suffered no ill effects as the result of his labours, but, on waking the following morning, he found that his right shoulder was stiff and aching. On stretching his arms in various directions to loosen up, he felt the edge of his shoulder blade press against the inside of his clothing, feeling, in his own words, "as though it were trying to come out." As the day advanced the pain and stiffness in his shoulder diminished, but were again present, though to a lesser degree, when he awoke the following morning.

On his return home five days later the pain and stiffness in his shoulder no longer troubled him, but the shoulder blade continued to "come out" when ever he raised his arm in front of him. He had never previously experienced any pain in a joint, nor had he suffered from any rheumatic manifestation. He visited his doctor on the evening of his return, and was promptly sent for massage treatment.

Examination showed him to be a well set up and exceptionally muscular man. Whilst standing with his arms resting by his side, no asymetry of the back was apparent. As soon as he raised his arms in front of him, however, the vertebral border of the right scapula, especially at its lower end, was seen to stand out. This could be exaggerated by making him push against a wall at a point level with his elbows. The serratus anterior reacted to Faradism in all its digitations, and on palpation slight contractions of the upper digitations could be felt, as he thrust forwards from the shoulder.

Treatment consisted of the maintenance of tone in the paralysed muscle by bi-weekly courses of intermittent Faradism, the patient in the meantime continuing his employment as an upholsterer.

At the end of ten weeks' treatment some return of power to the middle digitations was evinced by less marked "winging" and by palpable contractions of the muscle. In a further six weeks all digitations could be felt to contract, and the scapula moved

forwards and upwards, when the arm was raised above the head, "winging" now being practically absent. When seen two months later no abnormality was detectable.

DISCUSSION ON MODES & MECHANISMS OF PRODUCTION OF
PRESSURE NEURITIS.

In order to appreciate the mechanism, by which Pressure Neuritis is produced, it is necessary to bear in mind the relations of the peripheral nerve involved, particularly at such points in its course as protection, afforded by its surrounding structures, may be deficient. For diagnostic purposes, knowledge of the roots from which the peripheral nerve originates and its distribution is essential.

I shall, therefore, when discussing the modes and mechanisms of production of nerve compression affecting the several groups of cases, I have observed and described, introduce by way of preface a short summary of the anatomy of the peripheral nerve concerned; I shall also refer to similar types of nerve injury described by various writers, supported by extracts from their case records.

THE COMMON PERONEAL NERVE.

The Common Peroneal Nerve is one of the two terminal branches of the sciatic nerve, through which it gains origin from L.4 & 5 and S.1 & 2. Inclining laterally from beneath the biceps femoris, the nerve enters the popliteal fossa, where it is easily palpable as it runs, first across the lateral head of gastrocnemius and more distally on the dorsum of the head of the fibula. Winding downwards and forwards round the neck of the fibula, covered only by skin and fascia it enters the substance of peroneus longus and ends by dividing into its two terminal branches, superficial and deep. The superficial branch, having given motor fibres to the peroneus longus and brevis, supplies the skin over the anterior surface of the distal third of the leg and dorsum of the foot and toes.

The deep branch after supplying extensor digitorum longus, peroneus tertius, extensor pollicis longus and tibialis anterior, ends by assisting the superficial branch in supplying the skin over the dorsum of the foot and toes.

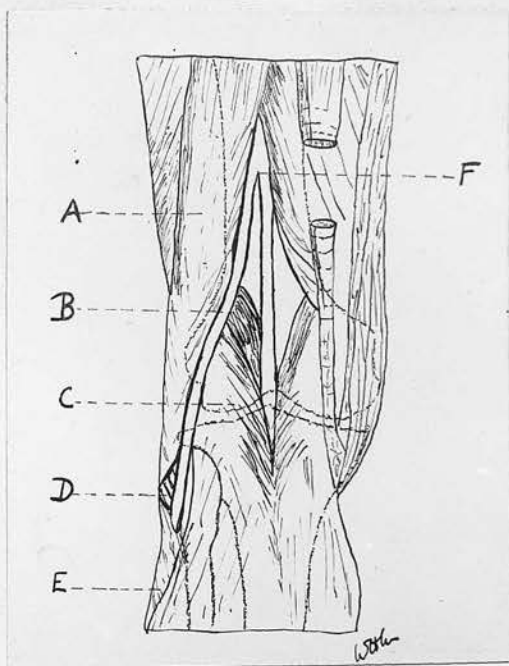


Diagram of dissection of back of knee.

A. Biceps femoris; B. Common peroneal nerve;
C. Lateral head of gastrocnemius; D. Head of fibula;
E. Peroneus longus; F. Bifurcation of Sciatic Nerve.

In the eight cases of injury to this nerve, which I have described, damage occurred in three different situations; (a) at the emergence of the nerve into the popliteal fossa (Nos. 1 - 4); (b) upon the dorsum of the head of the fibula (Nos. 5 - 7) and (c) on the outer aspect of the neck of the fibula (No. 8).

In cases Nos. 1, 2 & 3, the compressing agent was the hardened and contracted biceps femoris. Upward pressure upon this muscle from below by the calf muscles, whilst the leg was in a fully flexed position, must also be recognised as a contributory factor.

In case No. 4, a similar condition existed in both legs whenever the patient sat back on her heels to rest.

Three similar cases are recorded by Clayton (6). Two are of patients, in whom foot-drop had developed, following the assumption of a squatting posture. His third case was that of a young man, also suffering from foot-drop, who gave the history of having remained in a kneeling posture for over an hour. In commenting upon these cases he says, "Presumably the nerve must have been compressed between the biceps tendon and the fibula." Parker (16) mentions paralysis of the common peroneal nerve as occurring in beet planters, coal pickers and other workers, who are obliged to assume the squatting posture for long periods at a time. He states that the cause is compression of the nerve by the hardened and contracted hamstring muscles.

In case No. 5, in which the knee joints were allowed to remain hyper-extended, during the operation, the course, which the common peroneal nerves were obliged to take behind the knees became temporarily lengthened. Consequently not only was undue tension put upon the nerves, but they became tightly applied to the back of the knees and especially to the heads of the fibula, which, whilst the legs are in such a position, project into the popliteal fossae.

In cases Nos. 6 & 7, where hyper-extension as

well as general wasting was present, pressure of the nerve between the head of the fibula and the underlying mattress was probably the primary cause, although stretching of the nerve was no doubt a contributory factor.

Woltman (29) describes four similar cases of paralysis of the common peroneal nerve, occurring in patients, who had been confined to bed for considerable periods of time, and who had lost greatly in weight, so that the normal popliteal concavities had become obliterated, and the upper ends of the fibulae rested hard upon the bed causing compression of the common peroneal nerves. Paget (15) reports a case of isolated paralysis of the common peroneal nerve, occurring in an emaciated patient, who was convalescent from typhoid fever.

In case No.8, injury to the nerve occurred at its most vulnerable point. In this case vibration and cold also may to a certain extent, have aggravated the condition. The history given by this patient was far from spontaneous, and it was only after considerable questioning that the cause was elicited.

Russell (21) records a case of injury to the right common peroneal nerve which resulted in foot drop. His patient was a district nurse, who in lifting bed-ridden patients was in the habit of leaning with the outer side of her right leg against the edge of the bed.

He adds that "this evidently caused pressure neuritis of the peroneal nerve at the neck of the fibula."

Woltman (30) describes the following two cases, in which foot-drop was produced by crossing the legs in such a manner, that the common peroneal nerve became wedged between the head of the fibula of the same leg and the external femoral condyle and patella of the opposite leg.

