THE JOURNAL OF NEUROLOGY AND PSYCHOPATHOLOGY.

VOL. III.

MAY, 1922.

No. 9.

Original Papers.

ON CAUSALGIA AND ALLIED PAINFUL CONDITIONS DUE TO LESIONS OF PERIPHERAL NERVES.*

By H. S. CARTER, LEEDS.

THIS paper is based upon the personal examination of over one thousand individual cases of injuries to peripheral nerves during the past three years. The author also had the opportunity of investigating many more cases at various stages of their treatment and recovery. Altogether, more than five thousand routine observations have been made. In addition to this amount of neurological work, facilities have been available for the study of pathological and bacteriological conditions, and access to the operations and operation notes of the surgeons under whose care the cases were placed has been possible.

The work has been carried out at the Second Northern General Hospital, now the Ministry of Pensions Hospital, Leeds.

DEFINITION.

Causalgia (thermalgia) is an intensely painful condition almost entirely limited to certain sensory areas of the median and sciatic nerve distributions, and caused by lesions of these nerves at points more or less distant from these areas, and characterized by local

* This paper forms part of a thesis accepted for the degree of M.D. in the University of Leeds.

vasomotor disturbances and general hypersensitiveness of the nervous system: a painful vasomotor neurosis due to irritation of a mixed nerve. Most of the cases seen and on record have been due to gunshot wounds, but very occasionally the condition is found in civil life.

The less painful conditions which are discussed along with true causalgia are distinguishable in degree, but arise from similar causes and under similar circumstances.

HISTORICAL.

The first case on record was described by Denmark in 1813. The patient was a soldier wounded at Badajoz by a bullet which entered the arm above the inner condyle of the humerus and came out on the outer side of the arm in front of the elbow-joint. Denmark described the state of affairs as follows:—

“I always found him with the forearm bent and in supine position, and supported by the firm grasp of the other hand. The pain was of a ‘burning’ nature, and so violent as to cause a continual perspiration from his face. He had an excoriation on the palm from which exuded an ichorous discharge.”

Hamilton, in 1838, stated in reference to a case, that:—

“The pain may be accompanied by redness and swelling resembling the appearance of the skin in inflammation of the fascia or a deep collection of matter.”

Paget, in 1864, drew attention to pain and vasomotor disturbances occurring as symptoms of incomplete physiological and anatomical division of nerves, and described the conditions as follows:—

“Glossy fingers appear to be a sign of peculiarly impaired nutrition and circulation due to the injury of nerves, and I cannot tell what are the peculiar conditions of the cases in which they are found; but they are a very notable sign, and are always associated, I think, with distressing pain and disability. In well-marked cases the fingers which are affected are usually tapering, smooth, hairless, almost devoid of wrinkles, glossy, pink or ruddy, or blotched, as if with permanent chilblains. They are commonly also painful, especially on motion, and the pain often extends from them up the arm.”

In the same year, 1864, Weir Mitchell, Moorhouse, and Keen (*Gunshot Wounds and Other Injuries of Nerves*) wrote of it as a rare disease, coined the name ‘causalgia’ from two Greek words meaning ‘pain’ and ‘heat’, on account of the burning pain which is the outstanding symptom, and made the first complete and classical description of the condition. Weir Mitchell states in reference to cases which occurred during the American Civil War:—

“The skin affected in these cases was deep red or mottled, and red and

pale in patches. The subcuticular tissues were nearly all shrunken, and, where the palm alone was attacked, the part so diseased seemed to be a little depressed, firmer, and less elastic than common. In the fingers there were often cracks in the altered skin, and the integuments presented the appearance of being tightly drawn over the subjacent tissues. The surface of all the affected parts was glossy and shiny as though it had been skilfully varnished. Nothing more curious than these red and shining tissues can be conceived of. In most of them the part was devoid of wrinkles and perfectly free from hair. Mr. Paget's comparison of chilblains is one we often use to describe these appearances, but in some instances we have been more strikingly reminded of the characters of certain large thin and polished scars."

Later, Weir Mitchell says :—

"Further study led us to suspect that the irritation of a nerve at the point of the wound might give rise to changes in the circulation and nutrition of the parts in its distribution, and that these alterations might be of themselves of a pain-producing nature."

It is probable that Weir Mitchell included in the term *causalgia* painful conditions arising from vascular injury associated with injuries of peripheral nerves, as well as those cases of *causalgia* due to lesions of the trunks of the median nerve and internal popliteal branch of the sciatic nerve.

In the forty years following Weir Mitchell's observations, years of comparative freedom from war on a large scale, little or nothing was added to our knowledge. In 1908, after the Boer War, Sherren, in his book on *Injuries of Nerves and their Treatment*, reviewed the earlier literature of the subject, and presented descriptions of cases occurring in civil life as a result of penetrating wounds and primary injuries associated with fractures or direct trauma, as well as those due to war injuries. He described also, what had not been emphasized before, the operative findings of incomplete division and scarring around the nerves. He writes :—

"It (*causalgia*) occurs most often in gunshot wounds with delayed union, but in several cases which have come under my care the wounds healed by first intention, and it may occur as a complication of subcutaneous injury."

The late war with Germany provided much more material for observation; and the more systematic examination and recording, particularly in relation to the French Armies, where *causalgia* as a result of nerve injuries seems to be much more frequent in its most pronounced form than among the British Armies, have resulted in the reappearance of descriptions in the literature and a revival of interest in the condition. Modern warfare, with the vast numbers of wounds caused by irregular projectiles, provides peculiarly appropriate opportunities for the frequent causation of incomplete division of nerves and large scar-formation in the neighbourhood of nerve-trunks.

METHODS OF EXAMINATION OF CASES.

As a rule, in cases of major causalgia, the patient's limb is much too painful, and the patient himself is much too apprehensive, for anything more than a perfunctory examination of sensory changes to be carried out. The patient is intolerant of the lightest touch or jarring of the injured member; the emotional condition is such as to make his answers to questions unreliable; and even where it is possible to conduct a superficial examination, perception fatigue occurs very rapidly and vitiates the results. This difficulty no doubt accounts for cases published in which it is stated there are no sensory changes. In all the cases observed and recorded in this paper there were definite sensory changes in the peripheral distribution of the nerves affected, although in all cases the observations were made with great difficulty, two or three interviews with the patient being needed before any sensory chart could be made.

In the less severe cases, examination is more possible, and the methods used were, in effect, those employed by Head and Sherren in their classical experiments (*see also* Burrow and Carter, *British Medical Journal*, Nov. 16, 1918). Space forbids their recapitulation.

CLINICAL CONSIDERATION OF CASES.

TYPES OF LESIONS.

There were five varieties of clinical types recognized during this investigation of painful conditions caused by lesions of peripheral nerves.

1. *The Syndrome of Slight Nerve Irritation*, in which there was little or no pain when the limb was at rest, but where the interference with the nerve was manifested by altered quality of sensation (paræsthesia) in the peripheral distribution of the nerves. The sensory changes were, however, usually very slight. In these cases there were no marked trophic changes and no contracture, and little loss of function. They recovered quickly with suitable physiotherapeutic treatment.

2. *The Syndrome of Irritation of Mild Neuralgic Nature*, in which the pain was of an aching character along the course of the nerve and round the site of injury. In these cases also there was often a little paræsthesia in the peripheral domain supplied by the nerve, but sensory changes were almost negligible. There was little loss of function and no contracture or trophic changes, and the cases recovered rapidly with suitable treatment.

These two types may be found in relation to injuries of any peripheral nerves, and not especially in injuries of those mixed nerves possessing a large number of vasomotor and trophic fibres, irritation of which produces the profound disturbances seen in causalgia.

3. *The Syndrome of Severe Nerve Irritation*, found only in lesions of mixed nerves which contain numerous vasomotor and trophic fibres. Pain was the chief characteristic of these cases—intensified by warmth and also by cold. There was also cutaneous hyperæsthesia in the peripheral distribution of the nerve, and also in some cases there was some cutaneous anæsthesia to light touch. Deep pressure-pain sense was preserved and exaggerated. These cases seemed to be intimately allied with true causalgia in presenting the whole gamut of trophic changes found in the latter. Considerable loss of function was present. They resisted treatment.

4. *The Syndrome of Irritation of Severe Neuralgic Nature*, characterized by intense aching pain along the nerve and in the muscles supplied by the nerve and in the peripheral distribution of the nerve. These cases were mostly limited to the median and sciatic nerves. Trophic changes were not so marked in these cases. The pain varied in intensity, but was always enough to cause reduction of the capacity for function of the limb. Sensory changes were always present.

Types 3 and 4 are classified as minor causalgia.

5. *The Syndrome of True Causalgia*.—This occurred always in injuries of the median or sciatic trunks, or of the inner cord of the brachial plexus which seems to carry those fibres, injury of which causes the typical 'burning' pain characteristic of this disorder. In these cases, whilst their outstanding feature was the intense pain, the whole of the symptom-complexes of the previous two varieties were associated. A good deal of sensory disturbance was always present. These cases were found to be resistant to most forms of treatment.

These last three types classified as minor and major causalgia are the types illustrated in this paper. Strictly speaking, the presence of an area of impaired sensation indicates a neuritis rather than a neuralgia. In all the cases considered there were sensory changes.

INCIDENCE OF CASES.

The 23 war cases described occurred among about 3000 cases of peripheral-nerve injury of more or less severity. The incidence therefore was 1 in 130. True major causalgia was much rarer—about 1 in 750 cases. From the literature, severe causalgia seems to have been more common in the French Armies. Possibly the more emotional character of the race has had something to do with the greater prominence of mental and emotional symptoms recorded by foreign contributors in their cases.

The incidence of causalgia in civil practice is small. One case only is described (*Case 3*).

CLINICAL DESCRIPTION OF CASES.

Case 2.—Ptc. D., age 26, A.S.C. Patient wounded in the region of the right knee and along the outer border of the popliteal space, Aug. 27, 1917. His foot dropped immediately he was hit, but he had no pain. Operated upon at Boulogne, and bullet removed from the left leg, where it had lodged after traversing the right. About a fortnight later he began to have pain in the foot, chiefly in the sole. Sent to England, where, he says, the external popliteal nerve was sutured October, 1917 (there is much doubt about this). The pain got worse, and he became unable to bear his foot on the ground. For the next year he wandered from hospital to hospital, but had very little treatment except massage and interrupted galvanism, which did not relieve him. The pain was continuous, and was intensified by efforts to walk or any sudden movement. He found the pain located itself chiefly on the inner side of the sole of the foot, although there was some aching of the dorsum. He found it much worse in warm dry weather and much relieved in rainy weather. He says, "I feel as though I could walk miles in a nice steady rain."

He described the pain as a continual intense aching pain—on the sole 'burning' in nature, and on the dorsum like toothache.

CONDITION DECEMBER, 1918.—The patient is a little thin, nervous-looking man, with a scared, apprehensive expression. He has come for examination in a chair, although he can walk with the aid of crutches, and does so with the knee flexed and the foot clear of the ground. The leg is wasted from the knee downwards. The knee-joint is movable, but painful when moved. There is drop-foot with some inversion. There is a large fibrous scar over the outer side of the knee and across the popliteal space. Palpation and percussion of this cause referred tingling in the foot and exacerbation of the continuous burning pain in the sole of which he complains. All the muscles supplied by both external and internal popliteal nerves have a degree of voluntary power. Pressure and percussion over the line of the internal popliteal and posterior tibial nerves cause spasms of pain in the sole.

He is extremely intolerant of the slightest movement of the limb, and is emotional to tears when examination is attempted.

The whole foot appears to have lost character. The skin is dead-looking, especially on the sole; the nails are dull and brittle; there is some branny desquamation over the dorsum of the great toe. On the sole of the affected foot there is less furrowing than on the normal sole, and the skin on the inner side looks particularly sodden and dead. There is a tendency to sweating. The toes are fallen together (weakness of interossei), so that the whole sole looks smaller and more compact than the normal left sole.

