■ TRAUMA

Nerve injuries sustained during warfare

PART II: OUTCOMES

R. Birch,
W. G. P. Eardley,
A. Ramasamy,
K. Brown,
R. Shenoy,
P. Anand,
J. Clasper,
R. Dunn,
J. Etherington,
P. Misra,
M. P. M. Stewart

*From Defence
Medical
Rehabilitation
Centre, Epsom,
United Kingdom*

■ R. Birch, MChir, FRCS, Consultant-in-Charge
War Nerve Injury Clinic

■ J. Etherington, OBE, FRCP, Clinical Director
Defence Medical Rehabilitation Centre, Headley Court, Epsom, Surrey KT18 6JW, UK.

■ M. P. M. Stewart, CBE QHS, FRCS Glas, FRCS (T&O), Col L/RAMC, Lately Defence Medical Consultant Advisor in Trauma and Orthopaedics
James Cook Hospital, Middlesbrough, UK.

■ W. G. P. Eardley, MSc, DIPSEM, MRCSed, Specialist Registrar in Trauma and Orthopaedics

■ A. Ramasamy, MRCS, MFSEM, DMCC, Specialist Registrar in Trauma and Orthopaedics

■ K. Brown, MSc, MRCS, Specialist Registrar in Trauma and Orthopaedics

■ J. Clasper, DPhil, DM, FRCSed, Col L/RAMC, Defence Professor of Trauma and Orthopaedics
Royal Centre for Defence Medicine, Academic Department of Military Surgery and Trauma, Birmingham Research Park, Vincent Drive, Birmingham B15 2SQ, UK.

■ R. Shenoy, MD(Res), MRCS, Clinical Research Fellow

■ P. Anand, MA, MD, FRCP, Professor in Neurology

■ P. Misra, MD, FRCP, Consultant Neurophysiologist
Peripheral Neuropathy Unit, Imperial College London, Hammersmith Hospital, Du Cane Road, London W12 0NN, UK.

■ R. Dunn, MBBS, FRCS(Plas), Consultant Plastic Reconstructive and Hand Surgeon
Odstock Centre for Burns, Plastic and Maxillofacial Surgery, Salisbury District Hospital, Salisbury, Wiltshire SP2 0BJ, UK.

Correspondence should be sent to Professor R. Birch; e-mail: m.taggart@hotmail.co.uk

©2012 British Editorial Society of Bone and Joint Surgery
doi:10.1302/0301-620X.94B4.28488 \$2.00

J Bone Joint Surg Br
2012;94-B:529-35.
Received 19 October 2011; Accepted after revision 1 December 2011

The outcomes of 261 nerve injuries in 100 patients were graded good in 173 cases (66%), fair in 70 (26.8%) and poor in 18 (6.9%) at the final review (median 28.4 months (1.3 to 64.2)). The initial grades for the 42 sutures and graft were 11 good, 14 fair and 17 poor. After subsequent revision repairs in seven, neurolyses in 11 and free vascularised fasciocutaneous flaps in 11, the final grades were 15 good, 18 fair and nine poor. Pain was relieved in 30 of 36 patients by nerve repair, revision of repair or neurolysis, and flaps when indicated. The difference in outcome between penetrating missile wounds and those caused by explosions was not statistically significant; in the latter group the onset of recovery from focal conduction block was delayed (mean 4.7 months (2.5 to 10.2) vs 3.8 months (0.6 to 6); $p = 0.0001$). A total of 42 patients (47 lower limbs) presented with an insensate foot. By final review (mean 27.4 months (20 to 36)) plantar sensation was good in 26 limbs (55%), fair in 16 (34%) and poor in five (11%). Nine patients returned to full military duties, 18 to restricted duties, 30 to sedentary work, and 43 were discharged from military service. Effective rehabilitation must be early, integrated and vigorous. The responsible surgeons must be firmly embedded in the process, at times exerting leadership.

