Original Articles

CAUSALGIA

A REPORT ON 32 CASES

By H. R. PASRICHA

LIEUTENANT-COLONEL, I.M.S.

SINCE Mitchell *et al.* described causalgia in 1864, the opportunities to study this condition in detail have not been many. The following report is based on a series of 32 cases which came under my care during 1944 and early part of 1945. After March 1945 no cases were received up to the end of the year, a very important fact. As I shall explain later this can be definitely correlated with the general use of penicillin in forward areas. It is more than likely that infection in wounds of the nerves plays a very important part in the ætiology of causalgia.

All these cases were soldiers who had been wounded on the various battle-fields during the period mentioned above. Out of 32 cases 18 were Gurkhas, a preponderance which cannot be easily explained by assuming a larger number of Gurkha troops employed. For quite a long time it was not possible to offer any explanation for this increased liability of Gurkhas to this disorder. Later it was realized that Gurkhas as a race had a marked tendency to excessive scarring and keloid formation. A census of all the Gurkha patients suffering from all varieties of wounds revealed that out of 95 cases 38 showed a well-marked evidence of keloid, an incidence far exceeding any seen in Indian troops. The preponderance of causalgia in Gurkhas may be explained by assuming that just as they are more liable to form keloids so they are more likely to develop excessive intra-neural fibrosis. Support is lent to this view by the fact that the use of penicillin, since it reduced infection, reduced intra-neural scarring also. This is the reason why cases of causalgia stopped coming to this centre after a general use of penicillin in the forward areas.

Incidence

In a series of 1,390 cases of peripheral nerve injury 32 (2 per cent) had causalgia. It is rather a high percentage, but may be explained partly by the fact that some of the nerve cases were treated elsewhere, whereas most of the cases of causalgia were sent to us. Their distribution was as follows :—

Nerve involved	Number of cases
Median	19
Ulnar	1
Tibial	12
TOTAL ..	32

Types of missiles	Number of cases
Gun-shot wounds	16
Shell or bomb splinters	6
Grenade wounds	10
TOTAL ..	32

SITE OF INJURY

Upper extremity	Cases	Lower extremity	Cases
Neck ..	3	Buttock ..	3
Shoulder ..	3	Thigh ..	6
Axilla ..	1	Leg ..	2
Arm ..	10	Multiple ..	1
Forearm ..	2		
Hand ..	1	TOTAL ..	12
TOTAL ..	20		

Age incidence.—All the patients were young men between the ages of 19 and 40, the average being 24. The numbers in accordance to decades are as follows :—

	Cases
11 to 20	5
21 to 30	24
31 to 40	3
TOTAL ..	32

Associated Lesions

Compound fractures.—There were four cases with compound fractures : radial 1; ulnar 1; humerus and ulnar 1; clavicle 1.

Associated nerve lesions :—

	Cases
All branches of the brachial plexus ..	4
Radial nerve	1
Median nerve	4
Ulnar nerve	4
Median and ulnar nerves	3
Sciatic nerve	9
Tibial nerve	2
Peroneal nerve	1
No nerve damage was detected clinically in	4
TOTAL ..	32

This shows that although 19 cases were of median causalgia, only four revealed clinical evidence of damage to the median nerve alone. In another seven, the damage to other nerves was associated with damage to the median nerve. Four cases showed no signs, motor or sensory, suggesting any injury to any of the nerves of that limb. This would justify a conclusion that causalgic pain may arise even though the nerve is conducting fully its motor and sensory functions. In other words causalgia may be produced by a nerve lesion which interferes with neither the somatic sensory fibres nor the motor fibres.

An analysis of the sensory findings associated with causalgia showed that in seven cases there was complete loss of pain and tactile sensation in the area to which the patient referred his

pain. This would suggest that complete damage to the somatic sensory fibres may not prevent the onset of causalgic pain. Support is lent to this view by the results of some of the methods of treatment adopted for the relief of pain in this condition. Sicard's (1916) method of injection of alcohol or Smithwick and White's (1935) modification of crushing the peripheral nerves above the ankle may be employed. Foot can be rendered insensitive in this way. Smithwick and White are frank enough to admit that these methods frequently failed to relieve the pain. They go on to add, 'Why interruption of all known sensory fibres should fail and be followed by pain in an insensitive area, is not known.'