The sole is intensely painful to even the lightest touch. The slightest stimulation causes starting and drawing back of the foot. The most intensely painful area is in the internal plantar region. Sudden firm grip is tolerated well after the initial spasm due to superficial stimulation has passed off.

On the dorsum of the foot below the external malleolus there is a less painful area, and elsewhere there is paræsthesia to light touch.

SENSORY EXAMINATION.—Weber's compass-point test gives the following results:—

With the foot at room temperature, 54°, 4 cm. between compass

points: Normal left sole $\frac{1}{2} \left| \frac{11111}{222222} \right|$: all correctly identified. Causalgic right sole $\frac{1}{2} \left| \frac{1111 \times}{\times 22 \times 2 \times 2} \right|$: 33 per cent wrongly identified.

With the foot cooled and adapted to 45°, 4 cm. between compass points: Causalgic right sole, $\frac{1}{2} \left| \frac{1 \times 111}{22 \times 2 \times 2} \right|$: 27.3 per cent wrongly identified. Cooling diminishes the inaccuracy a little.

Sensation of roughness tested with Graham Brown æsthesiometer: Normal sole, $\frac{2}{2.5}$. The threshold is higher on the affected side.

ELECTRICAL EXAMINATION.—All the muscles supplied by both external and internal popliteal nerves responded to faradism, but they required more current than normally, and the contractions were sluggish. To galvanic stimulation the long muscles responded well, K.C.C. just = A.C.C., with fairly sharp contractions. The small muscles (flexor brevis digitorum and small muscles of the foot) react sluggishly, and there is polar reversal, i.e., A.C.C. > K.C.C. There is no reaction of degeneration, however.

SURGICAL PROCEDURE.—Operation was performed Dec. 12, 1918. An incision was made along the course of the internal popliteal nerve from the popliteal space to mid-calf. Both internal and external popliteal nerves were found to be involved in very extensive adherent scar tissue. The external popliteal nerve in one place was found adherent to the skin. Faradic current directly applied to the bared nerves showed by contraction of their supplied muscles that both conducted stimuli. Careful dissection of the nerves from scar tissue was attempted, but the scarring in and on the nerves was so extensive that it was impossible to free them entirely. The wound was closed.

This operation made not the slightest difference to the intense pain, and the wound suppurated in forty-eight hours.

A month later, the sepsis having settled down, but the pain being as bad as ever, the internal popliteal nerve was divided and left so, with a view to relieving the pain immediately. Resection and suture was to be done later, when patient's condition improved; but he was so much better and had so little pain after division of the nerve that he refused further interference.

POST-OPERATIVE EXAMINATION.—The pain did not disappear altogether, although it was very much diminished, but a few months later, in May, 1919, all the external popliteal muscles were acting and responding to faradism. All internal popliteal muscles were out of action. There was full sensory loss in the internal popliteal area, with loss of deep sensibility. The burning pain had vanished, although in the external popliteal area there was still some aching and paræsthesia.

He was given anodal galvanism for the external popliteal neuralgia, with good results.

EXAMINATION ONE YEAR LATER.—There is no burning pain now, although there is a certain amount of pins-and-needles tingling on jarring of the sole, and some paræsthesia also in the external popliteal area. The whole picture now is insignificant. All internal popliteal-supplied muscles are paralysed; all external popliteal-supplied muscles are working. There are some small dry pressure sores on the great toe.

Patient says he eats well and sleeps well, has gained weight, and is working as a tram conductor and gets along very well. His personal

appearance is vastly different from the nervous apprehensive state of the painful period.

Case 3.—This case of median causalgia in a woman, with pain of about seven years' duration, the writer was privileged to see by courtesy of Dr. J. le F. Burrow. As the patient was only seen once, an incomplete examination was made.

Mrs. W., age about 60, had suffered for seven years from a burning pain in the left palm and median-supplied fingers. The pain started after an attack of cholelithiasis, and was first felt as a burning pain along the radial margin of the thumb midway between the palmar and dorsal surfaces. It rapidly became worse, and was much increased by touching any object or using the hand in any way. The patient could not bear hot- or cold-water applications, and hot fomentations increased the pain. The skin of the affected hand became pink, and the fingers soon showed a tendency to become conical. Small scattered pin-head vesicles would arise under the skin, and larger blebs would arise suddenly in a few hours quite spontaneously, there being no question of trauma. The pain became so intense, and interfered so much with her daily work and her sleep, that she sought treatment.

For some time she was treated with local applications and with anodal galvanism, but without relief. There was pain on pressure and percussion over the line of the median nerve up to $1\frac{1}{2}$ in. above the elbow.

In November, 1917, alcohol was injected into the nerve in the upper arm. The relief was only temporary. The pain returned, and the skin over the bony prominences of the median-supplied fingers became thin and glossy.

In March, 1918, alcohol was again injected. Again the relief was only temporary.

In October, 1918, the nerve was divided and immediately re-sutured. In less than a month the pain made its appearance again.

In November, 1918, the nerve was again divided higher up in the arm and sutured. After this operation there was considerable diminution in the pain, and when the patient was seen early in 1919 it was limited to the index finger and the thumb. It is by no means as severe as it was, and is comforted by a simple woollen covering.

About this time the patient was put upon anodal galvanic baths, which certainly tended to relieve the condition.

This painful lesion of the median nerve is said to have been due to gouty deposits in the nerve. If this is so, then there must be irritation of the sympathetic fibres at a higher level than that at which the nerve was divided, to account for some persistence of symptoms. Following this argument it would seem that division higher up still might have abolished the pain altogether for a time, but in any case the pain would be very likely to recur with regeneration of the nerve, for ablation of a string of sclerosed patches due to localized deposits is impossible without rendering the nerve incapable of repair. Probably general treatment directed at the underlying condition, combined with local sedative treatment and lapse of time, promise better permanent results than surgical intervention, which, however,

is valuable because it promises at any rate temporary relief from pain. Higher division has not been carried out, and the patient remains with a certain amount of pain and a recovering median nerve, with paræsthesia in its peripheral distribution.

This is an example of a prolonged and intense causalgia apart from mechanical trauma of the nerve. The writer has also seen a case of persistent aching pain in the balls of both thumbs, with wasting of both sets of thenar muscles, in an old woman of 70, who sustained Colles's fractures of both wrists earlier in life. In this case there was not a great deal of disability, although the constant aching pain interfered with sleep, especially in warm weather. The pain did not arise for years after the fractures, and perhaps was attributable to the advance in fibrosis of old age, with nerve pressure in the regions of the ancient fractures.

Case 4.—Ptc. E. V. H., age 24, 1/20 London Unit. He received bullet wounds of the left forearm on Sept. 15, 1916. There was no fracture. Admitted to hospital on Sept. 22, 1916. On Sept. 28 he had a severe hæmorrhage from the arm wounds. On incision it was found that bleeding came from the bifurcation of the brachial artery. The brachial artery was ligated, as were also the radial and ulnar arteries below. Up to this time he had complained very little of pain in the hand, although he stated that pain was present and that it became much worse after the operation. On Sept. 30 the pain was getting unbearable; it was situated in the median area of the palm of the hand, and was of an intense burning nature. The patient's appearance also suggested pain. His expression of anxiety and apprehension, and the attitude of the injured limb, were typical of the gravity of the disorder. The pain continued, and increased in intensity. The hand began to assume the typical causalgic appearance, with trophic changes and contracture. The hand was too painful to examine properly. The patient's mental condition became perverted and his manner strange. He carried a wet rag about constantly in the left hand, because, he said, it relieved the burning pain. Heat, noises, or sudden jarring produced paroxysmal exacerbation.

Early in December, 1916, he was pronounced by a physician as being "mentally deficient (mild degree), which accounts for his rather peculiar manner". It was not thought that he had any nerve injury of importance. There was little or no wasting of muscles, and he had a degree of power in them all. The painful palm was painted with a mixture containing menthol, chloral, and camphor.

Dec. 12. Definite interference with the left median nerve was diagnosed, and it was found that the flexor longus pollicis and the small median-supplied muscles did not respond to faradic stimulation. There was no reaction of degeneration.

OPERATION.—Dec. 21. The median nerve was found to be apparently only slightly involved in scar tissue. It was freed and the wound closed. For a little while after this there was some amelioration of the pain, but only for a few days. The application of diathermy was now tried over the scar area, and the arm was put up on a splint with a moist pad in the palm. Contracture was commencing.

SENSORY EXAMINATION.—The sensory disturbances were those typically found in median causalgia. There was no total loss to light touch.

ELECTRICAL EXAMINATION.—There was faradic response in every median muscle a few months after the neurolysis.

In view of these findings further operation was postponed, but improvement under treatment by diathermy and paraffin baths was not maintained, and his mental and emotional state was as bad as ever. The pain was rapidly becoming intolerable again.

SECOND OPERATION.—March 4, 1918. The median nerve from a point 2 in. above the flexure of the elbow to $2\frac{1}{2}$ in. below was found involved in scar tissue, and the nerve itself was found to contain fibrous nodules over a length of $2\frac{1}{2}$ in. This portion was resected, and end-to-end suture was performed.

POST-OPERATIVE EXAMINATION.—A month later the median causalgia was cured. The pain had disappeared. The patient looked and felt a new man, and was not recognized by the physician who had formerly pronounced him mentally deficient. In June, 1918, he was invalided from the service. There was no return of pain, and the nerve was regenerating.

This was a severe case of median causalgia with associated mental change caused entirely by the intolerable pain in a suitable subject.

Details of the other cases of major causalgia are to be found in *Table I* at the end of this paper.

Case 6.—Pte. G. W., R.G.A. Shrapnel wound of the right upper arm, March 31, 1918. Patient stated that he felt the sudden pain of the wound at the time of the impact, but thereafter had no particular pain until that which developed in the hand ten days later. Since then it has been increasing in intensity, and is of a severe neuralgic type. When seen on June 19, 1918, the patient obviously had pain. He carried the right arm with the elbow flexed and the forearm pronated and held against the body. The hand was wrapped up in a wet cloth.

All the joints were found freely movable, and there was no paralysis of any muscle, although he was very chary of motion of any kind because of the pain. This, he said, varies in intensity, is always situated in the palm, and changes with the weather. It is much worse before rain and better after rain. Contrary to the state of affairs in major causalgia, it is very bad in cold and damp weather and much more comfortable on warm sunny days. Swinging the arm or allowing it to hang down whilst walking causes throbbing and bursting sensations in the hand, and sudden noises or bright lights cause exacerbation of the pain.

The skin of the palm was very pink in colour, and sodden with the continual contact with the wet rag. The fingers were conical and very pink; the nails were dull and arched. The whole median area and its overlap in the hand were painful to all forms of stimuli. There was commencing flexor contracture.

There was a good deal of interference with sensation, and much paræsthesia, but no total loss to light touch; considerable interference with discriminating sensibility. All the median muscles responded to faradic stimulation.

OPERATION.—On July 5, 1918, the ulnar and median nerves were exposed in the upper arm for 6 in. of their course. Both nerves were surrounded by scar tissue and were freed. No bulb or hardening of

the nerves was felt anywhere. Perfectly smooth and soft nerve-trunks. The scar tissue removed was not dense—just a soft matting together of structure. The vessels were freed for 3 in. Nerve section was not deemed advisable, and the wound was closed in the usual way.

POST-OPERATIVE EXAMINATION.—Sept. 4, 1918: The continuous pain had almost gone, but there was considerable hyperæsthesia to even light stimulation all over the median area. All the muscles were acting well, but there was a little contracture. The thenar muscles were painful to pinching. Deep pressure over the course of the nerve in the arm was painful, but caused no referred sensation.

Nov. 12, 1918: All muscles working well, but there was still some contracture. There was no pain, but a little 'douleur de pincement'.

This example of painful nerve lesion partakes more of the nature of *Types 3* and *4* than of true causalgia, although the pain was sufficiently severe to cause much disability and distress.

