

Surgical treatment of adult traumatic brachial plexus injuries

An overview

Mario G. Siqueira¹, Roberto S. Martins^{1,2}

ABSTRACT

Traumatic injuries to the brachial plexus in adults are severely debilitating. They generally affect young individuals. A thorough understanding of the anatomy, clinical evaluation, imaging and electrodiagnostic assessments, treatment options and proper timing of surgical interventions will enable nerve surgeons to offer optimal care to patients. Advances in microsurgical technique have improved the outcome for many of these patients. The treatment options offer patients with brachial plexus injuries the possibility of achieving elbow flexion, shoulder stability with limited abduction and the hope of limited but potentially useful hand function.

Key words: brachial plexus injury, surgical treatment, nerve repair.

Tratamento cirúrgico das lesões traumáticas do plexo braquial em adultos: uma visão geral

RESUMO

As lesões traumáticas do plexo braquial em adultos são severamente debilitantes e, em geral, afetam indivíduos jovens. Uma ampla compreensão da anatomia, da avaliação clínica, dos estudos eletrodiagnósticos e por imagem, das opções de tratamento e do momento apropriado para o tratamento cirúrgico irá permitir que o cirurgião de nervos ofereça o tratamento ideal ao paciente. Os avanços na técnica microcirúrgica melhoraram os resultados para muitos desses pacientes. As opções de tratamento oferecem aos pacientes com lesões do plexo braquial a possibilidade de obter flexão do cotovelo, estabilidade do ombro com abdução limitada e a esperança de função limitada mas potencialmente útil da mão.

Palavras-chave: lesão do plexo braquial, tratamento cirúrgico, reparo de nervos.

Traumatic brachial plexus lesions in adults are devastating injuries. The typical patient is a young man who was injured in a motorcycle accident. These lesions are of great social significance since most of the patients have to face severe residual morbidity after their injuries. The exact number of lesions occurring each year is difficult to ascertain, but it is certainly growing, in parallel with the increasing number of high-speed motor vehicle accidents, especially involving motorcycles.

Based on experience with 1,068 patients with brachial plexus injuries, Narakas¹ developed a rule of “seven sev-

enties” that gives an approximate idea of the statistics involved in brachial plexus lesions: approximately 70% of traumatic brachial plexus injuries are secondary to motor vehicle accidents; of these, approximately 70% involve motorcycles or bicycles. Of the cycle riders, approximately 70% have multiple injuries. Overall, 70% have supraclavicular lesions; of these, 70% have at least one root avulsion. At least 70% of patients with a root avulsion also have avulsions of the lower roots (C7, C8 or T1). Finally, of patients with lower root avulsion, nearly 70% will experience persistent pain.

Correspondence

Mario G. Siqueira
Rua Virgílio de Carvalho Pinto 381 / 42
05415-030 São Paulo SP - Brasil
E-mail: mgsiqueira@uol.com.br

Received 2 February 2011

Received in final form 10 March 2011

Accepted 24 March 2011

¹Peripheral Nerve Surgery Unit, Division of Functional Neurosurgery, University of São Paulo Medical School, São Paulo SP, Brazil; ²Neurosurgical Department, Hospital do Servidor Público do Estado de São Paulo, São Paulo SP, Brazil.

Despite major progress made over the last decades, the results from surgical treatment of traumatic brachial plexus lesions are far from ideal. Nevertheless, although current surgical methods produce a functional result that is significantly worse than the original performance, it is still much better than what would be achieved without nerve reconstruction. The purpose of this paper was to review the mechanisms of traumatic brachial plexus injuries in adults and their clinical evaluation, surgical treatment and outcomes.

History

The first report of successful surgery on a traction injury of the brachial plexus was reported in 1900, by Thornburn². Following publication of this report, surgical repair of brachial plexus injuries was performed on a few occasions in the early 20th century^{3,4} with apparently good results, but the initial enthusiasm was replaced with pessimism because of the poor subsequent results^{5,6}. In the early 1960s, the skepticism about the results from the surgical treatment was so intense that the treatment advocated for complete root avulsions was above-elbow amputation, combined with shoulder fusion in slight abduction and flexion, and complemented by a forearm-hand prosthesis.