Vascular damage.—In five cases the damage to the main blood vessel of the limb was associated. Ligature of the vessel of the limb preceded the onset of the pain by a few days. Three cases involved damage to axillary artery ; one to brachial and one to posterior tibial. If ischæmia is a cause of causalgia, as it is alleged, then it did not play an important part in this series of cases since there was evidence of damage to vessel only in a small number of cases.

Symptoms and Signs

Mitchell *et al.* (1864) first described causalgia in soldiers following penetrating wounds involving the nerves. They described it as hyperæsthesia of the hand or foot following an injury in the region of a peripheral nerve. The main features of this disorder may be grouped as follows : 1. Burning pain and disturbance of sensation. 2. Vasomotor changes. 3. Trophic disturbances.

1. *Pain and sensory disturbances.*—The time of onset of pain is very important ; it invariably follows the injury within a few days. There is no doubt that several conditions can give rise to burning pain of varying intensity which are not well understood. But in cases where injury to a nerve has resulted in a transient loss of conduction (neurapraxia) during the recovery stage the patients sometimes complain of burning pain very much like causalgia which is probably a part of protopathic pain of head. In all such cases the onset of pain is much later, the interval between the injury and occurrence of pain extending to several months.

The three classical characters of pain described by Mitchell *et al.* still constitute the best description of pain. The pain is burning, spontaneous and constant. It is liable to exacerbations on the slightest physical or emotional stimulus. The patient is never free of pain. His hand or foot is on 'fire'. The pain is always worse during the day but improves during the night. The patient is afraid of exposing his hand or foot to the sun. Very rarely, some of them (two in this series) preferred to wrap up the part. If the pain is severe, the patient derives great relief by soaking the affected part in water. The intensity of pain is variable from trivial burning sensation to a state of torture. In a very marked case the patient is afraid of exposing his limb to draught ; he resents noises close to him and is afraid even to talk or eat. In trying to engage some of the severe cases in talk, it was noticed that they insisted on soaking their hands in cold water before answering a question although the hand was already wrapped up in a piece of wet cloth.

In this series the pain was classified as trivial if it did not prevent the patient from engaging in normal activities in the hospital. In moderately severe group, the pain disturbed him considerably during the day preventing him from engaging in normal activities, but it did not disturb his sleep at night. In the severe group, the patient could not sleep at night. In the very severe group, the patient was very miserable, sleepless, unable to eat and with all the features of pain well marked. The numbers according to the classification were trivial 8, moderately severe 2, severe 8, and very severe 5.

Hyperæsthesia in the causalgic area was present only in nine cases, being marked in five. In all cases showing hyperæsthesia, the sensations in the area were intact. Conversely, cases with anæsthesia of the causalgic area did not, at any time, show hyperæsthesia.

2. *Vasomotor changes.*—Hyperidrosis. There was evidence of excessive sweating in nine cases (28 per cent). Four were very severe cases. Of the remaining 23 patients, in two the hands were excessively dry and rough although they were suffering from severe causalgic pain. Excessive sweating, therefore, is not a constant feature. This is what one would expect. Since hyperidrosis is due to irritation of the sudomotor fibres in the nerves, its incidence is bound to vary according to the degree of involvement of the sudomotor fibres and whether the nerve is completely divided or not. A lesion causing interruption of a large number of sudomotor fibres would obviously not cause excessive sweating but hypo- or anhydrosis.

Changes in circulation.—These changes, caused by involvement of the vasomotor fibres in a nerve, do not seem to bear a constant relationship to causalgia. Sometimes the affected limbs show signs of loss of vasomotor tone in the part distal to the lesion. The limb in such cases gets congested on hanging and blanches on elevation. More often there is no evidence of loss of vasomotor tone. The affected hand or foot tends to be paler and colder than its fellow and does not show any marked change on change of posture.