Details of the other cases of minor causalgia are to be found in *Table II*.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS.

The recognition of the clinical picture of true causalgia is not difficult. The outstanding feature, of which the patient invariably complains, is the intense, smarting, burning pain. It is almost always median or sciatic nerve lesions that give rise to this syndrome. The violent pain is localized, in the case of the median nerve, chiefly in an area bounded by the base of the index and second fingers, the root of the thumb, and the middle of the palm; in the case of the sciatic to the internal popliteal area of distribution, and of this to about the middle third of the inner half of the sole, i.e., the middle third of the internal plantar area. From these areas of maximum intensity the pain radiates to other parts in the sensory distribution of the nerve involved. History usually reveals that the pain arose some time after the injury—from one to four weeks as a rule.

The pain is always greatly intensified in a spasmodic way by movements, sudden jarring, sudden noises, bright lights, emotion, and keen sensations. The painful areas are excessively hypersensitive to the lightest touch, and yet firm grasp is tolerated well; that is, pressure-pain sensibility is hardly disturbed, and the intense burning pain is referred only to the superficial integument. Exacerbations of the pain are frequent, and take the form of throbbing, bursting, agonizing spasms of horrible intensity, excited by the slightest stimuli.

Patients affected soon show the signs of anxiety and apprehension, and come to develop as much immobility as they can. If they walk, they do so very carefully to avoid shock. Heat and dryness intensify the pain, as also does the dependent position of the limb, so that patients always keep the arm up, or, if the leg is affected, have the

greatest reluctance to assume the erect position. There is a tendency to hold the whole limb in flexion. If patients are bedridden, the whole body may be held in a curled-up posture indicative of defence. Cold and moisture relieve the pain to some extent, and frequently subjects of this disorder carry, in the affected palm, wet rags which they keep continually moist. Sometimes they carry the wet rag or handkerchief in the sound hand, which may, indeed, become the seat of a certain amount of pain—suggestive of disturbed reflexes. One patient sought relief by bathing his hand in methylated spirit. Slowly evaporating lotions give temporary relief. Insomnia is common in these cases, owing to the pain.

Along with the pain which is the central feature of causalgia there are other disturbances. Sensation was disturbed in the areas of supply of the nerves affected in all the cases recorded in this paper, although Stopford has published cases in which he says there were no sensory changes. They may be easily overlooked owing to the extreme sensitiveness of the limb and the patients' reluctance to submit to any detailed handling.

Almost invariably there is no paralysis of muscles, but a paresis only, with electrical reactions which are only diminished and not altered in quality. Idiomuscular contractions are usually brisk. There is never reaction of degeneration. There is usually slight contracture. Involuntary movements and tremor may be noticeable.

Vasomotor and trophic disturbances are usually well marked. Early in the evolution of the case, the skin of the affected area becomes delicate-looking and flushed pink, and later becomes smooth, thin, and warm and red-looking. Glossy atrophic skin over the more bony parts is observed. Sometimes the affected palm or sole is dry and hot, but more often there is pronounced sweating, made worse by attention. The constant application of moisture causes a sodden, ground-glass appearance of the skin, and there is often delayed desquamation of epithelium. The fingers become attenuated and tapering towards their distal ends. There may be subcutaneous vesicles or blisters, which arise spontaneously without trauma (*Case 3*). The nails grow rapidly, become curved, ridged, and brittle, lose their gloss, and become yellowish in colour. There is usually overgrowth of the subungual tissue, which may be painful.

The mental and emotional conditions of these patients depend a good deal on their normal temperaments. The normally highly-strung neurotic individual soon shows signs of instability (*Case 4*), but the normally phlegmatic type will suffer untold agonies without much outward sign of loss of balance (*Case 5*).

In cases of causalgia which are cured or are well on the way to recovery, there sometimes persists hysterical exaggeration of residual

symptoms, including simulated pain and spasm of the muscles of the limb affected. Persistent phobias of this and similar types may exist for some time after the cure is complete, possibly owing to persistence of abnormal impulses carried by sympathetic channels reacting upon a sensitized nervous system. Such extreme cases are, as a rule, only seen in persons of emotional individuality.

Considerations of space forbid a detailed discussion of differential diagnosis. The following conditions may, however, have to be distinguished from major causalgia : (1) minor causalgia, (2) recovering nerve lesions with altered sensation and paræsthesia, (3) ischæmic contracture (Volkmann), (4) physiopathic cases, (5) purely hysterical paralysis, (6) syringomyelia and Morvan's disease, (7) erythromelalgia, (8) polyneuritis, (9) tubercula dolorosa (multiple growths in the connective tissue of nerve-trunks), (10) focal lesions in the brain, with peripheral pain.

SUMMARY OF CONDITIONS OBSERVED IN CAUSALGIA AND ALLIED STATES.

1. Pain was present in all cases, varying in severity from the intense, intolerable burning pain of true major causalgia, to the dull, continuous, aching pain of the minor conditions.

2. Characteristic attitude, already described, of the affected limbs was observed in many cases, typically in the cases of major causalgia.

3. Motor disturbances were always noted, but there was never any absolute paralysis. There was always diminution of function.

4. Muscular atrophy was generally present, but was slight and much less marked than in total nerve lesions of similar duration.

5. Electrical reactions of muscles—these were always affected. Generally speaking, stronger currents were required to evoke contractions than normally.

6. Contracture of muscles was frequently observed, particularly in long-standing cases.

7. Vasomotor disturbances were present in all cases to a greater or less degree.

8. Sudomotor disturbances were frequently observed.

9. Trophic changes were always observed to be present to a greater or less degree, especially in regard to the nails, bones, and skin.

10. Sensory changes. Apart from the subjective disturbance manifest as pain, there were always objective sensory changes present, and limited to the more superficial varieties of sensibility.

11. Thermal symptoms. There was a certain amount of dissociation of thermal sensibility in most cases, although it was difficult to obtain information on this point. Patients, however, frequently

complained that the affected member felt hot and at times burning. Occasionally ordinary cool objects were felt as icy cold.

12. Involuntary movements were present in a few cases.

13. The tendon reflexes were usually a little brisker on the affected limb, but it was difficult to examine them in the severer cases. In no instance were they absent.

14. Mental and emotional instability was observed in the more severe cases, *Case 4* in particular.

PROGRESS OF UNRELIEVED CASES.

The intense pain, it was found, had a tendency to very gradual improvement, but in one instance it continued for years (*Case 3*). As a rule, in cases of twelve or eighteen months' duration, the pain had diminished considerably, but at considerable cost to the function of the limb. The power of voluntary movement improved very slowly with the pain, under suitable conditions; but frequently it became worse, and was complicated by a superimposed functional element. Atrophy of the hand or foot was progressive in untreated cases, until the member as a whole became smaller and more delicate-looking than the normal. Muscular contracture increased, arthritic changes occurred, and joints stiffened. The bones atrophied, the nails were coarsened and curled, the skin grew thin and glossy, the fingers spindle-like, and the whole hand or foot gradually became ischæmic, with fibrosis of muscles, assuming a claw-like character and ultimately developing into a painful, contracted, useless, deformed extremity (*Case 12*). Stopford has noted also that endarteritis of peripheral vessels occurs as an end-change in neglected cases. No opportunity for microscopic examination of arteries occurred in the cases studied. Trophic sores becoming gangrenous are prone to occur in a very few late cases. During the height of the pain the mental and emotional states of these patients become pitiful (*Case 4*). Thus the prognosis apart from suitable treatment is undoubtedly bad, and drastic measures are warranted without delay before the hand or foot becomes irrecoverably useless and before the intense pain has deranged the mentality of the individual.

PATHOGENESIS.

I.—ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS.

In discussing the pathogenesis of causalgia and allied painful conditions due to lesions of peripheral nerves, one factor immediately obtrudes itself for examination, namely, that these intensely painful conditions occur only in injuries to the median and sciatic nerve-trunks, or to the higher origins of the median nerve-fibres in the brachial plexus, or to the internal popliteal or posterior tibial branches

of the sciatic trunk. It is necessary, then, to consider in what way these nerves differ from other nerves of the limbs. These differences are as follows :—

1. Both sciatic and median nerve-trunks possess and supply specialized peripheral areas in the sole of the foot and in the greater part of the palm of the hand.

2. These areas of supply contain in their skin the majority of the varieties of sensory nerve-endings and end-organs—the corpuscles of Meissner, the Pacinian corpuscles, Ruffini's and Golgi's end-organs, etc.

3. Both nerves also possess a larger number of vasomotor fibres for the elaborate vascular supply of the hand and foot (Kramer and Todd, Potts). Section of the sciatic nerve causes active dilatation of the peripheral vessels of the foot (Claude Bernard).

4. Both nerves have themselves relatively large arteries of supply—the *comes nervi ischiadici* and the median branch of the anterior interosseous artery.

5. In the arm the median nerve is intimately associated in its upper reaches with the brachial artery, as are the sciatic and the internal popliteal nerves with the inferior gluteal, perforating, and posterior tibial arteries.

6. The arteries of the hand and foot—the palmar and plantar arches being placed, as they are, at the outposts of the vascular system and nourishing such important structures—receive a relatively very rich nerve-supply compared with the main trunks of the arterial tree.

In causalgia, the nature of the pain—burning (which is unlike that due to irritation of a purely sensory nerve) associated with throbbing, bursting sensations, and exacerbation in the hanging posture or on movement—and the vasodilatation of the skin, with local rise of temperature (Bénisty, Tinel, Stopford), are suggestive of derangement of vasomotor control. Obviously the vasodilator fibres are most affected, for there is no evidence of vasoconstriction, which would be shown by local anæmia and pallor of the skin surface. Moreover, the condition is relieved by assisting vasoconstriction by the application of cold and moisture. Stopford has suggested three possibilities to explain these phenomena of vasodilator instability :—

- a. *That the nerve injury in some way causes paralysis of vasoconstrictors.* There is no reason for this selective action, for the nerve injury in causalgia may be of the slightest, and there is rarely any degree of even partial division of the nerve (*see next section*). Moreover, although vasodilator fibres are more readily stimulated, they degenerate more slowly after section than vasoconstrictors; yet causalgia persists for many months, with no diminution in the vasodilator symptoms, even though there was considerable division of fibres.

b. Direct irritation of vasodilator fibres. There is no reason why there should be special selective action in this direction; moreover, uniform irritation of both vasoconstrictors or vasodilators would result in vasoconstriction, and therefore, as Stopford points out, it would be necessary to suppose either paralysis of the constrictors associated with irritation of the dilators, or specific action upon the dilators, for which conditions there is no supporting evidence. There is this point to consider, however. Bayliss has shown that stimulation which normally gives rise to vasoconstrictor effect, gives only vasodilatation after chloroform has been administered. Now the most important action of chloroform on a peripheral nerve is to produce a decrement in conduction, and therefore it is possible that the injury to the nerve has caused the interposition of a region of decrement which leads to the extinction of certain impulses and to modification of others.

c. Reflex phenomena. There is no doubt that the symptoms of causalgia can be explained along these lines, for if the various changes are considered as reflex phenomena, this state of affairs does not require that any selective action should be exercised; nor does it premise even the actual presence of vasodilator fibres in the nerves affected. The receptor channels for the vasomotor reflexes must be of two kinds:—

i. Those demanding an increase of blood-pressure, and known as pressor. The effect of calls sent along fibres acting as pressor channels would be vasoconstriction.

ii. Those calling for a fall in blood-pressure by relaxation of the normal vasomotor tone, and known as depressor. The effect of calls sent along fibres acting as depressor channels would be vasodilatation. It seems reasonable, argues Stopford, to assume that irritation will provoke a depressor effect as a protective and defensive mechanism. Moreover, the exacerbation of the pain in causalgia and allied states, by emotion, excitement, and attention can be explained by a psychical depressor effect.