With the introduction of operating microscopes, the modern period of brachial plexus surgical reconstruction began, thereby revitalizing the field. In 1969, Milesi started to repair defects of the brachial plexus under magnification using cutaneous nerves as grafts. In 1973, he published his series⁷ and was shortly followed by that of Narakas, who reported similar encouraging results⁸. Improvements in optics, imaging, electrodiagnosis, surgical instruments and suture material, and the dedication of many surgeons around the world, like Kline and Nulsen⁹, Hudson and Tamner¹⁰, Gilbert and Tassin¹¹, Brunelli and Monini¹², Merle D'Aubigne and Deburge¹³, Alnot et al.¹⁴, Allieu¹⁵, Samii and Kahl¹⁶, Leffert¹⁷, Terzis et al.¹⁸, Kotani et al.¹⁹, Jamieson and Hugues²⁰ and Sedel²¹, among many others, gave rise to evolution in the treatment of brachial plexus lesions. Today, it is possible to achieve reliable restoration of elbow flexion and shoulder abduction in many patients and, possibly, useful hand grip in a few selected cases.

Mechanisms of injury

Stretch/contusion is the most common mechanism of injury of the brachial plexus. Most frequently, these injuries result from high-speed motor vehicle accidents that produce a sudden shoulder movement downwards and backwards, and a neck movement in the opposite direction as the patient strikes the ground (Fig 1). The caudal traction of the shoulder and arm usually in-

juries the upper roots of the plexus (C5 and C6), lateral traction injures the C7 root and cranial traction injures the lower roots (C8 and T1). In the large series of David Kline (1,019 brachial plexus lesions operated between 1968 and 1998), reported by Kim et al.²², 509 injuries (49%) were produced by stretch/contusion.

Gunshot wounds produce penetrating injuries and are often associated with vascular injuries. Although less frequent, this type of plexus injury is becoming progressively more common. It is usually produced by low-velocity missile wounds from handguns. The lesions are mostly in continuity, but can also transect elements. The force associated with the injury varies and depends on the missile caliber, velocity and angle of incidence. Missile injuries produced by low-velocity shell fragments damage the nerve elements by direct impact and tend to be associated with less damage to the plexus. High-velocity gunshot injuries damage the nerve elements through three different mechanisms: direct impact (rare), shock wave effects and cavitation effects. The latter two mechanisms provoke compression and stretching of the nerve²³. These lesions are more intense and usually fail to recover spontaneously. In Kline's series²², 118 gunshot wounds represented 12% of the brachial plexus injuries.

Lacerations can result from sharp lacerations (knives or glass), or blunt transection (automobile metal, fan and motor blades, or animal bites) and can either transect a portion (most common) or the entire plexus. Vascular lesions are frequently associated with laceration injuries. Kim et al reported that there were 71 lacerations in Kline's series (7%)²².

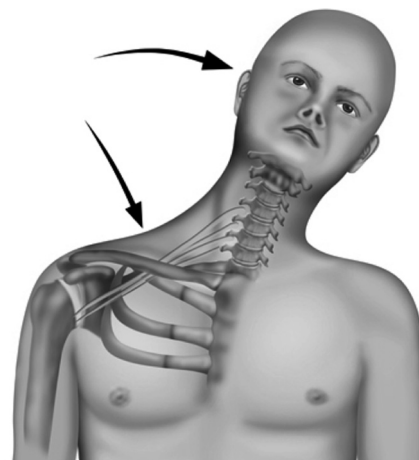


Fig 1. Most frequent mechanism for stretch-contusion lesions of the brachial plexus. Drawing modified from Siqueira MG, Martins RS, Socolovsky M. Mecanismos das lesões traumáticas do plexo braquial em adultos. In: Siqueira MG, Martins RS. Lesões do plexo braquial. Rio de Janeiro, Di Livros, 2011 (in press). With permission from the authors and publisher.