3. *Trophic disturbances.*—In cases of long standing the skin tends to atrophy. It is thin, dry and glossy, and the nails tend to get thickened and brittle. Only one patient of this series showed these changes. He had had

severe causalgia for five months before coming for treatment.

Differential Diagnosis

As already stated there is little doubt that pain of burning character can arise from causes other than causalgia. One example is the pain that occurs during the recovery stage of a nerve in which transient interruption of function has resulted from injury. In this series, before a pain was classified as causalgic, it had to be (*a*) in the region of a peripheral nerve subjected to damage, (*b*) spontaneous, (*c*) continuous, (*d*) liable to exacerbations, and (*e*) burning in character. The time of onset was considered very important. Causalgic pain follows injuries within seven to ten days or at the most a fortnight to three weeks. Any pain that took longer was looked upon with suspicion. This was done in order to exclude cases of protopathic pain. With this object in view all cases showing evidence of recovering function were not classified as causalgic. The cases where there was no evidence of sensory or motor interruption at the time of examination were assumed to have had transient sensory or motor changes which had been missed during the patient's journey down to us from the front line.

The line of treatment adopted depended upon the severity of the pain. Although Mitchell was of the opinion that causalgia was a self-limiting disease, our experience was different. If the pain was at all severe it resulted in profound changes in the mental condition of the patient. No attempt was made to allow severe causalgia to subside spontaneously, but trivial and moderately severe cases were allowed to subside by themselves. If the patient complained of pain interfering with his activities or sleep treatment was given. The first step always was to inject 1 per cent novocaine solution around the sympathetic trunk at the appropriate level—upper dorsal in arm cases and upper lumbar in leg cases. If the patient showed a good response and the pain was not severe, an attempt was made to see if the pain would subside on repeated injections. In some cases it did. Where the pain was severe the injection gave us a clue to the success or otherwise of surgical treatment. Where the response to injection was good and pain was relieved for sometime, treatment was undertaken with considerable assurance that the patient would benefit from it.

In this series the pain subsided spontaneously in four cases and yielded to repeated injections in four cases, while sympathectomy was done in 21 cases and periarterial sympathectomy in three.

Periarterial sympathectomy was undertaken as an experiment. Leriche in 1913 first reported relief of causalgic pain by this procedure. The present attempt was not an effort to confirm his findings but to enlarge upon them. The results were quite interesting. In two cases the posterior tibial artery behind the medial malleolus and in one case the radial artery just above the wrist were exposed under local anæsthesia taking good care that novocaine did not extend deeper than the deep fascia. In other words, an attempt was made to explore the perivascular sympathetics without interfering with their conduction. The arteries were denuded of the fibres surrounding them. The patient complained of the pain deep down in the foot or hand on interference with these fibres. The pain elicited was severe but vague and diffuse. It was in the foot or hand and deep down in the bones. Lastly, it was found that there was little or no pain on stimulating the fibres with rapid faradic current, but pain of the type described above was elicited on crushing the fibres. Apparently, dragging and crushing were the two effective stimuli with these nerve fibres. All the three cases were of moderately severe pain only and had no pain after perivascular sympathectomy. They have remained free of pain for four months and are still under observation. This would justify the conclusion that some of the afferent sympathetic fibres course to the destinations in skin and other peripheral *via* the vessels. These fibres are probably not really sympathetic but ordinary somatic fibres travelling with sympathetic fibres.

Lumbar sympathectomy was done in five cases. The approach was through an oblique incision in the flank and the trunk was exposed extraperitoneally. The second and third lumbar ganglia with the intervening trunk were removed. This was found quite effective in relieving the pain completely, although it is agreed that this does not completely denervate the entire leg. In a certain number of cases the fourth ganglion was also removed but this presented no advantage over the less complete operation. Dorsal sympathectomy was done in 16 cases, 14 being anterior operations (Leriche, 1913). In these cases the lower cervical and upper dorsal ganglia were exposed through an incision in the lower part of the neck. The clavicular head of the sternomastoid was cut and the scalenus anterior was divided near its insertion. The chain was then exposed by mobilizing the subclavian artery and pushing it down. The other two were posterior route (Adson) operations. One of these was performed by my colleague Major A. J. Slassor. My preference was for the anterior operation, because it was neater and easier and less damaging than the posterior, since it did not involve resection of ribs or cutting of big muscles. It was urged several times that an adequate exposure of the trunk was not possible through the anterior route. It was particularly pointed out that it was difficult to go down as far as the third dorsal ganglion through the anterior route. That was not my experience. In every case I found it quite easy to expose the trunk up to and just below the level