Bayliss has shown that dilator fibres to the limbs have a good deal in common with sensory nerves, and are able to convey impulses in both directions, that is centrally, and back to the periphery. Or, in another way, they act both as receptor channels for vasomotor reflexes and effector routes for vasodilatation responses and depressor effects. By this means it may be supposed that irritation of these fibres at the site of injury of the nerve may result in sensory impressions being conveyed to the cord and brain, which project 'antidromic' dilator impulses via the posterior root ganglion to the peripheral area supplied by the nerve-trunk conveying the fibres. This explanation is analogous to Bruce's explanation of the vesicular eruption and pain in herpes zoster.

Again, the pain in causalgia is similar, though multiplied many times in intensity, to the pain caused by a superficial burn of the skin, which, also, is relieved immediately by cold and moisture. Also, on a normal palm or sole, in the areas which are the site of pain in causalgia, a pain-provoking degree of heat causes pain more readily than in other areas.

The pain of the small burn can only be caused by irritation of the nerve-endings in the damaged area, and the pain in causalgia may also be due to irritation of these by the 'antidromic' impulses projected from central origins. In support of this, the pain in causalgia is a superficial pain, as is shown by the fact that the lightest stimulation provokes intense exacerbation, but deeper pressure is well borne. Also the areas of intense pain are not the areas exclusively supplied with deep sensibility by the nerve affected, by which is meant that, if the median and sciatic nerves are completely divided, deep sensibility is usually not lost entirely over the areas which are commonly the site of the intense pain in causalgia, these being partly supplied by overlapping neighbouring nerves.

Harris has described a case where he had reason to believe that in the median nerve there is separation of fibres destined for the dorsum of the fingers and those for the palm, and that this separation takes place early in the nerve-trunk. If this is so, it may be that the fibres which, when irritated, give rise to causalgic conditions, travel in this palmar bundle only. This would explain the occurrence of the pain in the palm in causalgia, for it never occurs on the dorsum of the fingers.

It seems probable, also, that the secretory and trophic disturbances in causalgia can be explained on the same lines as reflex disturbances; especially as vesicular eruptions and blisters, analogous to the eruption in herpes zoster, do occur in causalgia. In further support of the reflex theory is the fact that pain may be also felt in the hand or foot belonging to the uninjured limb. Also, even after amputation of the limb in these cases, pain referred to the peripheral area in the hand or foot may still persist. Simple division of the nerve without resection in causalgia may not cure the pain, which result again can be explained by the persistence of stimuli arising from the damaged central end or from some sclerosed area above the division, or even from below along anastomosing sympathetic fibres to central origins, with reflection of impulses along centrifugal fibres, which may not travel with the trunk of the affected nerve, to the sensory nerve endings of the skin (*Cases 2 and 3*).

Finally, normality of the economy of any living organism depends upon the integrity of its reflexes, and the vast majority of the symptoms of disease are due to disturbances of reflexes. In causalgia

reflexes are disturbed, with as one result that hyperalgesic zones occur in certain peripheral areas. The normal reflex arcs being so disturbed, any added stimulus, mechanical, emotional, or psychical, acts in a reinforcing manner on the stimulus already affecting the arcs from the damaged parts, and this superimposed stimulus is sufficient to produce exacerbation of pain, whereas if applied to a normal undisturbed arc it would produce only the sensation of touch or pressure or some other not unpleasant reaction.

Two other theories have been put forward to explain causalgia :

1. Leriche's theory that the condition is due to a neuritis of the peri-arterial sympathetic system, and is not due to the direct injury to the nerve-trunk. In injuries to the upper arm it is not uncommon for damage to the median nerve to be accompanied by damage to the brachial artery ; but causalgia can arise from a wound of the nerve in the forearm or wrist where there is no injury to any large artery, and in causalgia from injuries to the sciatic nerve there is rarely any serious arterial complication. Moreover, Kramer, Todd, and Potts have demonstrated that the large arteries are supplied at irregular intervals along their course by sympathetic fibres from nerve-trunks, and not by a sympathetic plexus formed proximally, and sheathing the vessels. Again, stripping the artery in the region of injury of all sympathetic fibres (Leriche's operation), or stripping the accompanying vein as well (Tenani's operation), on the ground that ablation of the perivenous sheath will produce dilatation of the vein and diminution of intravenous pressure, which will further the effect of the changes in circulation brought about by the resection of the peri-arterial sympathetic, has no effect in cases of true causalgia. Possibly there are instances of sympathetic peri-arterial neuritis which are benefited by this treatment, but they are not cases of causalgia.

2. It is claimed by some writers (Souttar and Twining, and others) that the fact of both median and sciatic nerves possessing a well-developed vascular supply of their own has something to do with the genesis of causalgia. They believe that the pathological basis of the pain, and the whole gamut of trophic and sensory changes, are due to some alteration in the vascular supply of the nerves, affecting the fibres which supply two of the most sensitive regions of the body. This view is quite untenable, because median causalgia often occurs as a result of injuries to the nerve above the point of entrance of its nourishing artery, even as high as the inner cord of the brachial plexus. Also, causalgia as a result of injury to the posterior tibial nerve is well known, and this can hardly be due to involvement of the vascular supply of the sciatic trunk. Moreover, in more extensive injuries to the median and sciatic nerves, where much interference with the vascular supply to the nerves in the region of the lesions occurs,

there is no development of the causalgia syndrome. Indeed, the actual discoverable injury to the nerves in painful lesions may be very small indeed.

II.—PATHOLOGY AND BACTERIOLOGY.

1. Marinesco and Corner have sought to explain the pain in amputation neuromata by the irritation of sepsis—of an infective inflammation—and Joyce by analogy suggests that inflammatory reaction to certain micro-organisms may account for the development of painful conditions in certain nerve injuries which apparently differ in no way from the many which are not associated with pain. Against this suggestion are the facts that causalgia may occur after clean puncture wounds which heal by first intention without a suspicion of sepsis, and in cases without even a breach in the continuity of the skin surface (*Case 3*). And also, what is perhaps more striking—for the above conditions do not absolutely exclude microscopic bacterial infection of a subacute nature—causalgia and associated painful conditions show no predilection to occur in grossly septic wounds involving nerves.

In *Cases 1, 2, 6, 8, and 9*, cultures were made upon agar slopes and bouillon from the resected portion of nerve, or from the excised fibrous tissue where neurolysis was done. In four of these experiments no growth whatever was obtained after forty-eight hours' incubation at blood heat. In *Case 2* a scanty growth of staphylococci was obtained, and forty-eight hours after the first exploration in this case the wound flared up and a popliteal abscess resulted. Thus in four of the cases at any rate there was no active bacterial infection at the time of operation. In *Case 2* the attempt at relief of the condition by neurolysis was a long and difficult operation, and so the result is vitiated by the possibility of infection occurring during manipulation, for the initial wound was soundly healed long before exploration was attempted, although of course micro-organisms may lie latent in the tissues for a long time. If they have any value, these experiments would seem to show that even the maximum intensity of pain was not associated with active sepsis.

2. From the findings at operations on cases of causalgia and allied conditions, and from the sensory changes noted in these before operation, the constant condition present is one of incomplete anatomical and physiological division of the nerve. From an anatomical standpoint, division is too definite a description, for there is rather an interference only with the anatomical continuity of the nerve, and as a rule there is but little microscopical evidence of actual division of fibres, and the bare nerve conducts electrical stimuli in a way scarcely to be distinguished from the conductivity of a normal

nerve. It is common to find a variable section of the nerve embedded in more or less adherent scar tissue at the site of injury, but often the only finding is a small lateral swelling on the exterior of the nerve, due to fibrosis and repair after a lesion of the sheath. When the extraneural fibrous tissue is dissected away, the nerve may be found swollen and pink, and of a rather denser consistency than normal. There is usually increased vascularity, as evidenced by the considerable oozing of blood during the neurolysis. In some cases the amount of fibrosis is very small, and may have led merely to a diffuse local thickening of the nerve, or the nerve for a short distance may be slightly swollen and indurated or constricted by a fibrous band. In other cases small fibrous nodules can be felt in the course of the nerve at the site of injury. On opening the sheath of the nerve it is possible sometimes to see a few strands of fibrous tissue penetrating between the fasciculi, and also during the stripping of scar tissue small capillaries are cut which are seen to be entering the nerve from the perineural scar area. In wounds involving the sciatic nerve resulting in causalgia, the internal popliteal fibres are always affected. The external popliteal fibres may or may not have received damage. Injury to the external fibres alone probably never results in causalgia.

The conditions found at operation suggest that two types of injury are favourable to the after-development of causalgia.

a. Damage to the sheath of the nerve, more or less extensive, with vascular extravasation into the tissue of the nerve and around it, and subsequent fibrosis during the effort at repair.

b. Bruising of a nerve by the passage of a projectile through adjacent tissues, or by a blow in the region of a nerve, or by any other form of trauma in the neighbourhood of a nerve which will cause rupture of small vessels and outpouring of blood into and about the nerve, with subsequent formation of fibrous tissue.

In the very rare cases in civilian practice not associated with wound trauma, a localized interstitial neuritis, either by reason of the aberrant and unusual selective action of some infection, or gouty deposits in the nerve, may develop into a true causalgia (*Case 3*).

The microscopical lesion in these cases of causalgia and similar painful conditions is one of intraneural fibrosis. In several cases it was possible to obtain the portion of nerve excised at the operation. These were immediately transferred to a 10 per cent solution of formalin in isotonic salt solution, and after fixing were treated in the usual way and embedded in paraffin. Later, thin sections of these tissues were cut, mounted, and afterwards stained by a modification of Cone's stain. From sections stained in this way the microphotographs accompanying this paper were prepared.

On microscopical examination of these sections of excised portions

of nerves, it was found that intraneural sclerosis is the outstanding feature. The fibrous tissue is perifascicular, compressing and distorting the nerve-bundles, and is also found penetrating the ruptured fascicular sheaths and invading the nerve-bundles. Scar tissue can be seen between individual nerve-fibres, distorting and compressing them and causing torsion. In some parts the appearance is of nerve-fibres struggling through masses of fibrous tissue, and in others the young varicose fibrils associated with an attempt at nerve regeneration are seen. Some nerve-fibres appear strangled and degenerate. In some cases many nerve-fibres are affected, in others few, depending partly upon the duration of the case and partly upon the extent of the original injury. Probably very few suffer absolute solution of continuity. The vascularity of the fibrous tissue depends upon its age, and there are no features to distinguish it from ordinary scar tissue found in many cases of damaged nerves which were not associated with the syndrome of causalgia.

The picture is consistent with an early extravasation of blood into the nerve from external capillaries, and also from the intrinsic blood-vessels of the nerve, with subsequent organization and repair processes, formation of granulation tissue in and about the nerve, and the ultimate contraction of this into more or less dense fibrous tissue, which by its irritation and pressure maintains a condition favourable to the production of pain. The same processes in injuries of nerves other than the median and internal popliteal nerves produce irritative symptoms without pain, but associated with paræsthesia and altered quality of sensation in the peripheral areas of supply of these nerves.

TREATMENT.

I.—NON-SURGICAL TREATMENT.

The fact that the majority of cases of causalgia and similar conditions arise as a result of war wounds or similar traumatisms, makes early surgical interference a matter for hesitation on account of the risk of sepsis. Consequently, despite the urgency of the symptom of intense pain, arising as it does soon after injury, surgical treatment must be deferred, at any rate until the risk of complicating sepsis can be eliminated as far as possible.

It is in these earlier stages of the severer cases, and in minor cases, that non-surgical procedures are adopted, for, in a condition which, unrelieved, is so devastating in effect, something must necessarily be done to protect the patient either until, in minor cases, the severer symptoms subside, or, in major cases, surgical interference can safely be undertaken.