Evaluation of neural function

Patients should be evaluated early on, but this is often not possible because the brachial plexus lesion is only one part of a multisystem trauma, and the deficits are either overlooked or their evaluation is deferred while life-threatening injuries are treated²⁴. If nerve surgeons are aware of patients' early post-injury neurological status, they can determine in subsequent evaluations whether the neurological deficit is stable, improving or deteriorating with time.

The goal in clinical evaluations on brachial plexus injuries is to determine as accurately as possible the extent of the nerve injury and, based on that information, to determine whether or not the patient is a candidate for either early surgery or a period of further observation. For this purpose, a thorough neurological examination cannot be replaced by any other type of test. The active and passive ranges of motion of the upper limb should be recorded, as well as the presence or absence of reflexes. All the elements of the brachial plexus must be evaluated (motor and sensory functions), in order to diagnose whether the element is totally or partially compromised.

However, it is important to bear in mind that this gradation is not static; with time, partial injuries may fail to improve, while recovery from complete lesions may eventually occur. Brachial plexus injuries are, in the great majority of cases, a blend of completely and incompletely injured elements. The clinical course should be followed by means of repeated (monthly) clinical examinations.

Lesion grades

Although Sunderland's classification²⁵ of nerve injury is more detailed, Seddon's system²⁶ is simpler and more useful for clinical purposes.

Neuropraxia, the mildest form of nerve damage, is probably due to focal demyelination. The patient presents with complete motor loss, with relative sparing of sensory function that disappears within days or weeks.

Axonotmesis is a more severe grade of injury, in which there is disruption of axons with preservation of the basal lamina tubes. Complete motor and sensory loss occurs, but spontaneous recovery along the intact endoneurial tubes is possible, provided that the distance from the lesion to the end organs is not too large. The recovery takes time, because axon regrowth takes place at a rate of approximately 1 mm per day under ideal conditions²⁷.

In Neurotmesis, which is the most severe grade of injury, both the axons and the supporting connective tissue are disrupted. In such lesions, there is no possibility of spontaneous recovery and surgical repair is mandatory.

Immediately after nerve injury, the clinical presentation is identical for all three grades of injury. Only after some time will the exact grade become established, based

on whether the deficit improves or not. It is important to bear in mind that brachial plexus injuries are usually a combination of all three different grades of lesion.

Lesion location

Most traumatic brachial plexus lesions in adults are closed injuries involving the supraclavicular region. The roots and trunks are more commonly affected than the divisions, cords or terminal branches. Diagnosing the lesion location in relation to the ganglion of the dorsal root is very important, because pre and postganglionic lesions should be approached differently. While postganglionic lesions can sometimes recover spontaneously, preganglionic lesions cannot and should be identified as soon as possible. Diagnosing preganglionic lesions is usually based on indirect evidence that heightens the suspicion of a proximal injury but does not prove its existence: [1] presence of Horner's syndrome; [2] injury to very proximal nerves like the long thoracic nerve (scapular winging), dorsal scapular nerve (rhomboid paralysis) and phrenic nerve (paralysis of the ipsilateral hemidiaphragm); [3] denervation of cervical paraspinal muscles seen in electromyography examinations²⁸; [4] normal sensory conduction assessments, despite the absence of feeling in the areas served by the nerves examined; and [5] absence of Tinel's sign in the supraclavicular fossa. Frequently, however, preganglionic and postganglionic injuries coexist, and hence the full extent of the injury may not be perceived until surgical exploration is undertaken. When a diagnosis of postganglionic lesion is made, it is important to localize it in the supra or infraclavicular area. In infraclavicular injuries, the cords, their branches and the origins of the peripheral nerves are involved.