The principles governing the surgical treatment of nerve injuries sustained during warfare were established during the First World War.^{1,2} They include resection of scar until a healthy bed is secured, excision of damaged nerve until healthy stumps are reached, and tension-free suture by adequate mobilisation and flexion of adjacent joints, or by grafting. Of grafting, Tinel² wrote: 'when the distance between the segments of the nerve trunk is too great to permit direct suture the only legitimate operation is nerve grafting'. The treatment of war wounds was described by Robert Jones in the same war³: the wound should be treated by debridement, excision and delayed closure.⁴ Jones oversaw the institution of hospitals dedicated to rehabilitation,⁵ where the principles of reconstruction were defined.⁶ Further rehabilitation units were developed during The Second World War. The work of the five special hospitals designated for the treatment of nerve injuries was analysed in the Medical Research Council Report No. 282.⁷ Results of repairs were graded by strict, even stringent, criteria. About 50% of all cases treated by direct suture made an effective recovery. Seddon⁸ reported a similar rate of recovery in 67 cases where grafts were used. Woodhall and Beebe⁹ brought together the experience of the military surgeons of the United States. In a review of

3148 nerve sutures in war wounds they reported that 'delay in suture involves a loss of, on average, about 1% of maximal performance for every six days of delay'.⁹ From their experience of the war in Vietnam, Omer et al¹⁰⁻¹² found that the outcome after grafting was disappointing and concluded that pedicled nerve grafts gave better results.

A review of papers describing 13 000 nerve injuries resulting from conflict suggested that the first operation to the wound is most important and that arterial injuries must be repaired and sepsis prevented.¹³ Unfavourable factors include delay before repair, extensive damage to the nerve, adjacent soft tissues and to the skeleton, the presence of arterial injury and the level of the lesion. The mortality of those injured in war has decreased and the severity of the problems facing survivors has consequently increased.^{14,15}

Patients and Methods

The grading of outcome for individual nerves is set out in Tables I to IV.^{13,16,17} There were 261 nerve injuries in 100 patients (98 men and two women). Their mean age at presentation was 26.5 years (18.1 to 42.6). Injured nerves included 134 nerves of the upper limb and 127 nerves of the lower limb. A total of 213 nerve lesions (82%) in 90 patients

Table I. Grading of outcome in lesions of the radial nerve, based on Shergill et al¹⁷ (MRC, Medical Research Council)

	For high lesions above nerves to triceps	For intermediate lesions, between medial head of triceps and brachioradialis	For low lesions, the posterior interosseous nerve
Good	Elbow extension $\geq$ MRC grade 4 Wrist extension $\geq$ MRC grade 3	Wrist extension $\geq$ MRC grade 4 Finger and thumb extension $\geq$ MRC grade 3	Full independent extension of the digits $\geq$ MRC grade 4
Fair	Elbow extension $\geq$ MRC grade 3 Wrist extension MRC grade 2	Wrist extension $\geq$ MRC grade 3 Finger and thumb extension $\geq$ MRC grade 2	Extension of digits $\geq$ MRC Grade 3
Poor	Less than above	Less than above	Less than above

Table II. Grading of results in median and ulnar nerves repaired in the infraclavicular region, the axilla and the arm (high lesions) (MRC, Medical Research Council)

Description		
Grade	Median	Ulnar*
Good	Long flexor muscles MRC $\geq$ 4 Localisation to digit, without hypersensitivity Return of sweating	FCU and FDP little and ring MRC $\geq$ 4 Intrinsic muscles MRC $\geq$ 2 Localisation to little and ring fingers No hypersensitivity Return of sweating
Fair	Long flexor muscles MRC $\geq$ 3 Protective sensation, moderate or no hypersensitivity Sweating diminished or absent	FCU and FDP little and ring fingers MRC $\geq$ 3 No intrinsic muscle function Protective sensation little and ring fingers No, or moderate hypersensitivity Little or no sweating
Poor	Long flexor muscles MRC $\leq$ 2 Protective sensation but severe hypersensitivity/no sensation	FCU and FDP little and ring fingers MRC 2 No intrinsic muscle function Protective sensation with severe hypersensitivity or no sensation No sweating

* FCU, flexor carpi ulnaris; FDP, flexor digitorum profundus

occurred in open wounds. Two or more main nerves were damaged within the same zone of injury in 70 patients. The ulnar nerve was the most commonly injured at the elbow, while the tibial and common peroneal nerves were particularly vulnerable in the buttock and around the knee. For the purpose of this study the outcome was graded into four categories: 1, fully fit for military duty; 2, restricted duties; 3, sedentary duties; and 4, unfit for military service.