Non-surgical treatment is of little use in true major causalgia

except as a palliative. In general, dry and wet heat, massage, rhythmic faradic baths, galvanic or faradic stimulation, radiant heat, diathermy, or the paraffin bath, aggravate the condition and increase the pain. Morphia will, of course, ease the pain. Local applications of slowly volatilizing substances, cold wet compresses, the anodal galvanic baths, and ionization are of use in minor conditions, but give very temporary relief in major causalgia. X-ray treatment, radium therapy, and fibrolysin have all been tried many times, without much success.

II.—SURGICAL TREATMENT.

Whilst a case is waiting for operation, a certain amount of relief can be obtained for the patient by wrapping the painful member in lint, which is kept continually moist with some evaporating solution or with water, and supporting the limb firmly upon pillows, in a position which is comfortable relative to the rest of the body. When pain and other symptoms show no sign of diminution in about two months, surgical interference is warranted, or earlier if the pain is so severe that it is reacting unfavourably on the patient. In minor cases which show signs of increasing compression and irritation of the nerve, evidenced by increasing loss of function and deeper sensory loss, and perhaps more severe pain, operation is at once to be undertaken.

Excision and Suture.—The surest and safest and only treatment for the relief of major causalgia is excision of the affected portion of the nerve, followed by end-to-end suture where possible, as it nearly always is, or by nerve-graft where a large section has to be removed and apposition of the divided ends is impossible. In view of the local naked-eye appearance of the nerve, which is often very good, this treatment may seem drastic, for the anatomical continuity of a nerve is deserving of great respect; but because of the slight tendency to spontaneous recovery and the negligible effect of all other treatment, and the fate of untreated cases, it is justifiable. Immediate relief from pain results, and temporary paralysis, with, in most cases, the practical certainty of progressive painless return of function, is much to be preferred to long months of intense pain and the gradual evolution of a useless contracted hand or foot.

In true major causalgia the precision of the knife is a welcome and desirable attribute, although its employment may appear harsh; but—

“Diseases, desperate grown,
By desperate appliance are relieved,
Or not at all.”

Sicard and Imbert have tried to cure causalgia by dividing the nerve well above the lesion without interfering with the site of injury, .

but without success. Souttar and Twining claim to have cured an obstinate case, which did not yield to high division of the sciatic nerve, by section of the anterolateral tract of the spinal cord in the dorsal region.

Neurolysis.—In minor and less painful cases, where probably the intraneural fibrosis is very small and the condition due to the effect of perineural scar, a partial operation may sometimes be undertaken. Careful separation of all adhesions is necessary, and the careful dissection and removal of all the perineural fibrosis. Control of hæmorrhage is very important to prevent recurrent adhesions and the re-formation of fibrous tissue. When a nerve is stripped bare and freed from all scar tissue, it should be placed in a protective bed of fat or muscle. Extraneous materials, such as Cargile's membrane, or a sheath of vein, or rubber, are liable to cause the formation of a fibrous sheath around the nerve—a result not to be desired. The muscle bed in contact with the stripped nerve should be of uninjured muscle, and should be sutured round the affected portion of nerve.

Careful neurolysis on these lines, followed by suitable physiotherapeutic treatment, such as massage, electrical stimulation of muscles, exercises, etc., will sometimes suffice for recovery in less severe cases; but it is necessary to remember that a good end-to-end suture is better than a bad neurolysis. Neurolysis in true major causalgia is useless, and it is waste of time to attempt it.

It is quite impracticable to dissect away the intraneural scar tissue as Stoeffel advises and claims to do. The microscopical appearance of the nerve demonstrates that it is impossible under any ordinary operative conditions to dissect even the perifascicular fibrous tissue, much less the intrafascicular scar tissue, without doing more harm than good.

Alcoholization of Nerve-trunks.—Various surgeons have injected alcohol or cocaine into the affected nerve as a form of treatment, and French writers claim a great deal for Sicard's method of injecting a few cubic centimetres of 60 per cent absolute alcohol into the nerve a little way above the lesion, and into all local branches, after surgical exposure. According to Sicard, this produces a physiological interruption of the nerve, with relief from pain.

There is no doubt that this procedure will relieve pain, but it is a poor substitute for surgical operation, which is aimed at the pathological basis of the condition. Moreover, frequently in causalgia, after alcohol injection, the nerve recovers in a few weeks and the pain returns with renewed intensity. Alcohol injection also is an uncontrollable method of treatment; it lacks the delicate accuracy of the surgeon's knife. It frequently fails, and may cause more fibrosis and so increase the pathological condition it is intended to cure; it

may cause absolute interruption of the nerve, with paralysis and R.D. in the muscular supply, which may never recover spontaneously. In short, it is not possible to estimate the extent of the additional fibrosis and destruction that may occur as a result of alcoholization, or the increased disability that may so be caused. Severe trophic ulceration may also result. It is a clumsy and hardly scientific method, and certainly not to be substituted for surgical procedures. Yet certain observers assert that they have used it with effect, and Lewis and Gatewood claim that in one of three cases of theirs of median causalgia there was no return of pain in four months. They say nothing about return of function, however. Giron uses it in true Weir Mitchell causalgia, and Sicard himself claims to cure 60 per cent of cases by his method.

The injection of cocaine, except for the temporary relief of pain in the worst cases, is not advisable.

Peri-arterial Sympathectomy.—Leriche's and Tenani's operations, or denudation of the associated arteries and veins of their sympathetic fibres as already described, are of no value in true causalgia, although they may be in certain cases of peri-arterial sympathetic neuritis. Tinel, Veyrassat, and Schlesinger report favourably upon its use; but Giron says that the causalgia of Weir Mitchell is not curable by this method, although another type, exclusively sympathetic, yields to denudation of the peri-arterial sympathetic. These operations have been performed on one or two cases of causalgia within the knowledge of the writer, but without success from a curative point of view.

POST-OPERATIVE TREATMENT.

After neurolysis of the nerve in minor cases, and diminution of the pain, ionization of the scar area with 1 per cent sodium chloride solution is a valuable adjunct to the daily treatment of the case. Where the pain is diminishing, it seems to hasten relief and, combined with massage and electrical stimulation of the weak group of muscles, materially aids recovery. Since the muscles always respond to faradism, surging faradism is usually to be employed; but if as a result of neurolysis one or two muscles fail to respond to faradism, as they sometimes do as a result of operative trauma of some nerve fibres, interrupted galvanism is used until faradic excitability returns.

Passive movements are employed for stiffened joints, and use of the limb is encouraged. Useful employment of the hand in some form of curative workshop treatment assists in re-education of muscles and helps co-ordination.

Minor cases rapidly recover good function.

After resection and suture of a nerve, the muscular supply of the nerve will be paralyzed, and complete sensory loss occurs in

the peripheral area of distribution. These cases demand the after-treatment of any nerve lesion after suture. As the nerve begins to regenerate, trophic changes commence to clear up rapidly. Massage, constant current (in the very early stages), and interrupted galvanic stimulation (18 to 25 contractions of each muscle at every treatment) to the paralyzed muscles maintain their tone until they are beginning to be innervated again. When a muscle recovers its excitability to faradism, surging faradism may be used. When the sensory loss has cleared sufficiently to permit of it, the paraffin bath is a great help in improving the circulation and helping the limb to become supple. Stiff joints are helped by passive movements and by diathermy or radiant heat, care being taken to avoid burns of anæsthetic skin. As soon as voluntary power returns, that is, in from nine to eighteen months as a rule, use is encouraged, and, later, re-education movements and the curative workshop treatment are called in to help in the final restoration of function. Obstinate contracture, if this has occurred and cannot be overcome by routine physiotherapeutic treatment, may call for tenotomy or tendon lengthening, combined perhaps with suitable intermittent splinting.

The patient should remain under supervision until every muscle has recovered its motor power.

PROGNOSIS.

The prognosis is excellent in all cases of causalgia, major and minor, due to wounds of nerves, if suitable treatment is undertaken early. Minor cases may clear up in a few months without active intervention at the site of injury, under proper physiotherapeutic treatment.

In major cases, with careful resection followed by end-to-end suture of the nerve under good conditions, the prognosis is that of any good nerve suture in non-painful cases. That is, the end-result is complete recovery of function without recurrence of pain in 90 per cent of cases, in a variable length of time, given suitable post-operative treatment.

In cases where repair of the nerve has to be undertaken by nerve-grafting, although pain is abolished, the prognosis as regards recovery of muscular function is poor. Recovery is very slow and uncertain, and is often arrested before it has gone far. There is usually considerable recovery of sensation in time, and trophic changes clear up slowly.

In cases of long duration, where contracture, bony atrophy, and ischæmic states have developed before operation is undertaken, the end-results may be vitiated by a residuum of permanent contracture, or stiff joints due to articular changes. There may also be so much

fibrous change in some muscles that complete return of muscular function never takes place. But at any rate the pain will be abolished, and the less grave trophic conditions will be much improved.

In cases of the nature of *Case 3*, where there is absence of physical trauma, the prognosis is less favourable, but at the same time much can be done to alleviate the condition. Fortunately these cases are very rare in civilian practice.

NOTES ON THE SENSORY CHANGES IN CAUSALGIA AND ALLIED STATES.

In all cases of causalgia and like conditions there is found some sensory change in the peripheral area of supply of the nerve, because there is interference with the conducting path. There seems to be doubt as to the existence of three different sets of fibres, as theorized by Head and others, for the conduction of three types of sensation—deep, protopathic, epicritic. Cutting the sensory nerves to the skin does not abolish deep sensibility in their area of supply. May this not be because they have no connection with the deeper structures?—e.g., deep sensation in the radial area is supplied chiefly by the median and musculocutaneous nerves. Were it possible to transplant the radial-nerve filaments to the deeper end-organs, there seems no reason why they should not conduct deep sensibility. Again, may not the altered quality of response to stimulus in the peripheral nerve area be due to changed interpretation due to alteration in the power of conduction of the nerve?—perhaps, in the case of tingling and pins-and-needles paræsthesia, to a phenomenon of rapid intermittence of conduction through regions of decrement in the damaged portion of the nerve? The slightest injury to a nerve causes fleeting modifications of its ‘chronaxie’ (or velocity of excitability) and to its power of conduction. The fact that the alteration in quality of response to stimulus is not uniform over the area supplied by the damaged nerve can be explained by unequal trauma—some fibres are injured more than others.

Recovery of epicritic sensibility (so-called) may be incomplete five years after suture of a nerve. Neither has the conductivity of the nerve returned to normal, and even the muscles innervated by the nerve show deficient reaction to condenser currents.

In most cases of causalgia, and in many non-painful partial lesions of nerves, there is no loss of deep sensibility, though there may be complete loss of epicritic and protopathic sensibility. It is hard to imagine why a lesion should be able to select and spare one set of fibres in three.

As a sutured nerve recovers, deep sensibility returns first, then

protopathic, and lastly epicritic, which is what one would expect. As the regenerating nerve recovers its normal conductivity and velocity (or delicacy) of excitability, the finest and most delicate differences of stimuli are appreciated last. Deep sensibility, being a comparatively coarse function, returns first, even when the conductivity of the nerve is much below normal. Absence of so-called epicritic sensation on the glans penis is a useful provision of nature, but there is no reason why it should not be due to lack of sensitivity of the transmitting mechanism rather than to absence of 'epicritic' fibres. Besides, the sensations which are aroused by light stimulation in other parts can be aroused by rather heavier stimulus of the glans; that is, the relatively coarser transmitter requires stronger stimulus to excite it.

Adrian has used short-duration currents to examine the question whether there is more than one excitable mechanism in the afferent nerves of man. The curves obtained showed no indication of two different excitable systems in human afferent nerves. This, while being suggestive, does not disprove Head and Rivers' assumption of protopathic and epicritic fibres.

Without a good deal more evidence it is impossible to jettison the idea of separate fibres for the transmission of different types of sensation, but there seems to be something to be said to the contrary.

The sensory changes in causalgia and allied states are what one would expect when it is considered that the lesion is partial anatomical and physiological division of the nerve. They vary in depth according to the degree of local damage to the nerve; as this is usually relatively slight, deep sensibility is as a rule undisturbed.