- (1) A blacksmith, age 55, came to the Clinic because of paralysis of the right foot. One week before he had been at a wake, where he had sat through most of the night, and, as was his habit, frequently crossed the right leg over the left. Before he left the house of his departed friend, he was completely incapable of dorsiflexion of the right foot. The palsy was moderate in degree, and he was recovering from it rapidly. There was no impairment of sensation.
- (2) A jeweller, age 62, had lost weight with great rapidity. He had been so heavy that for years he had been unable to cross his knees, but he had discovered now he could do so and enjoyed it thoroughly. Examination disclosed an exophthalmic goitre of rather severe grade, and slight weakness of the dorsi-flexors of the right foot, with subjective numbness over the dorsum of this foot. Following thyroidectomy his condition improved rapidly. A letter received subsequently stated that he had recovered his health completely, and the power in the foot had returned to normal.

The same author also mentions having seen a man, who suffered paralysis in the distribution of both sciatic nerves, as a result of having fallen into a drunken sleep, while seated on the edge of an open barrel, into which he had gradually subsided.

Stopford (25) has observed clinical evidence in the distribution of the superficial branch of the

peroneal nerve, indicative of changes in the nerve, which were apparently due to compression by a tight sock suspender or other form of constriction below the knee. He states that in every case the manifestations were discovered more or less accidentally in the course of a general examination. A small proportion of the patients admitted that they had occasionally experienced tingling on the outer side of the leg or dorsum of the foot. The objective manifestations were both sensory and trophic. The former ranged from small patches of hyperaesthesia to anaesthesia and analgesia, corresponding almost to the full cutaneous distribution of the superficial branch of the peroneal nerve. Trophic changes consisted of loss of hair and cutaneous elasticity, and greater smoothness of the skin in the distribution of the nerve. In one case, the absence of hair accurately marked out the area of skin, which it supplied. No patient exhibited any demonstrable paresis of any of the anterior tibial group of muscles.

THE RADIAL NERVE.

The Radial Nerve is a continuation of the posterior cord of the brachial plexus, receiving fibres from C.5 6 7 & 8. In the upper two-thirds of the arm it lies closely applied to the shaft of the humerus, first upon its medial side and more distally winding round the dorsum in the radial groove, between the heads of the triceps. Whilst in the arm it supplies the three heads of triceps, anconeus and brachioradialis, before terminating opposite the capitulum of the humerus by dividing into its terminal branches, superficial and deep. The superficial branch supplies the skin on the dorsum of the wrist, lateral half of hand, thumb and proximal phalanges of first and second fingers. The deep branch winds distally

and backwards round the lateral aspect of the radius, supplying extensor carpi radialis brevis and piercing the substance of supinator, which it supplies, to reach the dorsum of the forearm. Emerging from the lower edge of supinator, it immediately gives branches to extensores carpi ulnaris, digitorum communis and digiti quinti proprius, and continues distally to terminate in motor branches to abductor pollicis longus, extensores pollicis longus and brevis and extensor indicis proprius.

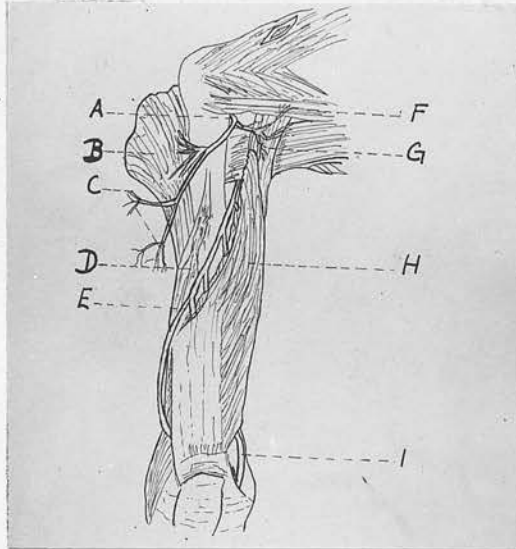


Diagram of dissection of back of shoulder and arm.

A. Axillary nerve; B. Deltoid; C. Cutaneous branches of axillary nerve; D. Lateral head of triceps; E. Radial nerve; F. Teres minor; G. Long head of triceps; H. Medial head of triceps; I. Ulnar nerve.

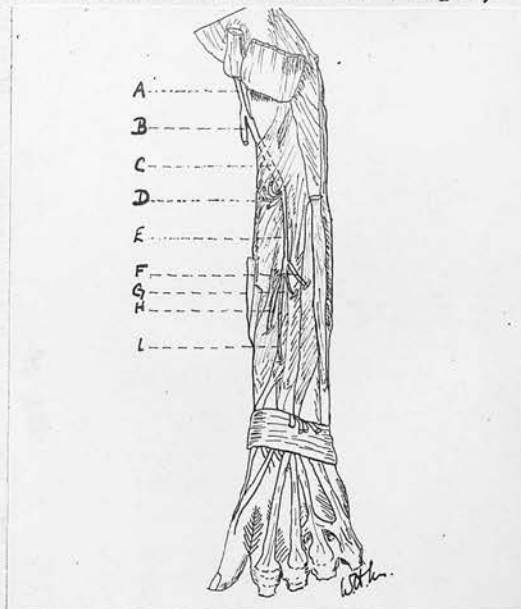


Diagram of dissection of back of forearm.

A. Radial nerve; B. Superficial branch of radial nerve; C. Supinator; D. Branches to superficial muscles; E. Dorsal Interosseous nerve; F. Extensor indices proprius; G. Extensor pollicis longus; H. Abductor pollicis longus; I. Extensor pollicis brevis.

In the first two cases of radial palsy (Nos. 9 & 10), which I describe, nerve injury had occurred in the axilla by compression, produced by the patient, in resting his weight on the upper end of a crutch. It is not a little surprising that in neither case, in which complete motor paralysis of the radial nerve was present, could sensory disturbance be found in the distribution of its external cutaneous branch, above the origin of which, injury had occurred.

Case No. 11 is of interest, as illustrating peripheral nerve compression between a firmly contracting muscle and a bone. The method of supporting a weight on the shoulder, which the patient employed necessitated extreme contraction of the triceps. When a similar movement is attempted, but gripping only with the thumb and first two fingers, the lateral head of triceps is felt to contract more firmly than the long and medial heads. No weakness of the triceps was present in this case, but the brachio-radialis was completely paralysed. Therefore the injury must have occurred near the point, at which the nerve pierces the lateral intermuscular septum. At this point, the nerve lies between the lateral head of triceps and the shaft of the humerus. It is therefore reasonable to conjecture that the patient's inability to grip with the ring and little fingers of his right hand was a contributory factor in occasioning the nerve lesion.

Cases Nos. 12 and 13 are illustrative of "Drunkard's Palsy," the best known example of pressure neuritis, and mention of which is seldom omitted from text books of Medicine and Surgery. Case No. 14 presents a similar condition, occurring in an unconscious patient. Similar types of radial palsy are seen when the arm of an anaesthetised patient has been allowed to hang over the edge of the operating table.

Case No. 15 is a further example of injury to the radial nerve by pressure. The mechanism of production here is obvious, and calls for no explanation. It resembles case No. 17, in which injury to the median nerve occurred.

In Case No. 16, the paralysis is limited to the distribution of the dorsal interosseous branch of the radial nerve. The mechanism whereby the nerve injury was produced was not at first apparent, but from the history, excessive muscular action appeared to have been the aggravating factor. This view was subsequently supported by the patient's recovery following the cessation of recreations, requiring repeated strong contractions of the forearm muscles.

Parker (16) describes a similar case, occurring in a hardworking farm girl, aged 23, who came for treatment on account of weakness in extending the fingers of her left hand. The condition had started in the little finger twelve months previously, and had spread gradually radialwards to involve all the

fingers and finally the thumb. Atrophy of the extensor muscles, supplied by the dorsal interosseous branch of the radial nerve, had produced a definite groove on the left forearm. Palpation over the neck of the radius revealed the nerve as a thickened nodular cord. In commenting on the cause of the condition, the author states that two possible factors present themselves for consideration. In the first place compression of the nerve by the contractions of the supinator might be responsible, and secondly, repeated knocking of the forearm against hard objects, in the course of her work, might have readily traumatised the nerve, owing to its superficial location.