Pain

Pain is present in up to 80% of adult patients who sustained a brachial plexus lesion²⁹. Usually, it is reasonably controlled with drugs and subsides within months. When the pain is intense and starts early, this suggests deafferentation and root avulsion. This severe neuropathic pain reacts poorly to conventional therapy and has two distinct features: constant burning background pain and periodic sharp paroxysms of shooting pain. A considerable number of patients with root avulsions and this severe type of pain will need a procedure for intraspinal coagulation of the dorsal root entry zone (DREZ) as their definitive treatment.

Electrophysiological and imaging evaluations

Whatever the clinical picture, all patients with traumatic paralysis of the brachial plexus who have not shown signs of recovery by the 30th day after the injury

should undergo additional work-up, including electrodiagnostic tests and image evaluations, in order to come to a decision regarding surgery.

Electromyography (EMG) can determine the distribution and extent of the lesion; can evaluate muscles that are difficult to test clinically; and can quantify the extent of denervation. Because of Wallerian degeneration, the EMG signs of denervation are not reliably demonstrated until three to four weeks after nerve injury³⁰, and for this reason, this examination should not be done earlier. Another important use of EMG examinations is in serial evaluations on the injury, to search for signs of reinnervation, which are seen several weeks before the onset of detectable voluntary muscle contraction.

Besides the importance of nerve conduction velocity (NCV) analysis in diagnosing preganglionic lesions that was mentioned earlier, these evaluations are useful in investigating neuropraxic injuries. Injured motor axons continue to conduct action potentials for several days, but as the Wallerian degeneration proceeds, this ability disappears. If distal motor conduction is positive after this period, even though the related muscles are still paralyzed, the injury is probably a conduction block (neuropraxia).

Somatosensory evoked potentials (SEPs) are obtained by means of stimulation of the distal median, radial and ulnar nerves and recording over the spinous process of C2 and the contralateral somatosensory cortex³¹. No spinal or cortical potential can be elicited in preganglionic lesions, despite their normal peripheral sensory NCV. In postganglionic lesions, NCV and SEPs are both absent, as in combined pre and postganglionic lesions. The clinical value of such analyses is limited by a number of factors: [1] because each peripheral nerve is made up of fibers from two or more spinal nerves, some evoked potentials will be transmitted to the brain, unless multiple roots are avulsed; [2] only a small number of functional axons is needed to transmit an evoked potential; and [3] there is no adequate peripheral stimulation site for the important C5 root. Thus, a negative SEP examination is clinically more useful than a positive one.

Intraoperative nerve action potential (NAP) is valuable for evaluations on lesions in continuity, but should be delayed until at least three months after the injury, to allow time for sufficient axonal regrowth. Using bipolar stimulating and recording electrodes, this can detect axonal regrowth across the lesion³². If a NAP can be elicited, many viable axons will have traversed the lesion and probably will reinnervate its previous end organs. In such cases, neurolysis is sufficient treatment. However, when no NAP can be recorded distally to the lesion, a neurotmetic lesion probably exists, requiring resection and grafting.

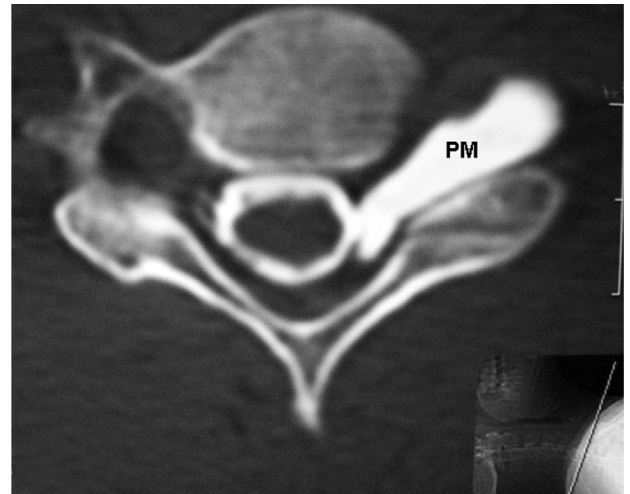


Fig 2. Cervical myelography followed by computed tomography scanning showing a large pseudomeningocele (PM).