1. Light Touch.—There is invariably disturbance in this direction. Sensibility to light touch may be absent in certain areas, or there may be simply hypo-æsthesia. As a rule, light touch stimulus in the peripheral area of the nerve affected in causalgia gives rise to that altered quality of response known as paræsthesia. The response seems more vivid, but the threshold is higher than normal.

2. Sensation to Pin-prick.—Is always altered in these cases over a large area. This is to be expected, sensation to pin-prick being a compound one composed of a sense of 'pressure' and a sensation of 'sharpness', the latter of which belongs to the epicritic class of discriminating sensibility. The change of response to pin-prick extends always over a much greater area than the absolute loss to light touch. After the complete surgical division of a nerve it was found that the margin at which pin-prick is felt normally is just outside the margin of total loss to light touch, and not vice versa as Stopford and Core state. Pin-prick is felt as a blunt sensation, it is true, inside the margin of total loss to light touch, but along this line the response is not normal.

3. **Thermal Sensibility.**—Reliable readings could not be obtained, but there was undoubtedly some disturbance.

4. **Deep Sensibility.**—

a. *Pressure.*—Undisturbed as a rule, though responses masked and changed by superficial sensory disturbances.

b. *Roughness.*—The radiating tingling and other sensory abnormalities made readings unreliable, though there seemed to be some diminution in this sense.

c. *Localization.*—Disturbed by the misleading references caused by the paræsthesia and abnormality of response to touch. Deep localization probably not impaired.

d. *Joint and Muscle Sense, Bone Sense.*—Unimpaired as a rule. Bone sense to tuning-fork vibration scarcely ever showed any change from the normal.

5. **Discriminating Sensibility.**—

a. *Size and Shape.*—Disturbed by reason of the pain and paræsthesia causing aberrant impressions. There was never absolute astereognosis.

b. *Texture.*—Recognition depending upon retention of finer sensibility, this sense was also disturbed.

6. **Weber's Test.**—There was generally some disturbance in the accurate recognition of two blunted points applied simultaneously, provided pressure sense was not evoked.

The examination of deep sensibility in conditions where superficial response to stimulus is perverted is a matter of great difficulty, because of the superficial reference sensations set up in applying the test, before deep sensibility is stimulated. Generally speaking, in causalgia and like conditions there is marked disturbance of the more superficial sensibility, and deep sensibility shows no change except in its finer degrees, such as in the 'blunt' sensation part of the compound sensation of pin-prick.

CONCLUSIONS.

The fundamental lesion in causalgia and allied conditions is one of intraneural and perineural sclerosis, and the irritation thus set up of fibres at the site of injury to the nerve causes perverted afferent impulses to be sent back to the cord, and possibly further to the subcortical and cortical centres, and thence efferent responses of vasodilator, secretory, and trophic natures are reflected to the peripheral distribution of the nerve, where reaction on the end-organs and sensory corpuscles in this area is interpreted as pain.

The reason why lesions of this type of the median nerve and internal popliteal fibres produce a true causalgia in some cases, pain of less severity in others, and in the remainder nothing but the

discomfort of partial sensory loss, is difficult to answer. It is improbable that inflammatory reaction to different groups of organisms provides a solution. It hardly seems possible that irritation of a special group of fibres is the cause, for causalgia is rare, and it is not within the range of probability that such fibres should be so seldom involved. The most that can be said is that there is some peculiarity in the trauma of the nerve in these few cases, which causes disturbance of those cells in the brain whose function it is to interpret as pain.

By resection of the irritated and sclerosed portion of nerve, the source of the perverted impulses is removed and the pain vanishes; and by restoring the continuity of the nerve, the path for normal impulses is re-made and gradually recovers its conductivity as the nerve regenerates.

I have to thank surgical colleagues at the 2nd Northern General Hospital for their courtesy in allowing access to operations, and operation notes, and Dr. J. le F. Burrow for drawing my attention to the method of using colours in charting sensory changes. The diagrams used for charting were kindly supplied by the Medical Research Committee.

TABULAR LIST

Table I.—MAJOR

Case Number, Name, Date, Nature of Wound, etc.	Duration of Condition.	History and Treatment before coming under observation.	SIGNS AND SYMPTOMS AT EXAMINATION.					
			Pain.		Motor Changes.			Sensory Changes.
			Time of Onset after Injury.	Description.	Voluntary Power.	Faradic Responses.	Galvanic Response.	
<i>Case 1.</i> W. H., age 36, G.S.W. left upper arm. 9/10/17.	15 months.	Jan., 1918: Neurolysis and stretching median nerve. Ap. '18: Intraneural injection 60 per cent alcohol below site of injury. Massage, electrical, whirlpool and paraffin baths. No relief.	10 days	Intense burning smarting pain in usual area, worse on warm days.	Power in all median muscles; little atrophy; slight contracture; marked tremor both hands.	All muscles react, diminished responses.	K.C.C. > A.C.C., brisk above site of alcohol injection. Thenar muscles, A.C.C. > K.C.C. sluggish No R.D.	No deep loss. Some loss to light touch. Much paresthesia in typical area. Localization poor. Weber's test: typical disturbance.
<i>Case 2.</i> A. D., age 26. G.S.W. Right knee. 22/8/17.	16 months.	Drop-foot developed immediately. External popliteal nerve said to have been sutured. Oct. '17: Massage, interrupted galvanism; no relief. No treatment for internal popliteal nerve.	14 days.	Intense continuous burning pain in sole. Some aching of dorsum. Worse warm dry days.	All muscles supplied by both nerves have degree of power.	As Case 1. Responses sluggish.	Long muscles K.C.C. > A.C.C. sharp. Small muscles A.C.C. > K.C.C. sluggish. No R.D.	As above applied to internal popliteal area. Some disturbance external popliteal area.
<i>Case 3.</i> Mrs. W., age 60. No injury.	7 years.	Arose in 1912 after attack of cholelithiasis. Pain left median area. Local application, anodal galvanism. Drug treatment. Nov., 1917: Intraneural alcohol upper arm. Mar., 1918: Alcohol injection repeated. Oct., 1918: Left median nerve divided and resutured.	—	Intolerable constant burning pain in median area and in index and thumb. Worse dry warm days.	All muscles had power before nerve division.	All muscles reacted to faradism.	All muscles K.C.C. > A.C.C. Fairly brisk responses.	No deep loss when first seen. Nerve recovering after suture. Some loss to light touch and much paresthesia. Localization poor. Weber's test: typical disturbance.
<i>Case 4.</i> E. V. H., age 24. G.S.W. left elbow. 15/9/16.	3 months.	Local applications, anodal galvanic baths. Not thought at this time there was any serious nerve lesion.	9 days.	Intolerable constant burning pain, usual area. Warmth makes it worse.	Degree of power everywhere. Flexor long. pollicis and thenar group very feeble. Commencing contracture.	All but flexor long. pollicis and thenar group react.	Perverted responses. No R.D.	Paresthesia over whole median area. Localization poor. Weber's test: typical disturbance.
<i>Case 5.</i> Pte. A., age 21. G.S.W. left shoulder. 2/11/14.	6 weeks	Treatment of wound only.	14 days.	Severe burning neuralgic pain in usual median area. Warmth makes it worse.	Paresis. Atrophy thenar group. Slight contracture.	Median muscles all react.	A.C.C. > K.C.C. No R.D.	Sensory changes in area of inner cord of brachial plexus. No deep loss. Paresthesia median area.

OF CASES.

CAUSALGIA.

Vasomotor and Trophic Changes.	Mental and Emotional State.	TREATMENT.		Operative Findings.	Post-operative Treatment.	Progress.
		Non-Surgical.	Surgical.			
Conical fingers, curved ridged nails, skin thin, pink, and glossy. Hyperidrosis. Stiff joints.	Nervous, jumpy, worried. Dreads sudden stimulus. Protective attitude. Carried wet rag.	Nil.	Jan. 16, 1919: Resection of hard scarred portion of nerve, followed by end-to-end suture.	1 in. median nerve found hard and scarred in lower third upper arm. Much perineural scarring.	Massage, interrupted galvanism, and later faradism, warm-water baths. Finally exercises and re-education	Jan. 29, 1919: No pain, full median motor and sensory paralysis. Jan., 1920: Nerve recovering, no pain, all muscles degree of power and faradic response. Trophic changes much improved. Residual sensory loss. Joints rather stiff.
Skin of sole dead-looking, smooth and sodden. Nails dull and brittle. Whole foot has lost character, and is smaller than normal. Toes fallen together. Hyperidrosis. Branny desquamation on dorsum.	Patient nervous, scared-looking, and apprehensive. Protective attitude. Intolerant of investigation, and emotional to tears. Carried wet rag.	Nil.	Dec. 12, 1918: Neurolysis both internal and external popliteal nerves. Sepsis followed. No relief. Jan., 1919: internal popliteal nerve divided and left so. Patient did not allow further operation.	Intense perineural scarring both nerves, and much intraneural scarring internal popliteal nerve.	Massage, anodal galvanic baths, and radiant heat.	May, 1919: All external popliteal muscles acting. Full motor and sensory loss internal popliteal supply. No burning pain. Some aching of dorsum. May, 1920: No pain. Paralysis of internal popliteal muscles. Patient working and getting on well. 1921: free from pain.
Conical fingers, curved nails, skin thin and pink. Crops of pin-head vesicles arising spontaneously. Sweating of palm.	Fairly good.	Anodal galvanic baths.	Dec., 1918: Left median nerve again divided higher up and again sutured. No resection.	Not seen. Condition said to be due to gouty deposits in the nerve.	Anodal galvanic baths, gentle massage and movements of fingers.	1919: Pain both means so severe, and easily comforted. Burning character lost. Aching still. Median nerve not generating. Some forearm muscles have power. Thenar group atrophied and paralyzed.
Typical trophic and vasomotor changes.	Bad. Pronounced mentally deficient before cause recognized. Very emotional and apprehensive. Protective attitude. Carried wet rag.	Dia-thermy; no relief. Anodal baths; no lasting benefit.	Dec., 1916: Neurolysis left median nerve. No relief. Mar. 4, 1918: Resection and suture.	2½ in. left median nerve found involved in scar tissue and containing fibrous nodules.	Massage, passive movements. Interrupted galvanism, anodal baths.	1918, late: Invalid from service. No pain now. Mental state normal. Paralysis of flexor long. pollicis and thenar group. Recovering median nerve. Not seen again.
Skin bluish-pink and sodden, glossy on dorsum, fingers conical. Nails ridged and brittle. Hand approaches bird-claw type. Joints rather stiff.	Fairly good. Carried wet rag in palm.	Nil.	Dec. 18, 1914: Inner cord brachial plexus resected and then sutured.	2 in. inner cord found embedded in young fibrous tissue. Some intraneural scarring. Brachial artery collapsed and pulseless.	Massage, interrupted galvanism, later faradism. Exercises.	May 25, 1915: pain except of minor character. Jan. 1919: Patient working. All muscles degree of power. most perfect functioning hand. Sensory return not quite perfect.