Woltman and Learmonth (31) record five personally observed cases of isolated paralysis of the dorsal interosseous nerve. In one case, they performed an operation exposing the nerve, and found that it pursued an anomalous course. Instead of passing through the substance of the supinator it lay superficially between the muscle and the aponeurosis of the common extensor. With the object of reducing pressure on the nerve, a strip of the overlying aponeurosis was removed. Up to the time of their communication, eighteen months later, no return of power had occurred. They consider that this anatomical abnormality was probably responsible for the condition, and add, that the absence of improvement, following operation, was no doubt due to

irremedial interstitial changes having previously occurred in the nerve.

Guillain and Courtellement (8) record a case of paralysis of the dorsal interosseous nerve, occurring in the conductor of an orchestra. Over a period of six months, the patient had gradually lost the power of extending first the little, then the ring, and finally the middle finger of his right hand. At the time of examination, ten months after the onset of the condition, partial reaction of degeneration could be demonstrated. The writers attributed the paralysis to partial damage of the nerve by the supinator muscle as the patient made the repeated movements of pronation and supination, necessary in his professional work, which they advised him to give up. Unfortunately no information as to the progress of this case is available.

Winkworth (28) commenting upon "Tennis Elbow" considers one form of the condition to be due to the frequent traumatisation of the dorsal interosseous nerve as it passes through the substance of the supinator, due to sudden jerky contractions of that muscle. Similar views are expressed by O'Sullivan (14) and Marshall (11).

THE MEDIAN NERVE.

The Median Nerve arises by two heads, receiving fibres from C.5 6 & 7 through the lateral cord, and from C.8 and T.1 through the medial cord of the brachial plexus. The path of the nerve as far as the distal two thirds of the forearm is sheltered, but it is to be noted, that in leaving the cubital fossa it passes between the two heads of pronator teres.

The median nerve gives no branches in the axilla or upper arm. In the forearm it supplies pronator teres, flexor carpi radialis, palmaris longus and flexor digitorum sublimus, and through its volar interosseous branch flexor pollicis longus, the radial half of flexor digitorum profundus and pronator quadratus. The main trunk enters the hand by passing beneath the transverse carpal ligament, its cutaneous branch to the palm passing superficial. In the hand the nerve splits into motor fibres to the first and second lumbricals, abductor, flexor brevis and opponens pollicis, and supplies the skin of the volar surface of the digits, dorsum of second phalanx of thumb and second and third phalanges of the radial two and a half digits.

The course of the median nerve in the axilla and arm is well sheltered. Consequently pressure neuritis is unlikely to occur, except in such a case as No. 17 where gross pressure has damaged not only the nerve, but also the structures, which normally surround and protect it.

In leaving the forearm, the median nerve passes between the two heads of pronator teres, repeated violent contractions of which, according to Price (18) may damage the nerve and give rise to one form of "Tennis Elbow."

Case No. 18, presented an intriguing diagnostic problem because the history was not readily elicited, nor was the diagnosis certain until verified by recovery, which occurred after the patient had protected the terminal fibres of his right median nerve from both pressure and cold.

The terminal fibres in the hand are those most frequently involved in pressure neuritis. Dr. Marion Seyring (23) describes a case, which occurred in a foundry worker who used a pneumatic chisel. He

suffered from weakness and wasting of the small muscles composing the thenar eminence. To work this type of pneumatic tool, the operator is obliged to depress a valve in the handle with the ball of his right thumb. By this action, there is transmitted a series of rapid shocks to the right hand, whilst the machine is in motion. Dr. Seyring considers the condition to be due to degeneration, the result of compression, of the fibres of the median nerve, which supply the muscles of the thenar eminence.

Legge (9) states that similar instances of pressure neuritis of the median nerve have been encountered in locksmiths, carpenters, drillers, weavers and cigarette-makers due to excessive muscular action.

He specially instances file-cutters, who suffer from compression of the nerve because of their tight grip on the hammer and chisel.

Damage to the median nerve by prolonged occupational pressure is mentioned by Brain (3), who states that cases have been observed in gardeners from pressure caused by the handle of the trowel, in scrubbers from the pressure of the scrubbing brush, and in tailors from the use of heavy shears.

Case No. 19 illustrates pressure neuritis produced by an ill fitting splint. I have heard of similar cases, in which more permanent nerve injury has been similarly produced.

THE ULNAR NERVE.

The Ulnar Nerve gains origin from the anterior rami of C.8 & T.1 through the medial cord of the brachial plexus. Its course, as far as the elbow, is well sheltered. At the elbow the nerve occupies a superficial position, crossing the dorsal surface of the medial humeral epicondyle, covered only by skin and fascia. In the forearm it supplies flexor carpi ulnaris and the ulnar part of flexor digitorum profundus. In the lower third of the forearm it gives off its dorsal branch, which runs obliquely distally and backwards to become superficial immediately above the wrist, at which point the distal end of the ulna is its immediate internal relation. It supplies the skin upon the ulnar side of the wrist and hand, and ends by supplying the skin upon the dorsum of the little and ulnar side of ring fingers. The main trunk terminates in the hand opposite the hook of the hamate, by dividing into a superficial and a deep branch.

The superficial branch gives motor fibres to palmaris brevis and supplies the skin of the volar surface of the ulnar side of the hand and little finger and the ulnar side of the ring finger. The deep branch is purely motor, supplying abductor and flexor brevis and opponens digiti quinti, the interossei, the third and fourth lumbricals and adductor and flexor brevis pollicis.

The two cases (Nos. 20 and 21), which I have recorded, illustrate pressure neuritis of the ulnar nerve, and are similar in three respects; (a) the nerves were injured at the bend of the elbow; (b) Injury occurred as the result of the weight of the arms pressing the relatively exposed nerves upon the underlying mattress; and (c) Neither patient was aware of the cause of the condition.

The onset appears to have been gradual in both cases, but in No. 21 the condition had become more advanced because the nerve had been subjected to repeated compression for a longer period before the patient sought medical advice.

Woltman (29) records five cases, in which bilateral ulnar neuritis developed. In four of them, the patients had been confined to bed for periods ranging from two weeks to two months. He attributes the selective neuritis of the ulnar nerves to compression at the elbow between the bone and underlying surface. In one case, the patient, who was too weak to alter his position, and in agony from an operation on the bladder, dug his elbows into the bed. This produced more severe nerve compression, than that resulting from the weight of his arms alone, and he developed severe bilateral ulnar neuritis. The fifth case, which he describes, occurred in a man, who, following a stroke, lay for forty-eight hours unconscious on a hard cement floor, upon which his elbows rested. He developed in addition to a bedsore over his sacrum, a bilateral ulnar neuritis.

Lloyd (10) records three instances of a similar condition. The first occurred in an elderly woman, whose elbows had rested upon the table, during a long gynaecological operation, her hands meeting across her abdomen. The second case, a unilateral ulnar neuritis, producing muscle wasting and sensory loss, developed in a young man, who had been confined to bed for nine weeks suffering from typhoid fever. In commenting upon this case, he expressed the opinion that pressure was the cause of the palsy, but added that the possibility of injury by attendants could

not be excluded as a possible factor. His third case occurred in a man aged 20, who sought advice on account of weakness and wasting of his right hand, which had gradually developed over a period of eighteen months. On examination, the muscular atrophy was found to be limited to the muscles supplied by the ulnar nerve. No sensory disturbance was complained of, nor could any abnormality of sensation be found on physical examination. The patient was employed as a clerk, and spent many hours a day writing at a desk. Enquiry elicited the fact that he consistently wrote with the edge of the desk pressing on the inner side of his right forearm, approximately one and a half inches below the medial condyle of the humerus. This persistent posture was concluded to be the cause of the palsy. An interesting point was the complete absence of sensory disturbance in the presence of such gross motor impairment.