Plain X-Rays of the neck and shoulder can document associated fractures and shrapnel from gunshot wounds. Chest X-Rays produced after inspiration and expiration can demonstrate the presence of hemidiaphragm palsy, thus indicating ipsilateral phrenic nerve injury. When vascular injury is suspected, arteriography or magnetic resonance angiography may confirm the arterial integrity or the patency of a previous vascular repair.

Cervical myelography followed by computed tomography (CT) scanning is helpful in defining the level of nerve root injury, through assessing the status of the egress of the spinal nerves from the spinal cord in supraclavicular brachial plexus injuries^{33,34}. Traction applied to the brachial plexus nerve roots may detach its rootlets from the spinal cord and, at the same time, tear the arachnoid sleeve of the root and pull it out into the neural foramen. The intrathecal contrast material will fill the sleeves, giving rise to the characteristic pseudomeningocele (Fig 2). Although highly suggestive, these meningoceles do not provide proof of rootlet avulsion. CT-myelography should be obtained at least one month after the injury in order to allow time for blood clots to dissipate and for pseudomeningoceles to fully form. This examination presents false-positive and false-negative result rates of 5 to 10%.

Although CT-myelography is still considered to be the "gold standard" for studying root lesions, recent advances in magnetic resonance imaging (MRI) are producing images of much higher resolution, such that MRI can now match the diagnostic accuracy of CT-myelography^{35,36}. Because of continuous development of MRI techniques, this imaging examination will soon become the most important method for evaluating brachial plexus injuries. Besides providing a noninvasive means of

detecting nerve root avulsion and easily demonstrating abnormal cerebrospinal fluid collection in pseudomeningeoceles in T2-weighted images, MRI can also show spinal cord edema (an indirect sign of nerve root avulsion), postganglionic lesions such as post-injury fibrosis and neuromas and associated inflammation or edema.

Indications for surgery

For multisystem trauma patients, the initial management is directed towards the associated life-threatening conditions, which include head, spinal and chest and vascular injuries. The need for surgical treatment of the brachial plexus lesion will depend on the degree of preliminary regeneration: approximately two thirds of the cases recover spontaneously over the first months. Surgery should be performed in the absence of clinical or electrical evidence of recovery or when spontaneous recovery is impossible. During the variable observation period, physical therapy should be provided to prevent contractures and to strengthen functioning muscles. Virtually all patients without significant spontaneous recovery may benefit from microsurgical reconstruction of the brachial plexus.

Timing of the surgical repair

The best time for the operation depends on the mechanism and type of injury and this has been debated over the years, with a move towards early intervention³⁷.

In cases of stretch/contusion lesions, a period of conservative management of three to four months prior to operative exploration is usually favored. Early evidence of spontaneous recovery takes longer to manifest in cases of stretch injuries than it does in cases of gunshot wounds, since the lesions are longer in stretch injuries³⁸. Earlier surgery is indicated for patients with total palsy and strong evidence of a preganglionic lesion.

Associations with vascular injuries may warrant emergency repair in cases of gunshot wounds, but low-velocity gunshot wounds are usually treated later on (3-4 months after the injury), just like in cases of stretch/contusion lesions, because most of these lesions have a neuropraxic component. On the other hand, high-velocity gunshot wounds are usually associated with significant soft-tissue damage and demand early surgical exploration.

In cases of sharp lacerations, emergency repair should be performed, especially if loss of function is complete regarding the distribution of one or more elements of the plexus. Early intervention is also indicated in cases of increasing neurological deficit, which may be associated with progressive pain due to hematoma, arteriovenous fistula or pseudoaneurysm. About one third of the patients with lacerating injuries to the brachial plexus

undergo acute surgical exploration because of suspected or angiographically-proven vascular injuries, and nerve surgeons should be involved in this emergency procedure. First to assess the nerve damage and secondly to guide the vascular surgeon in dissection of the distal vessels that present a close relationship to the plexus elements. If early exploration demonstrates the presence of blunt laceration, the nerve is fixed to adjacent planes to lessen the retraction and is repaired secondarily two to three weeks later. This delay in the repair allows the damage in each stump to become delimited.