Statistical analysis. Statistical analysis was performed using SPSS version 18.0 (SPSS Inc., Chicago, Illinois). For categorical data chi-squared tests were used, with Fisher's exact value calculated where frequencies were < 5 . For continuous non-parametric data the Mann-Whitney test was used. A p-value < 0.05 was considered statistically significant.

Median values have been used for duration of follow-up as early recovery in some patients led to an early discharge from follow-up, skewing the mean data.

Results

Nerve injuries. Of the 261 nerve injuries, 173 (66.3%) were graded good, 70 (26.8%) fair and 18 (6.9%) poor at a median follow-up of 28.4 months (1.3 to 64.2). The 18 poor results comprised seven common peroneal nerve injuries, four tibial nerve, four ulnar nerve, two median and one radial nerve injury (Table V). Factors contributing to a poor outcome included destruction of the target tissue, failure of regeneration and persistent severe pain.

Nerve operations. Of the 261 injured nerves, 46 were repaired, 35 at a mean of 8 days (1 to 14) after injury and 11 at a mean of 90 days (35 to 184). There were ten sutures, all of which were primary. A total of 25 primary grafts were performed at a mean of eight days (1 to 17), and seven (delayed) at a mean of 90 days (35 to 184). There were also three muscular neurotisations and one intercostal nerve transfer, resulting in a total of 46 nerve repairs.

The initial grades for the ten sutures were six good, two fair and two poor. The final grades were seven good, two

Table III. Assessment of recovery of the common peroneal nerve (MRC, Medical Research Council)

Grade	Motor recovery	Sensory recovery
Good	Dorsiflexion and version MRC ≥ 4	No spontaneous pain; no hypersensitivity
Fair	Dorsiflexion MRC 3 to 4 OR Eversion MRC 3 to 4 OR Dorsiflexion and eversion MRC 2 to 3	No spontaneous pain No, or only mild, hypersensitivity
Poor	Less than the above	Spontaneous pain or significant sensitivity

Table IV. Grading of outcome for the tibial nerve: all levels (MRC, Medical Research Council)

Grade	Motor	Sensory
Good	Heel flexors MRC ≥ 4 , tibialis posterior, flexor digitorum longus, and flexor hallucis longus MRC 3 Return of sweating No fixed deformity, no trophic disturbance Normal shoes	Protective sensation with no more than mild hypersensitivity; no spontaneous pain
Fair	Heel flexors MRC ≥ 3 No useful recovery in long flexor muscles Incomplete return of sweating No serious trophic disturbance, no fixed deformity Normal shoes with or without foot drop splint for common peroneal palsy	Protective sensation, hypersensitivity not interfering with daily activities
Poor	No useful motor recovery; and /or significant fixed deformity; and/or trophic ulceration	No return of sensation; and/or significant hypersensitivity interfering with daily activities

fair and one poor, after two revision repairs and one neurolysis with a free vascularised fasciocutaneous flap.

The initial grades for the 32 grafts were five good, 12 fair and 15 poor. The final results were eight good, 16 fair and eight poor after five revision repairs and ten neurolyses. A free vascularised fasciocutaneous flap was used in ten of these 15 patients with revision surgery.

The results after two of three muscular neurotisations were fair; the third failed, as did the only intercostal transfer. The overall results of all 46 repairs were good in 15 (32.6%), fair in 20 (43.5%), and poor in 11 (23.9%) (Figs 1 and 2).

Persisting severe neuropathic pain was the most common indication for revision operation. Relief of pain was the main aim in six revision repairs, 11 neurolyses of previously repaired nerves and 19 neurolyses of injured but not previously repaired nerves.

Quantitative sensory testing. A total of 17 patients (49 nerves) were studied at Hammersmith Hospital, London, United Kingdom, using quantitative sensory testing, which included the assessment of thermal and monofilament perception thresholds. Sensory abnormalities over the cutaneous territories supplied by the nerves included touch (monofilament) allodynia in seven nerves, elevated

monofilament thresholds in 33, pinprick hyperalgesia in six, and reduced pinprick sensation in 23. Thermal threshold testing revealed elevated warm threshold in 38 nerves, elevated cool threshold in 34 and cool allodynia over the territory of the injured nerve in five.