Parker (16) records two cases. The first occurred in a man suffering from a severely painful Iritis, who, for five or six days had spent his time sitting with his elbows on his knees, holding warm stupes to his face. Distracted by the pain in his eye he disregarded the warning tinglings along the inner sides of his hands. On recovery from the Iritis, the patient was found to be suffering from a severe bilateral ulnar neuritis. The second occurred in a man who, while suffering from right-

sided sciatica, was confined to bed for six weeks. During that time he carried on his work as a journalist, which involved a considerable amount of writing. While so occupied, he supported much of his weight on his left elbow, and later developed a severe left-sided ulnar neuritis with muscles wasting and sensory loss.

Russell (21) describes a case of bilateral ulnar neuritis, which occurred in an elderly woman, who was in the habit of sitting with her elbows resting upon a table. He adds that her ulnar nerves were found to be unusually exposed to damage, owing to the fact that she suffered from advanced pulmonary tuberculosis, and had lost considerable weight, which had deprived her ulnar nerves of some of their normal structural protection.

In Peel's (17) case, ulnar palsy appeared in a woman, who slept on her face with her arm placed under the abdomen to relieve pain.

Personal experience of pressure neuritis of the ulnar nerve is recorded by Buzzard (5). When being treated for fracture of the clavicle his arm was strapped in a position of extreme flexion at the elbow. Within a few hours he experienced painful paraesthesiae in the distribution of his ulnar nerve, which became so painful, that at the end of two days, he was obliged to have his arm released. In this case, although no pressure was applied from without, the nerve was maintained tightly pressed against the medial humeral epicondyle owing to the flexed position of the elbow.

In all the foregoing cases, pressure resulting in ulnar neuritis occurred in the neighbourhood of the elbow. Russell (21) records the case of a boy, who as a result of riding a distance of 20 miles on a bicycle with low handlebars, and on a bitterly cold day, developed bilateral ulnar neuritis. He considers the paralysis to be the result of compression of the ulnar nerves, as they enter the palms of the hands by the low handlebars, upon which the boy had lent heavily whilst riding.

A similar case is recorded by Clayton (6) of a patient, who developed a paralysis of the intrinsic muscles of the hand on two occasions, following long cycle rides. He states that the paralysis was apparently due to the pressure of the hand on the handlebar. In two cases observed by Stopford (25) subjective and objective sensory phenomena developed in the distribution of the dorsal cutaneous branch of the ulnar nerve, caused by compression of the nerve against the distal end of the ulnar by the tight band of a wristlet watch. In both cases the discomfort gradually disappeared on discarding the watch.

TOURNIQUET PARALYSIS.

Cases No.22 and 23, where both patients suffered from paralysis following the application of a tourniquet to the upper arm, serve to illustrate, that a nerve, which lies closely applied to bone no matter how deeply imbedded, is at times more prone to injury by compression than a nerve, occupying a more superficial position yet having a more yielding back-

ground.

While the tourniquet was in position, it may be assumed, that equal pressure was exerted at all points underlying it upon the circumference of the arm. Following the removal of the constricting band, both patients exhibited manifestations of pressure neuritis affecting the three main nerve trunks of the upper arm, namely, the median, the ulnar and the radial nerves, from which in time they recovered completely. It will be observed, however, that the last signs of pressure neuritis to disappear in both patients were those, occurring in the radial distribution.

At the points, at which the tourniquets were applied, the ulnar and median nerves lie nearer the surface than the radial, which lies deeply imbedded beneath the triceps, but resting directly upon the shaft of the humerus. This fact goes to prove that a nerve, no matter how deeply covered by overlying tissue, but having a hard bony background, will become more severely injured by compression than a nerve, whose path is bordered both superficially and deeply by resilient structures, and to which an equal amount of pressure has been applied.

THE LONG THORACIC NERVE.

The long thoracic nerve arises by three roots from C. 5, 6 & 7. The roots from C. 5 & 6 unite to form one trunk, which passes through the substance of scalenus medius. The root from C. 7 runs in front of the scalenus medius, uniting with the trunk formed

by C.5 & 6 at its posterior border. The nerve descends along the side of the neck, and entering the axilla continues its downward course over the axillary surface of serratus anterior supplying it. The fibres from C.5 supply the upper, those from C.6 the middle, whilst the fibres derived from C.7 supply the lower digitations of the muscle.

Case No. 24 illustrates a paralysis of the serratus anterior, resulting from injury to the long thoracic nerve, produced by pressure upon the shoulder and base of the neck by the patient pushing with the top of his shoulder. I have seen a similar paralysis in a railway porter caused by shouldering heavy weights.

Parker (16) mentions a case, in which the long thoracic nerve was injured by continuous pressure, occurring in a man, overcome by sea-sickness, who lay for several days on a hard bench.

The mechanism whereby the long thoracic nerve was injured in Case 25 is less clear. It is probably that the nerve was compressed and stretched during the unaccustomed strenuous muscular efforts, made by the patient with his raised arm. The neuralgic pains in the shoulder, of which he complained, suggest that some inflammatory exudate had occurred into the sheath of the nerve, which would further prolong the compression.

THE AXILLARY NERVE.

The Axillary Nerve arises from C.5 & 6 through the posterior cord of the brachial plexus. It leaves the axilla through the quadrilateral space, at which point the shaft of the humerus is its immediate lateral relation. Having given a motor branch to teres minor, the main trunk winds round the surgical neck of the humerus to supply the deltoid and overlying skin.

Although the axillary nerve runs for part of its course, closely applied to the neck of the humerus, its general location within the axilla is so well sheltered, that it is unlikely to be injured, save by direct pressure upwards and outwards into the armpit, such as might be caused by the head of a crutch. Examination of the records of the Massage Department of the Royal Infirmary, Edinburgh, show paralysis of the axillary nerve to be a comparatively rare occurrence.

It is stated (27) that paralysis of the deltoid muscle from direct pressure on the terminal filaments of the axillary nerve, may occur in miners, who work for long periods while lying on their side.

THE LATERAL CUTANEOUS NERVE OF THE THIGH.

The Lateral Cutaneous Nerve of the Thigh is purely a sensory nerve. It arises from L.2 & 3 and runs round the side wall of the pelvis to the anterior superior iliac spine, where it passes beneath the lateral end of the inguinal ligament, and enters the thigh. The next part of its course is variable for it may pass above, through, or beneath the origin of the sartorius. When the thigh is extended the angle subtended by the nerve at this point becomes acute. The nerve runs distally and laterally at first beneath the fascia lata, and more distally in a tubular investment of the fascia, before becoming subcutaneous and splitting into terminal anterior and

posterior branches. The former supplies the skin of the lateral aspect of the front of the thigh almost to the knee. The latter supplies the skin of the lateral side of the buttock and adjoining thigh.

The first description of a disorder of this nerve was given in 1895 by Bernhardt (2) and Roth (20) in independent communications. They described it as an area of pain, paraesthesia and sensory disturbance, occurring in the distribution of the external cutaneous nerve of the thigh.

Numerous theories have been advanced as to the cause of this condition, known as Meralgia Paraesthetica, which range from pressure by the foetal head to varicose veins, gout and lead poisoning. Rosenheck (19) considers the location of the pain to be merely of academic interest, and considers the condition to be "an expression of a radicular syndrome, secondary to osteoarthritis of the spine, and not a peripheral nerve affection." Admittedly, osteoarthritis of the spine may give rise to root symptoms, but in the original description Meralgia Paraesthetica was described as an affection of the external cutaneous nerve of the thigh, a peripheral nerve.