Repair priorities

The surgical plan should be individualized, depending on the extent of the injury and the reconstruction options available. In extensive lesions, a priority list must be established, based on functional importance and the prognosis after nerve reconstruction. There is no consensus in the literature about the level of importance of the different functions to be reinnervated, except for the two first indications. The following list presents, in our opinion, an ideal sequence of priorities: [1] Elbow flexion against gravity has the highest priority because its restoration allows the extremity to be better positioned in space and the hand to flex toward the trunk and mouth for use (Fig 3A). [2] Shoulder stabilization and recovery of abduction and external rotation comes next, because an unstable or contracted shoulder will impede use of the extremity (Fig 3B). [3] Elbow extension. [4] Brachiothoracic pinch (abduction of the arm against the chest). [5] Wrist and finger extension. [6] Wrist and finger flexion. [7] The ulnar innervated structures are the last priority because of the poor prognosis for recovery.

In extensive lesions, no matter what type of motor reconstruction is done, surgeons should always include sensory transfers in the surgical plan, in an attempt to recover protective sensation of the hand (median nerve area).

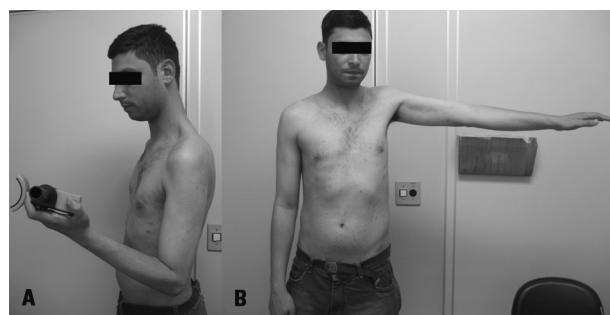


Fig 3. Picture of a patient fourteen months after brachial plexus microsurgical reconstruction of an upper trunk lesion. [A] recovery of elbow flexion; [B] shoulder stabilization and recovery of abduction.

Operative approaches and surgical techniques

An anterior operative approach is used in the great majority of cases of brachial plexus trauma³⁹. Depending on the type and extent of the lesion, the approach may be supraclavicular, infraclavicular or combined. Four surgical techniques are commonly used in repairing brachial plexus injuries:

[1] Direct repair by means of end-to-end suture –

On rare occasions, an end-to-end suture is possible with minimal tension, usually in cases of sharp laceration;

[2] External neurolysis –

Neurolysis is the surgical freeing of intact nerves from constricting scar tissue. It is carried out using a No. 15 scalpel blade or Metzenbaum scissors. Nerve segments are freed circumferentially and in the proximal and distal directions from either side of the injured segment. External neurolysis is a necessary prerequisite for implementing other techniques and for intraoperative electrical examinations and may be the only repair required when low-amplitude and slow-conducting regenerative NAPs are obtained in intraoperative nerve action potential recordings on non-regenerating nerves in situations of a lesion in continuity;

[3] Nerve grafting –

When there is a severe postganglionic rupture or a lesion in continuity with no recordable NAP, the injured element(s) should be resected. A large interneural gap usually persists and the retracted stumps cannot be brought together without significant

tension. In this situation, interposition of autografts is needed in order to bridge the stumps and enable grouped fascicular repair (Fig 4A). In most cases, the sural nerve, which can yield up to 30 cm of nerve for grafting, is used as a cable graft. Graft repair should not be performed when the fascicular structure cannot be viewed from the proximal stump (scar) or when the roots are severely damaged or avulsed at an intradural level⁴⁰. Sometimes only a portion of the cross-section of the plexus element is damaged. The damaged segment is split away from the nerve segment of normal appearance, and if no NAP is recorded across it, it is resected and repaired by means of grafting. This partial repair of the element is called split-repair.