Pain. A total of 36 patients experienced persisting and severe neuropathic pain and revision operations were undertaken in these. Of these, 30 patients experienced such relief post-operatively that analgesic medication was considerably reduced or abandoned. The operations included six revision repairs, 11 neurolyses of repaired nerves, and neurolysis of lesioned but not previously repaired nerves in 19 patients. The causes of persisting pain included displaced bone fragments, heterotopic bone, retained fragments of suture material, and most commonly, scar tissue that enveloped and constricted the nerve. A fasciocutaneous flap was used in 15 patients, in 11 to enhance nerve regeneration and in four for a painful scar. No case of false aneurysm or arteriovenous fistula was encountered.

Causalgia was relieved in two patients by repair or decompression of the nerves and repair of the main artery. Pain persisted in the third until an infected nonunion of the clavicle and indolent healing of the skin were corrected. Spontaneous resolution can occur,¹⁸ and did so in seven

Table V. Results by nerve

Nerve affected (n)	Good	Fair	Poor
Upper limb			
C5 (n = 6)	5	1	
C6 (n = 7)	4	3	
C7 (n = 7)	7		
C8 (n = 6)	5	1	
T1 (n = 4)	3	1	
Nerve to serratus anterior (n = 1)	1		
Suprascapular (n = 6)	6		
Circumflex (n = 6)	4	2	
Musculocutaneous (n = 3)	3		
Radial (n = 24)	22	1	1
Median (n = 29)	17	10	2
Ulnar (n = 35)	24	7	4
Lower limb			
Femoral (n = 5)	5		
Superior gluteal (n = 7)	6	1	
Inferior gluteal (n = 7)	6	1	
Common peroneal (n = 46)	26	13	7
Tibial (n = 47)	26	17	4
L4 (n = 2)		2	
L5 (n = 2)		2	
S1 (n = 2)	1	2	
S2 (n = 2)	1	2	
S3 (n = 2)	1	2	
S4 (n = 2)		2	

patients after wound treatment at the field and receiving hospitals.

Neurostenalgia was relieved by removal of the cause in nine of ten patients, in five cases following a fasciocutaneous flap. Pain persisted in the remaining patient, who came to below-knee amputation.

Of the 23 patients with post-traumatic neuralgia, 18 were improved by repair of the nerve and by improving its bed. One patient came to below-knee amputation and one required spinal cord stimulation. Pain persisted in three patients, who continue to take analgesic drugs and remain under review.

Conduction block/neurapraxia. Among the 261 lesions, 116 nerves (in 49 patients) had prolonged conduction block (PCB)/neurapraxia. The mean time to onset of recovery was 4.17 months (0.6 to 10.2). A total of 90 of these 116 nerves (78%) with PCB showed signs of recovery within six months of injury. Penetrating missile wounds accounted for 45 of these nerve injuries, explosion in the other 71. The mean time to recovery in the former was 3.8 months (0.6 to 6) compared with 4.7 months (2.5 to 10.2) in the latter (Mann-Whitney test, $p = 0.0001$).

Return of plantar sensation. In 42 patients (47 lower limbs, 18%) the initial presentation was with an insensate foot. At a mean of 27.4 months (20 to 36), recovery of sensation was graded good in 26 limbs (55.3%), fair in 16 (34.1%), and poor in five (10.6%). A total of 18 of these lesions were PCB, 16 axonotmesis and 13 neurotmesis, and nine of these

Fig. 1

Photograph of a 21-year-old patient at six weeks after injury. The patient had bilateral lesions of the median nerve from penetrating missile wounds. The right brachial artery was repaired as an emergency. The right median nerve was repaired within 48 hours. Recovery was good. The injury to the left median nerve was accompanied by destruction of the flexor muscles and fractures. Recovery was poor. Markings on the patient in darker ink represent complete sensory loss and lighter ink represents incomplete sensory loss.

nerves underwent repair. The poor results were seen in four nerves which could not be repaired, and one repaired nerve. **Return to duties.** Nine patients returned to full military service, 18 to restricted duties, 30 to sedentary work, and 43 were graded unfit for military service.