Irrespective of the aetiology, one fact appears in all histories, namely, that standing or walking increases the pain and that rest and flexion of the thigh relieves it. The following typical case is described by Parker:-

"A physician, aged 45, came for examination because of a numb, prickling sensation over the lateral aspect of his right thigh. Three years before he had noticed, that after standing for any length of time he had a disagreeable sensation in the area mentioned. This disappeared on resting, but re-appeared whenever he had to remain standing. The tingling feeling became a burning, painful experience and reached a certain degree of permanency during his waking life. Occasionally he had short attacks on the opposite side, but they were never prolonged and showed no tendency to be permanent. A year before, the burning and tingling had ceased, to be replaced gradually by a patch of anaesthesia over the outside of his right thigh. At great pains, he had mapped out this anaesthetic patch and was very apprehensive as to its significance, dreading disease of the spinal cord.

He showed no other defects of physical examination than an area of anaesthesia to touch, pain and thermal sensibility about the size of his hand, which could be plotted over the lateral aspect of his right thigh. The area involved was in the domain supplied by the lateral cutaneous nerve of the thigh. At the periphery of the anaesthetic area there was some hyperaesthesia to pin prick, but this was not marked. Palpation over an area just below the anterior, superior iliac spine showed a rounded nodular cord, that could be rolled under the finger tips, and forcible compression produced a tingling over the lateral surface of the thigh. This was assumed to be a thickened nodular and tender lateral cutaneous nerve of the thigh. A diagnosis of Meralgia paraesthetica was made, and on being assured as to the trivial character of his disease, the patient was willing to cease troubling about it, and to ignore the patch of anaesthesia that did not interfere with his daily functions."

Stookey (24) in operating on a patient, suffering severe pain in the distribution of the external cutaneous nerve of the thigh, found that at the point of emergence from the pelvis, the nerve was thickened, broadened and slightly injected, showing obvious signs of interstitial thickening. During the operation he was struck by the increase of tension on the nerve, and the narrowing of its angulation at that point, when the thigh was extended. He

made a number of dissections, and found that in every case, sharp angulation of the nerve occurred as it left the pelvis, especially in such cadavers as its course lay upon the bone immediately beneath the origin of sartorius. An examination made of a number of cadavers shows, that extension of the thigh visibly increases the angulation and tension of the nerve.

DIAGNOSIS.

When a peripheral nerve is damaged by pressure transmitted from without, or by some action or posture of the patient, to an extent sufficient to interfere with its normal function, the following clinical picture is presented.

In the distribution of the affected nerve, distal to the point at which injury has occurred, there will be muscular weakness or paralysis of a lower motor neurone type, accompanied by a variable degree of sensory disturbance. Pain, either at the site of injury or referred to the distribution of the nerve, is not a prominent feature of the condition. Paraesthesia of a mild type is sometimes complained of, but the type of sensory disturbance, which is most frequently encountered, is a diminution of cutaneous sensibility to pain, light touch and temperature. Occasionally the patient is unaware of the presence of numbness, until his attention is drawn to it in the course of a routine physical examination. In many cases the onset is sudden, no premonitory symptoms being complained of. In a few cases, in which the nerve injury has been caused by the frequent repetition of minor traumata, the onset is more gradual, and tinglings and stiffness of muscles may be experienced over a prolonged period. Trophic changes are not commonly seen, save for the disuse atrophy of muscles, encountered in cases, in which the condition has existed untreated for a considerable period of time.

Provided the patient can give a history, which reveals the cause whereby the nerve was injured, little difficulty will be experienced in recognising the condition. In a certain number of cases no helpful history is forthcoming, and careful investigation is called for in order to establish the diagnosis.

The first step is localisation of the lesion by analysing the muscles, which show loss of motor power, and by mapping out any area of sensory disturbance. When this has been done, it will be found, that all the manifestations of the nervous disorder occur in the distribution of one particular peripheral nerve. The lesion must therefore lie distal to the point, at which the various spinal nerves, which enter into the composition of this peripheral nerve, have united. Were the lesion proximal to this point its manifestations would have a "segmental" distribution. Again, the lesion must lie at or proximal to the point where the highest branch of the nerve is given off, in which conduction is impaired. Palpation along the course of the nerve, where possible, between these two points may lead to the discovery of a tender area, at which the nerve may be thickened and in long-standing cases indurated, and which will be the site of the lesion.

Once this accurate localisation has been carried out, careful interrogation of the patient with possibly leading questions relative to actions or postures, which could have produced nerve damage at that parti-

cular point, will bring to light the cause and establish the diagnosis.

The conditions, which must be distinguished from pressure neuritis, are other forms of peripheral neuritis, which fall into two main groups, Polyneuritis and Interstitial Neuritis, pressure on the lower cords of the brachial plexus by a cervical rib, and lesions of nerve roots especially those, in which the anterior root is injured. Other conditions, which must be eliminated, are lesions affecting the anterior horn cells primarily such as acute anterior poliomyelitis, progressive muscular atrophy and peroneal muscular atrophy and conditions, in which the anterior horn cells may be injured by compression, as in intramedullary tumour or syringo-myelia.

In Polyneuritis, which is a generic term covering degenerations of peripheral nerves from a variety of causes, it is the terminal fibres of the longer peripheral nerve, which are primarily affected. As the name implies several nerves are usually affected simultaneously. The characteristic premonitory symptom is paraesthesia in the fingers or toes, not restricted to the distribution of one peripheral nerve, but affecting all the fingers or toes. Later, if the condition advances, sensory loss is demonstrable, which spreads up the limb giving rise to the "glove" or "stocking" type of anaesthesia. Paralysis is often a relatively late manifestation of Polyneuritis. Its onset is gradual and it appears in the distal

muscles of the limbs, being accompanied by wasting.

In Interstitial Neuritis the prominent features are pain, referred to the distribution of the affected nerve, and local tenderness of the inflamed nerve trunk, which is increased by pressure or stretching. Interference with conductivity is seldom seen except in severe sciatica, where the ankle jerk may be lost, weakness may be apparent in the anterior tibial group of muscles, and numbness may be complained of on the outer side of the foot.

Cervical rib, by causing compression of the first thoracic and sometimes the eighth cervical nerves, gives rise to symptoms suggestive of pressure neuritis of the ulnar nerve, and is in fact virtually such a condition, since they are the spinal nerves from which the ulnar nerve is derived. The lateral head of the median nerve is however derived from the same spinal nerves, and the eighth cervical nerve gives fibres to the radial nerve. The manifestations will therefore not be restricted solely to the distribution of the ulnar nerve. Further points, which serve to distinguish the condition, are pains in the neck, vascular changes in the arm and demonstration of the rib radiologically.

Anterior nerve roots may be injured and give rise to lower motor types of paralysis in syphilitic amyotrophy. The diagnosis from pressure neuritis is made by observing that the paralysis is not restricted to the distribution of a single peripheral nerve. Severe pains, due to affection of the

posterior nerve roots, are a common accompaniment. The history, the Wassermann reaction and the characteristic changes in the cerebro-spinal fluid, all serve further to confirm the diagnosis.

In compression of the spinal cord sensory symptoms usually develop earlier than motor.

The sensory symptoms, associated with compression of the posterior nerve roots, are characteristic and not likely to be confused with pressure neuritis. Compression of the anterior roots primarily, due to collapse of the bodies of vertebrae, is a rare condition. It gives rise to weakness, wasting and fibrillation of the muscles, supplied by the affected segments. The condition is usually rapidly progressive, and manifestations of involvement of posterior roots and of tracts in the cord will not leave the diagnosis long in doubt. Similarly, compression of anterior horn cells by an intramedullary tumour will be accompanied by other manifestations of compression of the cord.

In acute anterior poliomyelitis, damage of the anterior horn cells will cause a lower motor neurone type of paralysis in the limbs, but the preceding constitutional upset and signs of meningeal irritation will point to the diagnosis.