Table II.—MINOR CAUSALGIA AND

Case Number, Name, Date, Nature of Wound, etc.	Duration of Condition.	History and Treatment before coming under observation.	SIGNS AND SYMPTOMS AT EXAMINATION.					
			Pain.		Motor Changes.			Sensory Changes.
			Time of Onset after Injury.	Description.	Voluntary Power.	Faradic Responses.	Galvanic Response.	
Case 6. G. W. G.S.W. right upper arm 31/3/18	3 months	Treatment of wound only.	10 days	Severe neuralgic pain. Variable. Worse cold days.	All median muscles have power.	All give responses.	K.C.C. > A.C.C., rather slow.	Paræsthesia to all forms of stimulation over median area. No total loss of light touch.
Case 7. W. O. H. G.S.W. right buttock, 13/11/16	12 months.	Neurolysis right sciatic nerve. Mar. 10, 1917: No relief. Neurolysis repeated June 24, 1917: No relief.	7 days	As above, in sole area. Calf muscles painful.	External popliteal muscles paralyzed. Internal popliteal all power. Some atrophy.	Nil in external popliteal group. Others react.	Not tested owing to pain.	Much paræsthesia in internal popliteal area. Total loss external popliteal area.
Case 8. J. W. H. G.S.W. right wrist, 27/11/17	21 months.	Aug., 1918: Neurolysis rt. median nerve. Some relief. March, 1919: Neurolysis repeated. Massage, ionization, anodal baths.	7 months.	Sensitive scar at wrist. Pain in palm and spasm of fingers. Aching of wrist.	Paresis of thenar muscles.	Nil.	A.C.C. > K.C.C., sluggish. No R.D.	A little total loss of light touch. Elsewhere intense paræsthesia.
Case 9. A. H. G.S.W. right thigh, 21/6/17.	10 months.	Treatment of wound only.	14 days	Severe neuralgic pain on sole. No burning quality.	All muscles very feeble.	Most muscles reacted feebly.	K.C.C. > A.C.C., sluggish. No R.D.	Some loss of light touch and paræsthesia, but internal popliteal area. Slight paræsthesia, external popliteal area.
Case 10. E. M. G.S.W. right knee, 20/9/17.	3 months.	Treatment of wound only.	7 days.	Intense aching pain in usual spot on sole.	All sciatic muscles feeble power.	Nil. Small currents only tried owing to intolerance.	Nil.	No total loss to light touch. Hypo- and paræsthesia.
Case 11. T. J. G.S.W. left upper arm, 4/11/18.	3 months.	Treatment of wound only.	14 days.	Gripping, burning pain, median area, eased by warmth.	All acting. Thenar group feeble and atrophied.	All reacted except thenar group.	A.C.C. > K.C.C., fairly brisk.	Loss light touch index finger. Paræsthesia usual area. Elsewhere hypo-æsthesia. Localization poor. Discriminating sensibility: not good.
Case 12. G. E. F. G.S.W. right upper arm, 18/8/16.	23 months.	Massage, interrupted galvanism, static breeze, ionization, high-frequency current, x-ray treatment, diathermy. No real benefit, but pain got more bearable as time went on.	4 days.	Constant severe burning pain in usual area.	All had power.	All muscles reacted.	—	Paræsthesia over whole median area. No loss to light touch. Localization poor. Discriminating sensibility diminished.

ALLIED PAINFUL CONDITIONS.

Vasomotor and Trophic Changes.	Mental and Emotional State.	TREATMENT.		Operative Findings.	Post-operative Treatment.	Progress.
		Non-Surgical.	Surgical.			
Skin pink and mottled in palm. Fingers conical. Nails dull and curved. Little flexor contracture.	Apprehensive. Protective attitude. Hyper-sensitive to sudden stimuli.	Nil.	July 5, 1918: Neurolysis right median nerve. Section not deemed advisable.	Much perineural scar tissue, but the stripped nerve smooth and soft.	Massage, anodal baths, interrupted galvanism, faradism, re-education exercises.	Sept. 4, 1918: Pain almost gone. All muscles acting. Little contracture. Nov. 12, 1918: No pain. Contracture worse.
Skin pink-purple. No sweating. Nails dull and brittle. Delayed shedding of epithelium.	Apprehensive. Protective attitude. Intolerant of examination.	Nil.	Nov. 23, 1917: Resection and suture external popliteal nerve. Intraneural alcohol internal popliteal nerve, after neurolysis.	External popliteal nerve divided. Internal popliteal nerve bound up in scar, but soft when stripped.	Anodal galvanic baths. Ionization of scar area.	Pain returned in week. May 30, 1918: Pain better. Sensory loss deeper. Paralysis all muscles. Mar. 28, 1920: Pain gone. Paralysis unchanged. Nerve not regenerating after alcohol. Trophic sore on little toe.
Very little. Skin damper and pinker than normal.	Very jumpy and emotional, and intolerant of examination	Nil.	Mar. 13, 1920: Resection right median nerve, followed by suture.	Nerve badly swollen and scarred. Perineural and intraneural scarring.	Massage and movements. Interrupted galvanism small muscles.	April 23, 1920: Much better. No pain. Mental state better. Some tingling on percussion of scar. Aug. 25, 1920: Nerve regenerating. Pain.
Foot swollen, blue, and cold. Nails dry and broken. No sweating. Slight contracture.	Good.	Massage, ionization, galvanism. No relief. Sensory changes more profound.	Aug., 1918: Resection right sciatic nerve followed by suture.	1½ in. of nerve badly scarred and involved in surrounding fibrous tissue, which was hard.	Massage, interrupted galvanism, warm-water baths.	Mar., 1919: Pain. Foot still blue and oedematous. Jan. 1919: Nerve regenerating. Power gastrocnemius. Dec., 1919: No return of pain.
Foot swollen, glossy skin on toes. Nails curved, dry, and ridged. No sweating.	Intolerant of handling; otherwise good.	Nil.	Jan. 5, 1918: Neurolysis both branches of sciatic nerve. No relief. June 30, 1918: Resection both nerves and suture.	Both nerves involved in much scar at bifurcation of sciatic. Bulk of each nerve healthy-looking.	Massage, interrupted galvanism, etc.	Sept. 5, 1918: Pain gone. Oct. 3, 1918: No return of pain. Nerves regenerating.
Skin of palm pink, damp, and delicate looking. Delayed shedding of epithelium.	Apprehensive. Protective attitude. Does not stir out of hosp. grounds	Anodal galvanic baths, with some relief.	Nil.	—	—	Some relief from non-surgical treatment. Patient very shortly transferred and lost sight of.
Very marked. Hand stiff and wooden. Joints very bad—obvious joint changes. Conical fingers. Nails dull and ridged. Skin thin and glossy. Sweating of palm.	Apprehensive, nervous. Sensitive to least jarring or other stimulus. Better than has been when pain at its height.	Paraffin baths, massage and movements. Anodal baths, and later faradism.	Passive movement of joints under anæsthetic. Result poor.	Too late for surgical help. Should have been undertaken earlier.		Dec., 1919: Pain almost gone, but hand unfit for useful purposes owing to permanently damaged joints. Early resection and suture with suitable after-treatment would probably have given a useful hand.

Table II.—MINOR CAUSALGIA AND

Case, Number, Name, Date, Nature of Wound, etc.	Duration of Condition.	History and Treatment before coming under observation.	SIGNS AND SYMPTOMS AT EXAMINATION.					
			Pain.		Motor Changes.			Sensory Changes.
			Time of Onset after Injury.	Description.	Voluntary Power.	Faradic Responses.	Galvanic Response.	
Case 13. M. A. G.S.W. right forearm. 16/9/16.	12 months.	Not known.	14 days.	Constant aching pain, usual area.	Median muscles all acting.	Diminished reactions.	A.C.C. > K.C.C., sluggish.	Paræsthesia whole median and ulnar areas. No total loss of light touch. Localization and discriminating sensibility poor.
Case 14. P. S. G.S.W. left forearm. 6/1/17.	14 months.	Not known.	10 days.	Severe aching pain in palm.	Paresis median muscles.	Reduced reactions.	—	Paræsthesia median area. Poor localization and discriminating sensibility.
Case 15. J. W. G.S.W. left upper arm. 6/1/17.	15 months.	Treatment for wound only.	1 year.	Constant boring pain in palm.	Paresis median muscles. Slight contracture.	All responded to faradism.	—	Hypoæsthesia whole median area. Paræsthesia to strong stimulation.
Case 16. R. D. J. Shell wound left upper arm. 22/3/18.	10 weeks.	Treatment for wound only.	11 days.	Constant burning pain, usual area.	Paresis median muscles.	Poor responses.	K.C.C. > A.C.C., fairly brisk.	Hypoæsthesia as above, and paræsthesia to stronger stimulation in typical area.
Case 17. W. L. G.S.W. right forearm. 28/8/17.	9 months.	Treatment for wound only.	1 month.	Severe aching pain in palm.	Paresis median muscles. Slight contracture.	Poor responses.	—	Paræsthesia all over median area. Localization poor.
Case 18. J. H. S. G.S.W. left upper arm. 26/9/17.	3 months.	Treatment for wound only.	7 days.	Dull aching pain usual area.	No paralysis of muscles.	All gave responses.	—	Paræsthesia over whole median area. Localization good. No astereognosis.
Case 19. F. W. S. G.S.W. left forearm. 28/12/17.	2½ months.	Treatment for wound only.	2 months.	Throbbing bursting pain. Worse in cold weather.	Thenar group feeble and atrophied.	Thenar group no responses.	K.C.C. > A.C.C., brisk.	Some loss of light touch, which is increasing. Paræsthesia to strong stimulus. Hypoæsthesia radial area. Localization poor.
Case 20. A. W. A. G.S.W. right upper arm. 26/11/17.	4 months.	Treatment for wound only.	21 days.	Intense aching pain in palm, getting worse.	Paresis only median group.	All gave responses.	A.C.C. > K.C.C., brisk.	Paræsthesia most of median area. Some hypoæsthesia. Loss of light touch - internal cutaneous area.
Case 21. Pte. W. G.S.W. left upper arm. 22/5/18.	12 months.	Treatment for wound only, and a little massage.	14 days.	Constant aching pain in palm.	Paresis only median muscles.	All gave responses.	—	Paræsthesia whole median area. Deficient localization and discriminating sensibility.

ALLIED PAINFUL CONDITIONS—*continued.*

		TREATMENT.		Operative Findings.	Post-operative Treatment.	Progress.
Vasomotor and Trophic Changes.	Mental and Emotional State.	Non-Surgical.	Surgical.			
Tapering fingers, nails curved and overgrown, skin pink, thin, and glossy. Branny desquamation of palm.	General health suffering from pain.	Nil.	Sept. 8, 1917: Resection followed by suture, median nerve; neurolysis ulnar.	Large bulb involving whole circumference of median. Nerve fibres not distinguishable in mass. Ulnar bound in scar.	Massage, interrupted galvanism, surging faradism.	Nov. 19, 1917: No pain. Full median motor and sensory loss. June 4, 1918: No pain. Median nerve regenerating. Ulnar nerve recovered.
Tapering index and thumb, arching of nails, sweating of palm. Skin very pink.	Fairly good.	Nil.	Nov. 5, 1917: Neurolysis left median nerve.	Much perineural scarring. Small lateral bulb on nerve.	Diathermy scar area, massage, anodal baths.	Jan. 1, 1918: Pain reduced. Feb. 8, 1918: Pain almost gone. Hand comfortable except for some tingling of palm.
Tapering median fingers, arched ridged nails, glossy skin.	Good.	Nil.	June 1, 1918: Neurolysis left median nerve, which was placed in new muscle bed.	2½ in. of nerve seemed very poor but resection not done. Musculo-cutaneous nerve found damaged.	Massage, anodal baths, galvanism, etc.	Sept. 17, 1918: Pain insignificant. All muscles acting well. Hand becoming useful.
Skin of palm pink and sweating. Minute trophic vesicles in median area.	Good.	Anodal baths, Ionization of scar area, massage, etc.	Nil.	—	—	Nov. 13, 1918: Pain practically gone. Trophic blisters cleared up. Inability to extend index properly.
Conical fingers, skin of palm smooth and bluish-pink. Nails brittle. Hyperidrosis.	Good, but reluctant to be examined.	Nil.	May 27, 1918: Resection right median nerve, followed by suture.	About 2 in. of nerve embedded in scar tissue. Some hardness of nerve when stripped.	Massage, galvanism, etc.	Aug. 2, 1918: No pain. Dec. 21, 1918: No pain. Power in forearm flexors. May 13, 1919: No return of pain, nerve regenerating.
No trophic changes of note.	Good.	Ionization scar area and anodal baths; massage	Nil.	—	—	Pain gone in a few months. No operation necessary.
No marked trophic changes. Some delayed shedding of epithelium.	Good.	Nil.	June 10, 1918: Resection left median nerve, followed by suture.	Small lateral bulb found on median nerve. Perineural scarring.	Massage, galvanism, etc.	July 3, 1918: No pain. Nov. 18: No pain. Nerve regenerating. Feb. 8, 1919: No return of pain. Progressive regeneration.
Tapering fingers, glossy skin. No hyperidrosis. Skin mottled pink.	Good.	Anodal galvanic baths and massage. No relief.	June 13, 1918: Neurolysis right median nerve.	A little perineural scar tissue. No naked-eye lesion of cleaned nerve.	Anodal baths, ionization scar area, massage.	Pain cured by Feb. 10, 1919. Muscles more power. Some residual hypoaesthesia.
Skin sodden-looking and sweating. No contracture.	Good.	Anodal baths, ionization, diathermy, exercises, work-shops.	Nil.	—	—	Much improvement in 2 months. Condition rapidly cleared up.