of the third dorsal ganglion. This was confirmed in left-side operations by going down as far as the arch of the aorta. The only time I had difficulty in exposing the trunk was in a case where the patient had a big bony mass around the middle of the clavicle as a result of previous fracture. In one case in which the posterior operation was adopted the choice was forced by the fact that the patient had had his subclavian artery exposed already by another surgeon in an attempt to control bleeding.

The operation in all cases consisted of division of the sympathetic trunk below the level of the third dorsal ganglion and resection of the second and third rami. The divided trunk was then turned up and stitched away from its normal situation to prevent regeneration (Telford). This did not result in a Horner's syndrome.

The individual results of the cases are given with the case summaries. In general the results in all cases were dramatic. As soon as the patient came round he was free of pain and remained free of pain subsequently. In one case only, a certain amount of hyperæsthesia persisted. The patient, however, maintained that he was much better after the operation in so far as the burning pain had disappeared. The hyperæsthesia persisted for about three months and then slowly disappeared. This was a very severe case of five months' duration. Twelve out of these 21 cases have been discharged. On an average, they remained under observation for three and a half months after operation, the shortest period being one month and the longest eight months. Nine cases are still under observation for a period varying from four to ten months after operation. All the cases are still free of pain.

Case Reports

1. *Case* 6.—Median causalgia. Machine-gun wound, right arm, on 26th April, 1944. Severe burning pain in right hand seven days after injury. Typical causalgic pain. Severe case. No paralysis. Sensation intact in causalgic areas. Hyperæsthesia present. Injection of stellate ganglion on 29th July, 1944. Relief of pain for three hours. Injection repeated on 1st August, 1944. Short-lived relief. Chain divided on 25th August, 1944. Complete relief. Discharged on 1st August, 1945.

2. *Case* 7.—Median causalgia. Gun-shot wound, left arm, on 24th July, 1944. Severe burning pain started five days after injury. No notes of initial sensory findings. On 13th October, 1944, pin-prick and touch sensations present in the causalgic area, but dull. No hyperæsthesia. No paralysis. Chain injected with novocaine on 23rd October, 1944. Pain relieved for short time. Injection repeated on 6th November, 1944. Typical Horner's syndrome. Relief of pain. Chain divided on 15th November, 1944. Complete relief. Discharged on 18th January, 1945.

3. *Case* 13.—Median causalgia. Multiple grenade wounds, left palm, on 27th May, 1944. *X*-ray revealed three metallic foreign bodies; one in the vicinity of median nerve in the palm at the base of thenar eminence. Very severe pain with marked hyperæsthesia. No loss of sensation. Paralysis of thenar muscles only. Duration five months. Chain injected on 15th October, 1944. Relief for a very short time. Chain divided on 18th October, 1944. Relief of burning pain after operation but hyperæsthesia persisted for three months. Discharged on 17th February, 1945.

4. *Case* 18.—Median causalgia. Grenade wound, right shoulder, on 12th June, 1944. Severe causalgic pain in palm of right hand. Started eight days after injury; very severe. Dulling of pin-prick and touch in the causalgic area. No hyperæsthesia. No initial sensory findings. Paralysis of all median muscles except pronator teres, flexor or carpi radicals, and palmaris longus. Injection on 18th August, 1944, with short relief. Operation on 20th August, 1944. Chain divided. Complete relief. Discharged on 14th April, 1945.