Case report 1: late onset of neurostenalgia and deepening nerve lesion; relief following a fasciocutaneous flap. A 30-year-old serviceman was injured in a helicopter crash, sustaining fractures to the second to eighth right ribs and right clavicle, a brain injury, and full-thickness burns to the right arm and chest (8% of total body surface area). Emergency treatment included fasciotomy of the right forearm. Wound cover was achieved by split skin grafting. After eight months he complained of increasingly severe pain about the elbow and deterioration of sensation and power in the hand. Function in the ulnar nerve was poor. There was blunting of median nerve sensation. A further operation was performed 14 months after initial injury. The median and ulnar nerves were tethered by scar and the epineurial vessels of the ulnar nerve obliterated. These filled before a fasciocutaneous flap based on the patent brachial artery was undertaken (Fig. 3). There was considerable improvement in pain and sensation post-operatively, and seven months later median and ulnar nerve function was normal. **Case report 2: prolonged conduction block and axonotmesis of the left brachial plexus; delayed onset of post-traumatic neuralgia of the right radial nerve.** An 18-year-old rifleman sustained massive multiple injuries from an improvised explosive device. These included bilateral high transfemoral amputations, amputation through the left forearm, extensive facial wounds with fracture of the mandible, and bilateral lung contusions. There was a wound in the right axilla. Emergency treatment included tracheotomy,

Fig. 2

Photograph of a 28-year-old patient at ten weeks after injury. There was a penetrating missile wound to right forearm, which destroyed the flexor muscles and damaged both median and ulnar nerves. The median nerve was grafted and a lateral thigh flap was performed after five days. Both nerves recovered. There was no pain at any time. There was a minor wound in the left forearm. Markings on the patient in darker ink represent complete sensory loss and lighter ink represents incomplete sensory loss.

laparotomy, and debridement of the wounds to the limbs, which were closed with split skin grafts. There was a complete and painful left-sided supraclavicular brachial plexus palsy and an incomplete and painful lesion of the right radial nerve. After nine weeks there was recovery into C5, which coincided with relief of pain, and he was able to abduct his shoulder. Tinel's signs for the radial, median and ulnar nerves were detectable in his arm. Reinnervation of infraspinatus, teres minor and posterior deltoid was confirmed by electromyography (EMG) at 4.75 months. Biceps and triceps remained denervated. Planned exploration of the brachial plexus was delayed because he required a laparotomy for bowel obstruction. Electromyography at 10.75 months confirmed reinnervation of triceps and the residual flexor muscles of the forearm. At one year biceps was recovering. By this point the right radial nerve had recovered, but there was constant severe pain in the distribution of the cutaneous nerves of the arm and forearm. He could not use his sole remaining limb. Conduction in the superficial radial nerve was diminished. Quantitative sensory testing (QST) demonstrated elevated thresholds to pinprick and light touch in the territory of the superficial radial

Fig. 3

Photograph of the patient in the first case report with neurostenalgia. Pain was relieved after excision of scar and insertion of a fasciocutaneous flap.

nerve: the thermal thresholds were normal. At 14 months the axillary scar was replaced by an ipsilateral pedicled thoracodorsal artery perforator fasciocutaneous flap. The cutaneous nerves were embedded in scar (Fig. 4).

The lesion of the left brachial plexus was a mixture of conduction block and axonotmesis. Pain improved as the nerve recovered, which enabled the subsequent use of a myoelectric prosthesis. Thus, the worsening post-traumatic neuralgia in the right upper limb arose from lesions to the cutaneous nerves in the axilla in which the thinly myelinated afferent fibres were affected. It was abolished by excision of the scar and resurfacing.

Discussion

The technique of tagging divided stumps to the adjacent tissue plane facilitated subsequent exploration and minimised retraction of stumps. In cases with extensive skin loss it seems preferable to defer the nerve repair until the patient is fit enough to endure another prolonged operation. Nerve grafts must obtain their blood supply from the bed in which they lie.¹⁹

Plantar sensation. The severely injured lower limb often imposes a rapid decision between salvage and amputation: it is often difficult to decide whether the limb is viable. The absence of plantar sensation has been an important variable in the decision-making process.²⁰ Our findings confirm those from the Lower Extremity Assessment Project.²¹ Plantar sensory loss is not a sound indication for amputation.