Table II.—MINOR CAUSALGIA AND

Case, Number, Name, Date, Nature of Wound, etc	Duration of Condition	History and Treatment before coming under observation	SIGNS AND SYMPTOMS AT EXAMINATION.					
			Pain.		Motor Changes.			Sensory Changes.
			Time of Onset after Injury	Description.	Voluntary Power.	Paradic Responses.	Galvanic Response.	
Case 22. J. W. S. G.S.W. right elbow. 15/7/17.	7 months.	Treatment for wound only, and a little massage.	—	Unpleasant sensations in palm. No real pain now.	Paresis median muscles. Flexor contracture Atrophy.	All gave responses.	—	Paræsthesia median and ulnar areas, chiefly for mer. Some total loss light touch.
Case 23. H. R. C. G.S.W. right elbow. 13/11/16.	5 months.	Treatment for wound only.	10 days.	Severe aching pain, median area.	Paresis only, little contracture, and atrophy.	All gave responses.	A.C.C. > K.C.C. thenar group, but brisk.	Paræsthesia median area. Deficient localization and discriminating sensibility. Little total loss light touch.
Case 24. W. W. G.S.W. left elbow. 4/3/17.	9 months.	Typical history minor causalgia relieved by blocking nerve with 60 per cent alcohol.	8 days.	Had had typical pain.	Paralysis muscles, due to alcohol.	Nil	A.C.C. > K.C.C., sluggish. No R.D.	Paræsthesia turned to total loss by alcohol injection.

BIBLIOGRAPHY.

- ABERCROMBIE, R. G., "Physiopathic Paralysis", *Lancet*, 1920, ii, 764.
- ADRIAN, E. D., "Response of Human Sensory Nerves to Currents of Short Duration", *Jour. of Physiol.*, 1919, liii, 70.
- BABINSKI, J., and FROMENT, J., *Hysteria or Pithiatism*, Eng. Trans., London, 1918.
- BÉNISTY, ATHANASSIO, *Clinical Forms of Nerve Lesions*, *Treatment and Repair of Nerve Lesions*, Eng. Trans., London, 1918.
- BROWN, W. LANGDON, *The Sympathetic Nervous System in Disease*, London, 1920.
- BURROW, J. LE F., "War Lesions of Peripheral Nerves", *Med. Record*, 1919, May 31.
- BURROW, J. LE F., and CARTER, H. S., "Preliminary Note upon One Thousand Cases of Peripheral Nerve Injury", *Brit. Med. Jour.*, 1918, Nov. 16.
- CONE, SYDNEY M., "Surgical Pathology of Peripheral Nerves", *Brit. Jour. Surg.*, 1918, v, 524.
- CORE, D. E., "Dissociation of Cutaneous Sensations in Injuries to Peripheral Nerves", *Lancet*, 1916, April 1.
- CORNER, E. W., *Brit. Jour. Surg.*, 1918, vi, 277.
- GIRON, *Presse méd.*, 1918, Nov. 14, 584 (ext. *Med. Annual*, 1919, 286).
- HARRIS, W., *Lancet*, 1915, Nov. 13.
- HEAD, H., *Studies in Neurology*, London, 1920.
- HUMPHRIS, F. H., "The Melted Paraffin Wax Bath", *Brit. Med. Jour.*, 1920, Sept. 11.
- JOYCE, J. L., "A Study of a Series of Peripheral Nerve Injuries from a Surgical Standpoint", *Brit. Jour. Surg.*, 1919, vi, 418.
- KRAMER, J. G., and TODD, T. W., "Distribution of Nerves to Arteries of the Arm", *Anal. Record*, 1914, May.
- LEWIS and GATEWOOD, "Treatment of Causalgia", *Jour. Amer. Med. Assoc.*, 1920, lxxiv, 1.
- LUCAS, KEITH, *The Conduction of the Nervous Impulse*, London, 1917.

ALLIED PAINFUL CONDITIONS—*continued.*

Vasomotor and Trophic Changes	Mental and Emotional State.	TREATMENT.		Operative Findings.	Post-operative Treatment.	Progress
		Non-Surgical.	Surgical.			
Skin dry, nails dry, curved, and brittle, fingers tapering, hand stiff and wooden.	Good.	Massage, ionization, diathermy, anodal baths.	Nil.	—	—	Much improvement very soon. Paræsthesia given way to hypo-æsthesia. Full movement of joints not obtained.
Skin sodden-looking, and bluish-pink; hyperidrosis; nails curved, ridged, and long.	Good.	Nil.	July 2, 1917: Neurolysis right median nerve.	Nerve found bound down in scar tissue and somewhat bulbous.	Anodal baths, massage, ionization, galvanism.	Sept. 11, 1917: No pain, all muscles acting. Sensory loss clearing. June 14, 1918: Good hand, no pain, all muscles acting, joints free.
Typical trophic changes well marked.	Good.	Interrupted galvanism, massage, movements.	Nil.	—	—	Nerve recovering very slowly from devastating effects alcohol. Few signs recovery in 12 months though no pain.

MACKENZIE, SIR JAMES, "Disturbed Reflexes in the Production of Symptoms", *Brit. Med. Jour.*, 1921, Jan. 29.

MITCHELL, WEIR, *Gunshot Wounds and Other Injuries of Nerves*, 1864.

MURPHY, J. B., "Neurological Surgery", *Surg. Gynecol. and Obst.*, 1907, iv, No. 4.

NUNNELEY, F. P., "The Whirlpool Bath", *Brit. Med. Jour.*, 1916, Nov. 25.

POTTS, L. W., "Distribution of Nerves to Arteries of Leg", *Anat. Anzeiger*, 1914.

PATTERSON, A. M., *Anatomy of the Peripheral Nerves*, London, 1919.

SHERREN, J., *Injuries of Nerves and their Treatment*, London, 1908.

SONNTAG, C. F., "Physiological Action of Whirlpool Baths", *Med. Press*, 1918, March 20 and 27.

SOUTTAR, H. S., and TWINING, E. W., *Injuries of the Peripheral Nerves*, Bristol, 1920.

STEVENSON, W. C., "Effect of Radium Treatment on War Injuries in the Neighbourhood of Nerves", *Lancet*, 1920, ii, 862.

STEWART, PURVES, and EVANS, A., *Nerve Injuries and their Treatment*, London, 1919.

STEWART, PURVES, *Diagnosis of Nervous Diseases*, London, 1916.

STOPFORD, J. S., "Thermalgia (Causalgia)", *Lancet*, 1917, ii, 195.

STOPFORD, J. S., "Preliminary Note on Trophic Disturbances in Gunshot Injuries of Peripheral Nerves", *Lancet*, 1918, March 3.

STOPFORD, J. S., "Gunshot Injuries of Peripheral Nerves; the Syndrome of Compression", *Lancet*, 1916, April 1.

STOPFORD, J. S., "Late Changes in Cases of Thermalgia", *Lancet*, 1918, March 3.

TENANI, O., *Polichinico* (Pract. Sect.), 1918, Feb. 10.

THOMPSON, H. C., *Diseases of the Nervous System*, London, 1915.

TINNEL, J., *Nerve Wounds*, Eng. Trans., London, 1917.

TODD, T. B., "Blood-vessel Changes consequent on Nervous Lesions", *Jour. Nerv. and Ment. Dis.*, 1913; *Lancet*, 1912, ii, 362; *Ibid.*, 1913, i, 1371.

VEYRASSAT and SCHLESINGER, *Jour. Amer. Med. Assoc.*, 1917, i, 1214 (ext. *Med. Annual*, 1918, 528).

FIG. 1.—Diagram to show the average typical area in which the intense pain is felt in median causalgia.

FIG. 2.—Diagram to show the average typical area in which the intense pain is felt in sciatic and internal popliteal causalgia.

PLATE I

Case 1.—Diagram to show:—

- (1) The method of recording sensory loss by means of colours (in practice ordinary coloured crayons are used).
- (2) The sensory disturbances in a typical case of median causalgia.

Red = Total loss of sensation to light touch.

Brown = Hypo-aesthesia to light touch.

Green = Paraesthesia and altered quality of sensation to light touch. (Coarser stimulation, of course, also evokes paraesthesia in this area.)

Degrees of variations in sensation may be indicated by varying shades of the appropriate colours.

O indicates that pressure causes paraesthesia.

X indicates that pinprick causes paraesthesia.

\ indicates that coarse scraping causes paraesthesia.

(It will be seen that in the whole of the outlying area of hypo-aesthesia both pressure and scraping evoke paraesthesia, and that at the tip of the ring finger pinprick does also.)

A = The margin at which pinprick is felt normally. In places (indicated by radiating green lines), paraesthesia is immediately evoked.

B = The margin at which pinprick is felt as a blunt sensation: e.g., in exploring the index finger with the spring aesthesiometer (at mark 8), no sensation is evoked until the level of line B is reached, when the feeling is described as blunt. On the second finger, pinprick is felt as blunt all the way from the tip to the line A, when it becomes sharp.

C = Outer dotted line enclosing the typical painful area.

D = Inner dotted lines enclosing the areas of maximum pain and tenderness (dark green).

PLATE II

Case 1.—Typical appearance of hand in median causalgia. Note conical fingers, overgrown curved nails, trophic changes in skin, and slight contracture.

Case 2.—Sole of the right foot in a case of internal popliteal causalgia. Note the general atrophy of foot in comparison with the left foot, with falling together of the toes and alteration in character of the skin surface.

Case 3.—The hand in an old-standing non-traumatic case of median causalgia. Note the skin surface, the trophic blisters, and a slight degree of contracture.

Case 4.—The hand in a very severe case of median causalgia, showing the typical appearance of the skin, and the contracture.

PLATE III

Case 6.—Appearance of the hand in a case of minor median causalgia.

Case 10.—Left foot in a case of minor causalgia of the internal popliteal nerve. Note glossy skin over the toes, slight flexor contracture, trophic ulcer and general appearance of skin surface.

Case 13.—A hand in a case of minor causalgia of the median nerve, with a mild irritative lesion of the ulnar nerve.

Case 24.—Showing a hand in a case of minor median causalgia ten months after the intra-neural injection of 60 per cent alcohol. Note the persistence of trophic changes.

PLATE IV

MICROPHOTOGRAPHS OF SECTIONS OF EXCISED PORTIONS OF NERVES

Case 1a, b.—Showing intrafascicular and perifascicular fibrosis, with nerve fibrils struggling through a mass of fibrous tissue.

Case 4a, b.—Showing intrafascicular fibrosis with breaking up and distortion of nerve fibres.

Case 9.—Excised portion of sciatic nerve cut transversely, showing perivascular fibrosis with compression and obliteration of nerve bundles.

Case 13.—Showing intrafascicular and perifascicular fibrosis with compression and distortion of nerve fibres.

Case 17.—Section cut obliquely, showing nerve fibres in a mass of fibrous tissue.