5. *Case* 21.—Median causalgia. Gun-shot wound, neck, left. *X*-ray shows fracture of lateral masses of 6th and 7th cervical vertebræ and small metallic foreign body. Had complete flaccid paralysis of left arm and paralysis with marked exaggeration of all reflexes of left leg. No sphincteric trouble. Typical causalgic pain eleven days after injury in palm of left hand. Injection of chain on 10th March, 1945, with short relief. Chain divided on 21st March, 1945. Neurolysis of roots of the brachial plexus through the same incision at the same time. Complete relief of pain and almost complete recovery of arm muscles. No anæsthesia on causalgic area. Discharged on 2nd November, 1945.

6. *Case* 33.—Tibial causalgia. Gun-shot wound, left thigh, on 30th March, 1945. Moderately severe. Causalgic pain in sole of left foot. Paralysis of all leg muscles except biceps semitend. and semimemb. Injection of lumbar chain on 14th May, 1945. Marked relief. Foot congested, dry and warmer than its fellow. Periarterial sympathectomy of the posterior tibial artery behind the medial malleolus on 20th May, 1945. Complete relief. Discharged on 24th September, 1945.

7. *Case* 24.—Tibial causalgia. Gun-shot wound, left thigh, on 30th March, 1945. Severe causalgic pain in the sole of left foot. Started seven days after injury. Paralysis of leg muscles the same as in the above case. Complete loss of sensation in sole of foot. Injection on 11th April, 1945. Short relief. Chain divided on 16th April, 1945. Complete relief. Under observation.

8. *Case* 31.—Tibial causalgia. Multiple gun-shot wound, left elbow and both buttocks, on 23rd November, 1944. Severe causalgic pain in sole of left foot started four days after injury. Paralysis of tibial muscles. Complete loss of sensation in sole of foot. Injection of lumbar chain on 3rd April, 1945. Chain divided on 11th April, 1945. Complete relief. Discharged on 7th August, 1945.

9. *Case* 30.—Tibial causalgia. Shell wound, left buttock, on 8th November, 1944. Moderately severe causalgic pain came on three days after injury. Exploration of the sciatic nerve on 5th March, 1945. Nerve found completely divided at the brim of the sacro-sciatic foramen. Suture not possible. Injection on 16th April, 1945. Short relief. Chain divided on 9th May, 1945. Complete relief. Discharged on 11th October, 1945.

10. *Case* 29.—Median causalgia. Shell wound, right forearm and right thigh. Severe burning pain of hand came on four days after injury. Paralysis of all ulnar muscles. Pain also in the sole of the right foot but not severe. Injection of dorsal chain on 21st March, 1945. Short relief. Chain divided on 26th March, 1945. Complete relief. Pain in sole of foot subsided by itself. In hospital.

11. *Case* 28.—Tibial causalgia. Gun-shot wound, left thigh, on 25th January, 1944. Moderately severe burning pain in sole of foot came on 14 days after injury. Can sleep at night. No loss of sensation. Paralysis of tibial and peroneal muscles. Exploration and suture of the peroneal nerve on 19th July, 1944. Tibial muscle had recovered spontaneously. Pain had completely subsided by 26th May, 1945.

12. *Case* 32.—Tibial causalgia. Shell wound, left thigh, on 3rd February, 1945. Moderately severe pain came on eight days after injury. Paralysis of peroneal muscles only. Loss of sensation in sole of the foot and

peroneal area. Posterior tibial artery denuded of its sympathetic fibres behind the medial malleolus on 20th May, 1945. Complete relief of pain. In hospital.

13. *Case* 11.—Median causalgia. Shell wound, right upper arm, on 7th June, 1944. Median causalgic pain noticed on 16th June, 1944. Brachial artery aneurysm noticed on 14th July, 1944. Aneurysm explored on 6th November, 1944. Arterial, saccular, median nerve found adherent to its wall. Carefully separated. Aneurysm excised. Pain subsided by itself gradually. Free of pain from 17th February, 1945.

14. *Case* 9.—Median causalgia. Gun-shot wound, left arm, on 21st May, 1944. No paralysis. No loss of sensation. Injection on 8th November, 1944. Complete relief of pain. Injection repeated on 8th January, 1945, because slight pain had recurred. Complete relief.