Depth of lesion and quantitative sensory testing. QST proved useful in detecting preservation of some types of nerve fibre, which showed that the nerve had not been wholly divided; in detecting recovery into some nerve fibres; in demonstrating the differential susceptibility of different nerve fibres in prolonged conduction block; and by revealing the behaviour of nociceptor fibres. The characteristic features of conduction block caused by a penetrating missile

Fig. 4a

Fig. 4b

Photographs of the patient in the second case report, showing a) post-traumatic neuralgia caused by axillary scar, and b) after a pedicled fasciocutaneous flap was undertaken, resulting in relief of pain.

include paralysis which exceeds loss of sensation; the nerves responsible for proprioception are more profoundly affected than those conveying the sensation of light touch; vasomotor and sudomotor function are least affected.²² Recovery is usually well advanced by the sixth day, but with more severe pressure or distortion there is local demyelination and more conduction block.^{23,24} The structural effects of focal compression are not caused by ischaemia²⁵: this lesion was more common in cases of prolonged conduction block. It is more difficult to account for the conduction block following blast injuries in which the patient is exposed, at close range, to the shock wave of an explosion without any fracture and with no signs of significant injury to the soft tissues. The underlying mechanism is under investigation.

The slowly developing conduction block in patients with neurostenalgia is probably partly due to ischaemia, because relief of pain and recovery of cutaneous sensibility are rapid. **Neuropathic pain.** The events that underlie neuropathic pain after nerve injury include: 1) spontaneous firing in nociceptor and other neurons, and abnormally increased firing in response to noxious stimulation²⁶⁻²⁸; 2) sensitisation of the axons, their cell bodies in the dorsal root ganglion and the dorsal horn of the spinal cord, so that pain and sensory disturbance are increasingly experienced in new areas outside the distribution of the injured nerve²⁹⁻³¹; 3) sensitisation of mechanoreceptive neurons or a change in their behaviour so that non-painful stimuli are perceived as pain^{32,33}; and 4) afferent and efferent fibres in the sympathetic system, which are conveyed with the nerve trunks, are involved in the injury, and may to some extent provoke or maintain symptoms and signs arising from abnormalities at the level of lesion or more centrally.³⁴

The spread of pain beyond the lesion of the injured nerve is a common example of central sensitisation. Increased somatic C-fibre and A δ -fibre input in the spinal cord³⁵ may account for the enhanced biceps reflex seen in one patient

with neurostenalgia from a median nerve injury. This, together with his pain, was abolished by revision repair and a fasciocutaneous flap. The convergence of visceral and somatic afferents into lamina V of the spinal cord³⁶ may explain the worsening of pain with filling of the bladder and bowel experienced by one patient with a high sciatic injury who eventually needed spinal cord stimulation. This phenomenon has been seen in other patients since the completion of this study, and is under investigation.

Important principles in the treatment of neuropathic pain include³⁷: 1) removal of the cause; 2) promotion of healing or regeneration; 3) correction of the micro-environment of the nerve; 4) restoration of afferent pathways; 5) modulation of the central inhibitory pathways; 6) reduction of sympathetic overactivity; and 7) changing pain thresholds by modification of emotional or behavioural components of pain interpretation.

Surgery is required for the first four of these, and it may be necessary for the reduction of sympathetic overactivity. It is particularly effective for neurostenalgia, which may be relieved by surgery years after onset,³⁸ and in causalgia. Sympathectomy can be effective for patients with causalgia^{39,40} but was not carried out in these patients.⁴¹ In general, the pharmacological treatment of nerve pain is disappointing: about 60% of patients do not experience even moderate improvement.⁴²⁻⁴⁴ In these military patients drugs were given in appropriately high doses, with close monitoring of their effects.

The successful treatment of pain in these patients requires close collaboration between the treating surgeons and physicians with a particular interest in pain management. It is important to remember that the persistence of neuropathic pain after focal injury to a nerve indicates that the agent responsible for that injury is still active.

Rehabilitation. Rehabilitation of the war-wounded starts at the time of injury, where early assessment is undertaken by

deployed medical rehabilitation teams based at the nearest field hospital or even further forward, so that a plan for rehabilitation is in place by the time the injured service men and women return to their base hospital.⁴⁵ The level of outcome is expected to be higher than in civilian practice. The whole patient must be considered: treatment cannot be compartmentalised into one area of therapy alone, and the psychological effects of near-death, of losing friends, of disfigurement, often heightened by 'mild' brain injury, have a major bearing on the patient's ability to learn new activities or return to work. Patients are admitted to Headley Court once their general condition is stable and they are free from serious infection. Uncertainty about the future of their limbs, which are painful and weak or even paralysed, is extremely demoralising for these patients. The War Nerve Injuries Clinic serves to provide a definitive prognosis for recovery and to implement speedily a coordinated and integrated plan of treatment to relieve pain and improve prognosis. That only nine of our patients were deemed fit enough to return to full duties is a reflection of the severity of their injuries.