[4] Nerve transfer –

In nerve transfer, a functioning nerve of lesser importance is transferred to a distal denervated nerve that is functionally more important. Primarily used only to treat preganglionic injuries, nerve transfers reduce the time taken to establish reinnervation by decreasing the distance between the nerve repair site and the end organ. This has given rise to expansion of its original indication such that it is now also used for delayed cases, when faster recovery is desired. The commonest donor nerves for transfers include the distal spinal accessory nerve⁴¹, intercostal nerves⁴² (Fig 4B) and medial pectoral nerve⁴³. More recently, fascicles from a functioning ulnar and/or median nerve⁴⁴⁻⁴⁶ have started

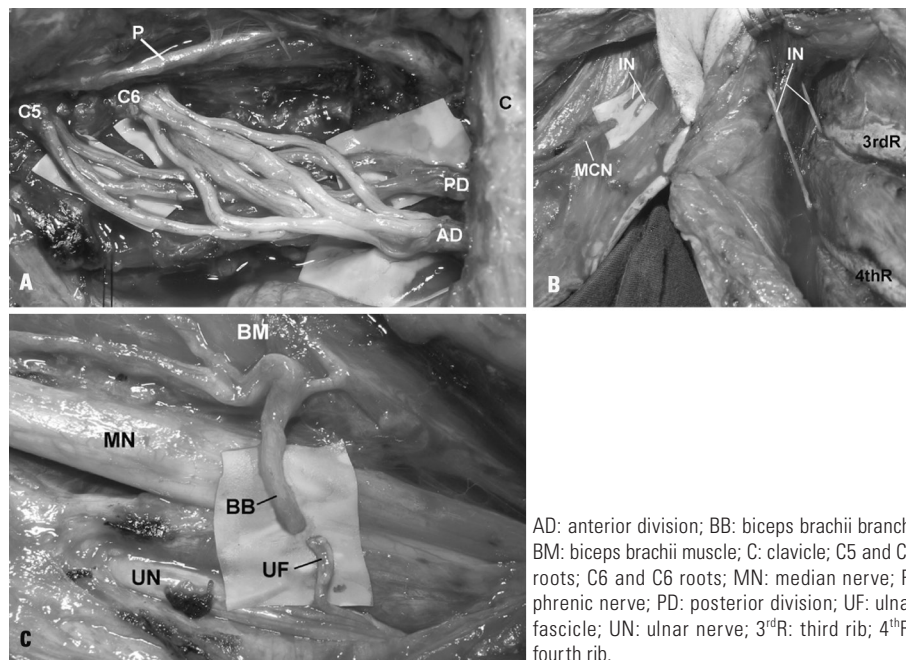


Fig 4. Surgical techniques for treatment of brachial plexus lesions. [A] Microsurgical reconstruction of an upper trunk lesion with autografts (sural nerve). [B] Transfer of two intercostal nerves (IN) to reinnervate the musculocutaneous nerve (MCN). [C] Transfer of motor fascicle of the ulnar nerve (related to the flexor carpi ulnaris muscle) to reinnervate the motor branch of the musculocutaneous nerve that goes to the biceps brachii muscle.

to be used (Fig 4C), as well as branches of the radial nerve that go to the triceps muscle⁴⁷, the phrenic nerve⁴⁸ and the contralateral C7⁴⁹. The deep cervical plexus and hypoglossal nerve have also been used, but poor motor recovery has been reported⁵⁰.

Secondary reconstruction

Secondary reconstruction may be necessary to improve function, either to augment partial recovery or to obtain function when none has been achieved. This may include soft-tissue reconstruction (e.g. tendon/muscle transfer or free muscle transfer) and bone procedures (e.g. arthrodesis or osteotomy). A combination of these techniques is often used.