In progressive muscular atrophy and in peroneal muscular atrophy, the onset is insidious and accompanied by muscle wasting and fibrillation. In the former, signs of interference with the pyramidal tract

and the medullary neuclei are usually present, and in the latter the age incidence is characteristic.

In syringo-myelia the dissociated sensory loss is typical and precedes motor manifestations. The "segmental" distribution of the sensory impairment will usually make the diagnosis clear.

PREDISPOSING FACTORS.

While the action or posture, responsible for pressure neuritis, is largely a matter of chance, there is no doubt that certain factors render a patient more susceptible to such a condition.

These factors may be divided into two main groups; lack of structural protection of the peripheral nerves, and devitalisation of the nerves themselves. Prominent in the former group is deficiency of the muscular and fatty coverings of the limbs, upon which the nerves rely for their protection. Confirmation of this is found in the preceding descriptions of twenty-five patients, eleven of whom were definitely emaciated, whilst of the remainder, only five were of stout bodily habit. That less than a third of the patients were females may be accounted for by the tendency, with which every one who has dissected the cadaver is familiar, of the female to possess greater adipose padding than the male.

Woltman (30), commenting upon a series of twenty-seven cases of peroneal palsy, produced by crossing the legs, states, that in the majority loss of weight was a prominent feature. In sixteen of his patients the average loss of weight was over two stones. There were in his group of cases only three women, a fact which surely cannot be accounted for by the assumption, that the habit of crossing the legs is less deeply ingrained in women than in men.

Although the elderly subject may be less liable to perform actions or assume postures, which will cause nerve compression, there is no doubt that with increasing years as the "give" and elasticity of the body decrease, so does its ability to withstand injury diminish.

The vitality of the peripheral nerves is lowered by the same factors which are responsible for the production of either the inflammatory or degenerative types of peripheral neuritis.

The fact that the majority of cases of pressure neuritis develop in the colder months of the year suggests that cold and damp may be a predisposing cause.

PROGNOSIS.

When pressure neuritis of a peripheral nerve is recognised as being due to an action or posture, for which the patient is responsible, and repetition of which can be avoided, a favourable prognosis may be given.

In this condition, by which any of the longer peripheral nerves may be affected in a diversity of ways, each individual case must be judged upon its own merits in estimating the length of time likely to elapse before return of function can be expected. Apart from such general conditions as senility, debility and infectious states, all of which tend to delay recovery, the most important factor to be taken

into account in forecasting the probable duration of the condition is the mode of onset. An insidious onset is evidence that the nerve has been injured by repeated minor traumata, which in all probability will have produced in it interstitial thickening. This overgrowth of the supporting stroma will obstruct regeneration of nerve fibres and will retard recovery. A sudden onset, on the other hand, usually indicates that, resulting from a single major trauma, an area of acute degeneration exists in the nerve. Recovery from this type of lesion is more rapid.

If the electrical reactions in a nerve, the site of pressure neuritis, remain unaltered at the end of 10 days, return of function may be looked for in a matter of weeks. Even if the reaction of degeneration is present, recovery may yet be anticipated although after a more prolonged period, always provided that the cause of the injury has been removed and its recurrence prevented. These conclusions are borne out by a consideration of the preceding twenty-five cases, in all of which, save one where death intervened, recovery occurred.

On referring to the case records, appearing in the literature, it is seen that mention of the ultimate result of the condition has not infrequently been omitted. But, in all cases where it has been mentioned, recovery, with two exceptions, has been the invariable result. The exceptions are the records of meralgia paraesthetica and the case of

paralysis of the dorsal interosseous nerve described by Woltman and Learmonth (31). Both these exceptions have, however, one factor in common, namely a congenitally abnormal course of the peripheral nerve involved, which has permitted it to be injured by normal movements of the body; a cause which is hard to eliminate.

The only account of an artificially produced pressure neuritis, which I can find, is made by Cunéo (7). He describes an experiment made by Waller, who compressed his own radial nerve for forty-five minutes, producing a paralysis in its distribution, which lasted for eleven days.

It may be added that the rapidity and completeness of recovery are expedited by treatment of the paralysed muscles, to maintain them in the optimum condition to resume their normal function, whenever their motor innervation is restored.

PATHOLOGY.

For obvious reasons it is not possible to obtain material for pathological examination from patients, suffering from the mild and acute forms of pressure neuritis. The condition is never fatal, and the removal by biopsy of a portion of the radial nerve from a patient suffering from a crutch palsy, for example, would change a transient disability into one, from which recovery would be prolonged and possibly incomplete.

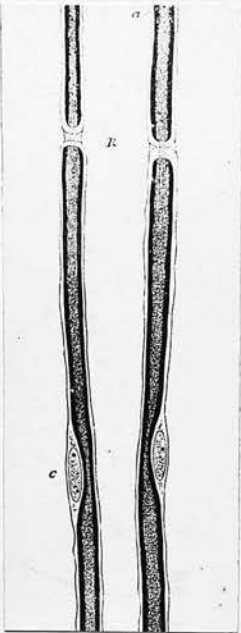
Observations, therefore, on the pathology of human nerves, damaged by pressure, have been restricted almost exclusively to nerves traumatised in a manner, which can only be relieved by surgical intervention. The clinical manifestations exhibited by such cases are similar to those of pressure neuritis due to action or posture, but conform more closely clinically to the cases, which have been produced by the frequent repetition of minor traumata. The condition is invariably of long standing, and one which has not yielded to the conservative measures of physiotherapy.

Examples of these so called "surgical" cases occur in meralgia paraesthetica, and in the late ulnar neuritis of cubitis valgus. The condition is also seen in a nerve involved in cicatrising fibrous tissue, formed during the healing of a wound in the vicinity of its path, and in a nerve pressed upon by overgrowth of bony callus, formed during the union of the fractured ends of an adjacent bone.

Before proceeding to a description of pathological changes a short summary of the histology of the peripheral nerve may be given.

The essential structure for the conduction of impulses in a peripheral nerve is the axis cylinder, which is an elongated expansion of a neurone or nerve cell. It is cylindrical in cross-section and has a uniform smooth surface. That its consistence is semi-soft is proved by the deformities produced upon it by the slightest pressure, as seen during the microscopical examination of a fresh unstained section. Surrounding the axon is the sheath of Mauthner, a transparent liquid, serving the purpose of a nutrient plasma for the axis cylinder. Immediately external to this fluid sheath lies a refringent oleaginous material, the myelin. Enclosing the myelin layer is a hyalin elastic cover, the sheath of Schwann. This sheath closely invests the peripheral nerve fibre, but is absent from fibres in the brain and spinal cord. It is the toughest part of the fibre and remains unbroken if the nerve is pinched, whereby both myelin sheath and axis cylinder may be ruptured. Septa, known as the segments of Schmitt-Lantermann, run from the sheath of Schwann or neurolemma through the substance of the myelin to the sheath of Mauthner. Thus communication is established between the exterior and the nutrient plasma surrounding the nerve fibre. At regular intervals along the course of a myelinate fibre are inserted circular discs at right angles to the course of the nerve. The axis cylinder passes uninterrupted

through these discs, which appear in some measure to maintain it in its central position within its coverings. The disc is smaller in diameter than the remainder of the nerve fibre, and at each one the sheath of Schwann is, as it were, caught-in, producing constrictions at regular intervals, known as the nodes of Ranvier. Upon each segment of the neurolemma formed by Ranvier's nodes occurs a Schwann cell, consisting of a nucleus surrounded by a condensed perinuclear area of cytoplasm. The Schwann membrane is a cytoplasmic process of the Schwann cell.