The authors wish to thank Dr C. Cordivari (Queens Square) for ensuring the rapid investigations of patients by electrodiagnostic methods. The patients illustrated in Figures 1 and 2 were treated by Mr D. Power, FRCS and Mrs J. Webb, FRCS at the Queen Elizabeth Hospital, Birmingham. The figures are reproduced from *Surgical disorders of the peripheral nerves*, 2nd edition, 2011, by kind permission of Springer UK.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References

1. Delorme E. The treatment of gunshot wounds of the nerves. *Br Med J* 1915;1:853–855.
2. Tinel J. *Nerve wounds*. London: Baillière, Tindall and Cox, 1917:297–300.
3. Jones R. *Orthopaedic surgery of injuries*. London: Frowde, 1921:145–226.
4. Hull RB, Rob CG. Amputation. In: Ryan JM, Rich NM, Dale R, Morgans BT, Cooper GJ, eds. *Ballistic trauma: clinical relevance in peace and war*. London: Arnold, 1997:149–159.
5. Goldthwait JE. The place of orthopedic surgery in war. *J Bone Joint Surg* 1917;15:679–686.
6. McMurray TP. Discussion of the indications, technique and results of transplantation in gunshot injuries of nerves. *J Bone Joint Surg* 1919;14:125–135.
7. Seddon HJ. Peripheral nerve injuries. In: Seddon HJ, ed. *Medical research council special report, series 282*. London: H.M.S.O., 1954:451.
8. Seddon HJ. Nerve grafting. In: Seddon HJ, ed. *Peripheral nerve injuries: medical research council special report, series 282*. London: H.M.S.O., 1954:402–403.
9. Woodhall B, Beebe GW. *Peripheral nerve regeneration: a follow-up study of 3,656 World War II injuries*. VA Medical Monograph. Washington: U. S. Government Printing Office, 1956.
10. Omer GE Jr. Injuries to nerves of the upper extremity. *J Bone Joint Surg [Am]* 1974;56-A:1615–1624.
11. Omer GE, Eversmann WW. Peripheral nerve problems. In: Burkhalter WE, Ballard A, eds. *Surgery in Vietnam: orthopedic surgery*. Washington DC: Office of the Surgeon General and Center of Military History, U.S. Army, 1994:155–188.
12. Omer GE, Spinner M, Van Beek A. *Management of peripheral nerve problems*. Second ed. Philadelphia: Saunders, 1998:398–405.
13. Birch R. Compound nerve injuries. In: Birch R, ed. *Surgical disorders of the peripheral nerves*. Second ed. London: Springer, 2011:303–374.
14. Champion HR, Holcomb JB, Lawnick MM, et al. Improved characterization of combat injury. *J Trauma* 2010;68:1139–1150.
15. Hofmeister EP, Mazurek M, Ingari J. Injuries sustained to the upper extremity due to modern warfare and the evolution of care. *J Hand Surg [Am]* 2007;32:1141–1147.
16. Birch R, Raji AR. Repair of median and ulnar nerves: primary suture is best. *J Bone Joint Surg [Br]* 1991;73-B:154–157.
17. Shergill G, Bonney G, Munshi P, Birch R. The radial and posterior interosseous nerves: results of 260 repairs. *J Bone Joint Surg [Br]* 2001;83-B:646–649.
18. Barnes R. Causalgia: a review of 48 cases. *Spec Rep Ser Med Res Council (G B)* 1954;282:156–185.
19. Seddon HJ. The use of autogenous grafts for the repair of large gaps in peripheral nerves. *Br J Surg* 1947;35:151–167.
20. Bosse MJ, MacKenzie EJ, Kellam JF, et al. A prospective evaluation of the clinical utility of the lower-extremity injury-severity scores. *J Bone Joint Surg [Am]* 2001;83-A:3–14.
21. Bosse MJ, McCarthy ML, Jones AL, et al. The insensate foot following severe lower extremity trauma: an indication for amputation? *J Bone Joint Surg [Am]* 2005;87-A:2601–2608.
22. Seddon HJ. Three types of nerve injury. *Brain* 1943;66:237.
23. Fowler TJ, Danta G, Gilliatt RW. Recovery of nerve conduction after a pneumatic tourniquet: observations on the hind-limb of the baboon. *J Neurol Neurosurg Psychiatry* 1972;35:638–647.