All the case summaries are not given to avoid repetition. The cases given above are sufficiently representative. Case 30 requires some comment. It shows that causalgic pain can occur with the nerve supplying the limb completely divided. That again would lead us to the conclusion that all the sympathetic fibres do not run with the main nerve of the limb. They either join it later or reach their destination *via* the blood vessels.

Another interesting point elicited during treatment of these cases was the result of experimental stimulation of the lumbar sympathetic trunk during operation. In three cases the lumbar sympathetic chain was exposed under local anæsthesia taking good care that the chain itself was not affected. On stimulation of the chain below the second lumbar ganglion with rapid faradic current little or no pain resulted, but compression or pinching of the nerve fibres produced marked pain which the patient vaguely referred to the leg on the same side. The pain could not be accurately localized, but all the patients maintained that it was felt deep down in the leg. In spite of carefully controlled laboratory experiments conducted on animals (Burget and Livingston, 1931), the above experiments would go to show that in human beings at least pain fibres of some sort travel through the sympathetic chain.

Another noticeable fact about these cases of causalgia was the constancy of the site of the pain. Irrespective of the level of the lesion in the median nerve, the pain always started in the lateral half of the palm of the hand. The level of the lesion in the nerve varied from the axilla to the palm of the hand, but the pain always started in the same area of the hand. Similarly, the lesion in the tibial nerve varied from the buttock to the leg, and yet the pain was always felt in the sole of the foot. One wonders if there is not a close similarity between the causalgic pain and the visceral pain with which we are so familiar. The character of the pain in the two, no doubt, varies but that can be explained by the difference in stimuli in the two cases. The focus of irritation in causalgia lies in the intraneural fibrosis in which the central fibres of the divided nerves are involved. Impulses from this focus of irritation set up changes in the spinal or thalamic centres. This would explain why causalgia was so commonly seen among Gurkhas and also why the cases of causalgia diminished to a negligible number after the general use of penicillin. This view of ætiology of causalgia is my own interpretation of facts always known. It is put merely as a suggestion.

Summary

1. Thirty-two cases of causalgia are described.

2. Their treatment by sympathectomy is discussed.

3. Case histories of some of the cases are included.

My thanks are due to Brigadier D. Denny-Brown, consulting neurologist, India, and Lieut.-Colonel R. A. Elliot, R.A.M.C., advisor in neurology, for valuable suggestions, and to General Gordon Willson, D.M.S., India, for permission to report the cases.

REFERENCES

BURGET, G. E., and LIVINGSTON, W. K. (1931). *Amer. J. Physiol.*, **97**, 249.

LERICHE, R. DE (1913). *Lyon Chir.*, **10**, 378.

MITCHELL, S. W., MOREHOUSE, G. R., and KEEN, W. W. (1864). *Gun-shot Wound and other Injuries of Nerve.* J. B. Lippincott Co., Philadelphia.

SICARD, J. A. (1916) .. *Presse Méd.*, **24**, 241.

SMITHWICK, R. H., and WHITE, J. C. (1935). *Surg. Gyn. and Obstet.*, **60**, 1106.

PSYCHIATRY IN BURMA AFTER RE-OCCUPATION

By R. M. LLOYD-STILL

LIEUTENANT-COLONEL, I.M.S.

I ARRIVED in Burma on the 10th September, 1945, and was appointed in Cas (B) as Officer Commanding, Mental Hospital. This at that time only existed on paper. A few old dements and criminal insanes who had survived the Japanese occupation were accommodated in the local jail. The old Mental Hospital which was a comparatively new building, and before the war accommodated over 1,000 patients, presented a grim picture due to our bombing. This hospital had been one of the best in the east with spacious grounds, extensive gardens, big dairy and every facility for occupational therapy, etc. It had been used by the Japanese as a wireless transmitting station and the Indian national army were kindly permitted to have a hospital there ! Many buildings had completely disappeared—the female section no longer existed and it was difficult to recognize the European wards or the hospital block.

The first problem was to find accommodation and a small jail which at one time was the Borstal Institute was decided upon and efforts were then made to get possession from the military authorities. Early in October the move was made and the place was tidied up. This