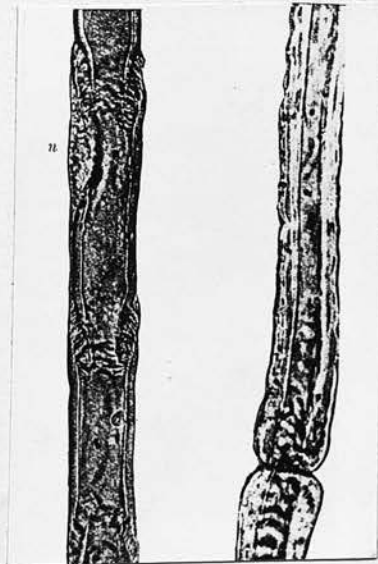


Portions of two nerve-fibres stained with osmic acid.
(E. Sharpey-Schafer.)

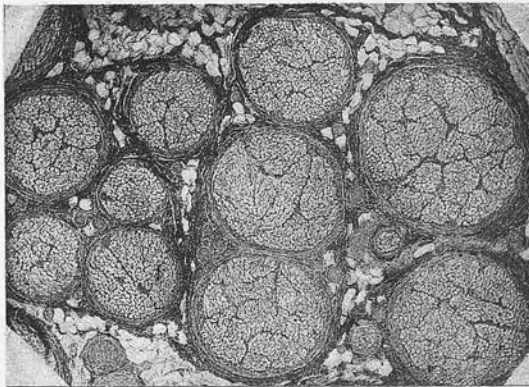
R.R. Nodes of Ranvier.
a. Neurolemma.
c. Schwann cell.

Two portions of myelinate nerve fibre. That on right shows node of Ranvier.
(E. Sharpey-Schafer.)

N. Schwann cell.



The neurolemma is bordered by a connective tissue membrane, the endoneurium, consisting of fibrils, each covered by an endoneurial cell. Thus each myelinated nerve fibre, apart from the Schwann membrane, possesses a mesodermal endoneurial sheath. Bundles of nerve fibres are loosely enveloped by a stronger mesodermal membrane, the perineurium. This consists of several lamellae of connective tissue, each covered by endothelial cells. Prolongations of the perineurium between the fibres of the bundles form the endoneurium. The bundles of nerves, held together by the perineurium, are themselves bound together by a connective tissue sheath, the epineurium, the fibres of which run longitudinally.



Section of Sciatic Nerve 60 (E. Sharpey-shafer.)
A dozen fasciculae, bound together by perineurium
are to be seen.

The pathological changes, produced by compression, vary with the amount of compression applied, with the duration of the compression, and with the frequency of its repetition.

No records of pathological examination of nerves, showing such temporary disfunction as the transient paraesthesia of "sleeping foot," are to be found, and indeed it is hard to see how portions of such nerves might be obtained. But, from the rapidity with which normal function is restored, it may be concluded that no structural alteration of the nerve can have occurred. Possibly the cause is a temporary ischaemia resulting from compression of the blood vessels running in the mesodermal sheath, and analagous to syncope, the result of temporary cerebral anaemia.

Stroebe (26) and B"unger (3) record early changes in peripheral nerves, which have been compressed experimentally although to an extent, which produced less transient symptoms than those mentioned above. They found that, following upon removal of the compressing agent, there was a general congestion of the blood vessels running in the mesodermal membranes. In the majority of cases small extravasations of blood were present, but no inflammatory reaction, as typified by perivascular leucocytic infiltration, was seen. A number of fibres, which varied with the amount of compression, to which the nerve had been subjected, showed no structural changes beyond in some instances a certain flattening. Others however, in more exposed positions, and which had therefore borne more of the brunt of the trauma, did not escape so easily. In them, within twenty-four hours, the myelin and axon appeared granular. At the end of five

or six days the myelin disappeared completely, the continuity of the axon remaining intact, although in some cases it appeared distorted. The Schwann cells showed early activity. Within twenty-four hours they became larger, the amount of chromatin in their nuclei increased, and they exhibited early mitotic changes. The cytoplasm became increasingly vacuolated and harboured droplets of myelin. Ultimately the cytoplasm of some was transformed into numerous minute vacuols, each of which contained a droplet of lipid material, whilst the nucleus, rich in chromatin, was displaced to the periphery. The Schwann membrane remained intact.

Whilst this increase of Schwann cells was going on, the mesodermal cells of the endo- and peri-neurium in the neighbourhood proliferated also, but did not show the same phagocytic activity as the Schwann cells.

From the above it will be realised that the condition is a partial degeneration of the nerve fibre, affecting the myelin sheath, and not interrupting the continuity of the axon. Degeneration of the distal portion of the nerve fibril does not occur, and return of function is consequently earlier than if the nerve had to regenerate its axon from the site of trauma down through the distal portion of its path.

In cases, in which the injury is more severe, the axis cylinder necroses, and is ultimately removed, along with the degenerated myelin, by the hyperplastic Schwann cells.

Here the condition is virtually one of nerve division, and Wallerian degeneration occurs, involving the entire divided sector down to the end plates. The pararenchyma (axon and myelin) is digested and removed, and an empty tube, consisting of proliferated Schwann and endoneurial cells, is left. Later the Schwann cells are removed by the mesodermal cells of the peri- and endoneurium. After such pathological changes have taken place, regeneration is accomplished by outgrowth of new fibrils from the central stump, which run to the periphery. These young fibrils often form themselves into spirals and branching networks. Such masses of atypically grown fibrils and newly-formed axons are known as the phenomenon of Perroncito. The majority of over-produced fibrils however, soon disappear and are replaced by parallel coursing fibres, enclosed within, and held together by accompanying cells of Schwann and those of the adjacent endoneurial membrane. Both groups of cells accompany the new axons to the periphery, and protect them. The Schwann cells regenerate along with the new axon from the central stump. The endoneurial cells are regenerated from the mesodermal elements of the neighbouring undegenerated nerve fibres.

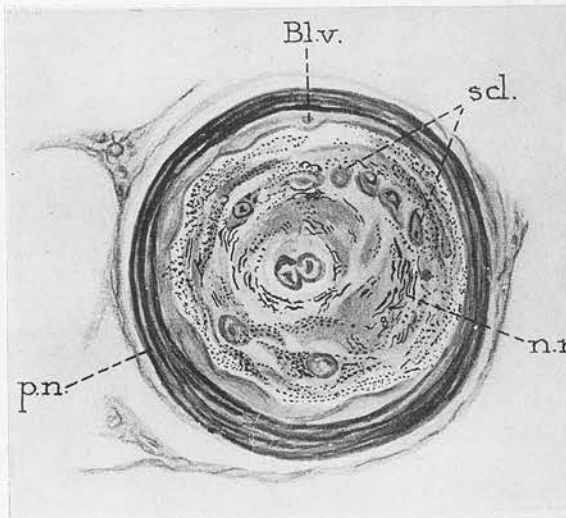
According to Schafer (22) if the severance of the axis cylinder had been produced by crushing so that the neurolemma remained intact, the proximal end of the axon might simply grow down into the distal part of the sheath as a single fibre. More-

over, when the nerve is only crushed and not cut right across, the neurolemma is not severed and complete restoration of function may take place within a few weeks, because each axon grows down the original sheath and is by it conducted directly to the original termination, whatever that may be.

The more chronic types of traumatic neuritis, which require surgical treatment as previously stated, have been studied pathologically in greater detail. Stookey (24) investigated a number of cases of meralgia paraesthetica. Having arrived at the conclusion, that the external cutaneous nerve of the thigh was subjected to repeated minor injury at the point where it was sharply angulated as it left the pelvis, he divided the nerve at this point. He thus relieved the patient of an area of painful paraesthesia, transforming it into one of anaesthesia. He found in every case upon which he operated, that the nerve was thickened and fibrous, and that its outer sheath was abnormally adherent to surrounding structures.