24. Ochoa J, Fowler TJ, Gilliatt RW. Anatomical changes in peripheral nerves compressed by a pneumatic tourniquet. *J Anat* 1972;113:433–455.
25. Dyck PJ, Dyck J, Engelstad PJB. Pathological alteration of nerves. In: Dyck PJ, Thomas PK, eds. *Peripheral neuropathy*. Fourth ed. Philadelphia: London: Elsevier Saunders, 2005:745–753.
26. Michaelis M, Vogel C, Blenk KH, Arnarson A, Jänig W. Inflammatory mediators sensitize acutely axotomized nerve fibers to mechanical stimulation in the rat. *J Neurosci* 1998;18:7581–7587.
27. Wall PD, Gutnick M. Ongoing activity in peripheral nerves: the physiology and pharmacology of impulses originating from a neuroma. *Exp Neurol* 1974;43:580–593.
28. Wall PD, Gutnick M. Properties of afferent nerve impulses originating from a neuroma. *Nature* 1974;248:740–743.
29. Wall PD, Devor M. The effect of peripheral nerve injury on dorsal root potentials and on transmission of afferent signals into the spinal cord. *Brain Res* 1981;209:95–111.
30. Wall PD, Lidierth M, Hillman P. Brief and prolonged effects of Lissauer tract stimulation on dorsal horn cells. *Pain* 1999;83:579–589.
31. Woolf CJ, Shortland P, Coggeshall RE. Peripheral nerve injury triggers central sprouting of myelinated afferents. *Nature* 1992;355:75–78.
32. Coward K, Plumpton C, Facer P, et al. Immunolocalization of SNS/PN3 and NaV/SNS2 sodium channels in human pain states. *Pain* 2000;85:41–50.
33. Michaelis M, Liu X, Jänig W. Axotomized and intact muscle afferents but no skin afferents develop ongoing discharges of dorsal root ganglion origin after peripheral nerve lesion. *J Neurosci* 2000;20:2742–2748.
34. Ramer MS, Thompson SW, McMahon SB. Causes and consequences of sympathetic basket formation in dorsal root ganglia. *Pain* 1999;83(Suppl):111–120.
35. Woolf CJ, Wall PD. Relative effectiveness of C primary afferent fibers of different origins in evoking a prolonged facilitation of the flexor reflex in the rat. *J Neurosci* 1986;6:1433–1442.
36. Pomeranz B, Wall PD, Weber WV. Cord cells responding to fine myelinated afferents from viscera, muscle and skin. *J Physiol* 1968;199:511–532.
37. Willner C, Low PA. Pharmacologic approaches to neuropathic pain. In: Dyck PJ, Thomas PK, eds. *Peripheral neuropathy*. Vol. 2, Third ed. Philadelphia: WB Saunders, 1993:1709–1720.
38. Camp SJ, Milani R, Sinisi M. Intractable neurostenalgia of the ulnar nerve abolished by neurolysis 18 years after injury. *J Hand Surg Eur Vol* 2008;33:45–46.
39. Kline DG, Hudson AR. *Nerve injuries: operative results for major nerve injuries, entrapments, and tumors*. Philadelphia: WB Saunders, 1995.
40. Jebara VA, Saade B. Causalgia: a wartime experience: report of twenty treated cases. *J Trauma* 1987;27:519–524.
41. Birch R. Pain. In: *Surgical disorders of the peripheral nerves*. Second ed. London: Springer, 2011:527–561.
42. O'Connor AB, Dworkin RH. Treatment of neuropathic pain: an overview of recent guidelines. *Am J Med* 2009;122(Suppl):22–32.
43. Anand P, Birch R. Pain following brachial plexus injury and its management. In: Venkataswami RM, ed. *Surgery of the injured hand: towards functional restoration*. New Delhi: Jaypee Brothers, 2009:359–363.
44. Rowbotham MC. Mechanisms and management of neuropathic pain. In: Dyck PJ, Thomas PK, eds. *Peripheral neuropathy*. Fourth ed. Philadelphia: Elsevier Saunders, 2005:2637–2652.
45. Etherington J. Rehabilitation in the war wounded. In: Mahoney PF, Brooks AJ, Ryan JM, et al, eds. *Ballistic trauma: a practical guide*. Third ed. London: Springer, 2011.