Nawratski (13) gives a good description of the microscopic changes found in a portion of nerve, removed during such an operation. He found marked loss of nerve fibres, proliferation of endoneurial connective tissue with formation of broad bands among the fibres and occasional thickening of the walls of the blood vessels and of the perineurium. An interesting feature was bud-like formations among

the nerve fibres. They appeared as lumps of a homogenous mass, enveloped by solid lamellar capsules, situated on the inner surface of the perineurium. These lumps or bulbs varied in size and consisted of sclerosed connective tissue of the endoneurial nerve membrane. They appeared secondary to the degeneration of the nerve fibres. The description of the bulbs, as given by Nawratzki, is similar to that of the onion-shaped formations as described in the hypertrophic type of chronic interstitial neuritis.



Transverse section of external cutaneous nerve of thigh from a case of Meralgia Paraesthetica (Nawratzki)

P.N.	Hyperplastic perineurium.
N.N.	Hyperplastic endoneurium.
BLV.	Blood vessels.
SCL.	Bulbs or buds of sclerosis.

Adson (1) describes the pathological process occurring in ulnar neuritis in cubitus valgus as follows:-

"With repeated slight trauma, such as that due to
 "bruising or to stretching of the nerve over the
 "bony prominence, small haemorrhages in the perineur-

"ium and endoneurium result, causing inflammatory
"reactions and the deposit of scar tissue. As the
"scar tissue tends to contract many of the fibres be-
"come strangulated and are eventually destroyed, re-
"sulting in a gradual and progressive atrophy of the
"nerves".

That this would appear to be so is proved by
the results of the treatment adopted in cases, in
which a peripheral nerve is involved in scar tissue,
e.g. the late sequel to a gun-shot wound. The
treatment is to free the nerve from the compressing
fibrous tissue, in which it is involved, and to in-
cise it in its long axis at any point where it shows
hypertrophic interstitial thickening. This freeing
of the nerve and breaking down of its intrafascicular
adhesions give, in many cases, good results, which
would appear to prove that strangulation of nerve
fibres, as described by Adson, was taking place and
had been arrested.

TREATMENT.

As pressure neuritis is specially liable to occur in emaciated or unconscious patients, care must be exercised in their nursing in order to avoid damage to the longer peripheral nerves.

In an unconscious patient, a limb must never be allowed to rest against a hard object, such as a splint, a hot water bottle, or the edge of the operating table, unless protected by adequate padding.

Emaciated patients, who are too weak to move, must have their position frequently altered, for in them, owing to the normal structural protection of the peripheral nerves being deficient, as a result of tissue wasting, pressure even upon a soft mattress may be sufficient to interfere with the normal conductive function of a nerve.

The first step in the treatment of a patient, suffering from pressure neuritis, is to explain to him the nature of his disability and the mechanism of its production, for thus, not only will his mind be set at rest and the fear of a grave and deep-seated nervous disorder banished, but his co-operation will be enlisted in avoiding further traumatisation of the affected nerve by repetition of the aggravating action or posture. The safety of the nerve may further be assured by placing a shield, composed of some firm material, or a soft woollen pad over the site of the lesion.

Trophic changes, such as alteration in temperature in the affected limb, loss of cutaneous

elasticity and retardation of desquamation, are not commonly seen in pressure neuritis. Areas of skin, however, in which temporary anaesthesia or analgesia are present, may be unconsciously injured by the patient and should be protected by suitable wrappings. Any small areas of sepsis, such as may occur around the nail folds, must be promptly dealt with.

Unless attention is paid to the paralysed muscles, not only will they waste and be unfit to resume their normal duties, when voluntary power is restored, but also they will tend to become stretched by their unopposed antagonists, which in their turn will become shortened and contracted.

The tendons and joints, normally activated by the paralysed muscles, will atrophy if allowed to remain immobile for any length of time, and adhesions will form between the tendons and their sheaths and within the joint spaces. These deformities, the result of disuse owing to the loss of voluntary motor power, will, when conductivity is restored, require prolonged treatment for their correction, and in elderly subjects, or if the condition has become at all advanced, restoration to the normal may never be achieved. To avoid such a contingency everything possible must be done to maintain the nutrition and tone of the affected muscles, and to avoid secondary deformities occurring in those structures, normally activated by them. This is done by passive movements, massage, and electrical stimulation of all paralysed muscles, to which also

adequate support must be given to avoid over-stretching. In every case, which I have seen so treated, no muscle wasting has occurred, and the length of time occupied by recovery, has depended only upon the restoration of the conductive function to the damaged nerve.

Treatment, to maintain muscle tonus and nutrition, should be conducted along the following lines. The patient is instructed in suitable passive movements, which he should carry out himself for periods of ten minutes three times a day, reapplying the splint, if one is necessary, at the end of each period. The paralysed muscles are massaged and stimulated to contract by electricity, three times a week. It is advantageous to induce a hyperaemia in the affected muscles, prior to treatment, by alternate application of hot and cold water, or better still by diathermy and radiant heat. Provided a brisk response to the alternating current is obtainable, electrical stimulation is best administered in the form of intermittent Faradism. Should the response be sluggish, a brisker muscle contraction may be obtained by Galvanism.

When voluntary power commences to return, the patient should be encouraged to perform frequently active movements, thus further maintaining muscle tonus.

In dropped wrist and foot the deformity is increased by gravity, and suitable splints must be applied to counteract this.

The distal portion of the "cock-up" splint, used in the correction of wrist drop, must extend far enough on to the hand to assure extension of the metacarpo-phalangeal joints, as well as dorsiflexion of the wrist. The distal portions of the fingers may be left free, allowing the patient a limited use of his hand, and in many cases enabling him to continue his occupation.

A less rigid extension of the metacarpo-phalangeal joints may be obtained by inserting the finger tips into the fingers, cut off a leather glove, from which elastic tapes are led over the dorsum of the hand to a cuff, attached to the splint at the wrist.

In foot drop the wearing of a boot, fitted with metal stiffeners, will maintain the ankle dorsiflexed to a right-angle. In practice a more satisfactory appliance is the spring toe-brace, consisting of a strap, in the centre of which a steel-spring is inserted, attached at one end to the toe of the boot and at the other to a well-padded leather gaiter, encircling the leg below the knee. A light right-angled posterior splint should be worn during sleep.

If there is any suspicion, that owing to an incipient state of degenerative neuritis, nerve injury has occurred with unusual ease, parenteral administration of Vit. B₁ may prove advantageous.

C O N C L U S I O N S .

- I. The normal function of peripheral nerves is frequently interrupted by pressure, produced by some action or posture of the patient.
- II. The condition usually passes off rapidly, and it is only the more persistent types, which come to the notice of the practitioner.
- III. The longer and more exposed peripheral nerves are those most commonly affected. It is also to be noted that the nerves, compression of which causes little pain, are those most frequently affected by pressure neuritis, due no doubt to pain reaching consciousness, as a warning signal, sooner than paraesthesia.
- IV. The patient seldom connects his action or posture and his neuritic manifestations as cause and effect.
- V. Establishment of the diagnosis as well as the treatment depends upon the discovery of the action or posture responsible. To achieve this, great care must be exercised in taking the history, with possibly occasional resort to leading questions.
- VI. The condition appears more frequently in thin than in fat subjects, and in men more often than in women. Cold and damp appear to play a part in increasing the incidence of the condition.

- VII. The pathological changes, occurring in the nerve, depend upon the severity and duration of the compression. They range from temporary ischaemia and a localized area of degeneration, with or without solution of continuity of the axon, to intraneural fibrosis and complete nerve destruction.
- VIII. A favourable diagnosis can usually be given, spontaneous recovery occurring in every case once the cause has been recognised and its recurrence obviated.
- IX. Treatment, apart from the safeguarding of the nerve against further injury, is directed chiefly towards maintaining the tone and nutrition of the structures, which have been temporarily deprived of their nerve supply.
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