Postoperative follow-up

Surgeons should provide information to make patients and their families aware of the long periods of waiting that are necessary for muscle reinnervation to take place. Before undergoing surgery, patients should already have been taught the exercises needed for maintaining normal range of motion in the paralyzed joints. Therapists can teach this and record the progress, but patients must be co-responsible for carrying out the exercises. The shoulder and elbow are immobilized in a splint for two weeks, and then the patient should restart the rehabilitation program. After the healing of the surgical wound, the progress of nerve regeneration is evaluated every four months for at least two years, in order to assess whether full recovery is achieved.

Outcome

Many factors adversely influence functional recovery after surgical treatment of brachial plexus lesions, such as the patient's age, the interval between injury and surgery (denervation period), the two coaptation sites, long nerve grafts, scar tissue, ischemia and the degree of root lesion^{51,52}. Young patients, with short denervation periods (<6 months) and postganglionic lesions, usually present a better outcome than do patients with root avulsions. Isolated lesions of the upper trunk present the best outcome: the hand is not compromised and the targets for reinnervation are close to the donors. Persistent pain (lasting for more than six months) is a bad prognostic sign for neurological recovery, no matter where the lesion is located. It is very difficult to compare the results from surgical treatments for traumatic brachial plexus lesions because of differences in patient selection, reconstructive techniques and outcome evaluations, but some general conclusions can be reached by analyzing the larger series in the literature: In cases of supraclavicular lesions, in which paralysis affects the upper roots, nerve grafting provides good results in approximately 75% of

the cases. Involvement of the upper trunk or the C5-C6 roots in the interscalene region is a favorable setting for nerve repair or grafting. Injury to middle and lower roots carries a poor outcome prognosis. In cases of infraclavicular lesions, the results from nerve grafting in lesions that are relatively close to the innervated muscle, i.e. the musculocutaneous nerve, are generally good. A satisfactory recovery in 70-80% of the cases can be expected.

The results from grafting median, radial and ulnar nerves in lesions that are distant from the innervated muscle, i.e. the lateral and medial cord, are variable. Reinnervation of the wrist and the digital flexor and extensor can be expected in 50-60% of the cases but no reinnervation of the intrinsic muscles of the hand should be expected. Sensory reinnervation of the median nerve area can be achieved in 70-80% of the cases.

Selective combination of nerve transfers provides moderate to good shoulder and elbow control. Although some wrist and finger movement may occasionally be achieved, the results from restoration of useful hand function are still far from satisfactory.

The use of combinations of nerve grafts and nerve transfers is increasing and, apparently, improving the results.

Conclusion

Even in the hands of expert surgeons, the vast majority of patients with brachial plexus injuries remain handicapped, especially with regard to hand functioning. Further research is needed on innovative reconstructive procedures, especially for root avulsion treatments. Use of these new techniques, along with better understanding of central-peripheral function integration, may provide improved results and greater purposeful function for our patients in the future.

REFERENCES

1. Narakas AO. The treatment of brachial plexus injuries. *Int Orthop* 1985; 9:29-36.
2. Thoburn W. Secondary suture of the brachial plexus. *Brit Med J* 1900;1: 1073-1075.
3. Kennedy R. Suture of the brachial plexus in birth paralysis of the upper extremities. *BMJ* 1903;1:298-301.
4. Taylor AF. Results from the surgical treatment of brachial birth palsy. *JAMA* 1907;48:96-104.
5. Nulsen FE, Slade HW. Recovery following injury to the brachial plexus. In Woodhall B, Beebe G (Eds). *Peripheral nerve regeneration: a follow-up study of 3,365 World War II injuries*. Washington, DC: U.S. Government Printing Office, 1956:389-408.
6. Bonney G. Prognosis in traction lesions of the brachial plexus. *J Bone Joint Surg* 1959;41:4-35.
7. Millesi H. Résultats tardifs de la greffe nerveuse intrafasciculaire. *Chirurgie réparatrice des lésions du plexus brachiale*. *Rev Med Suisse Romande* 1973; 93:511-519.
8. Narakas AO. Surgical treatment of traction injuries of the brachial plexus. *Clin Orthop* 1978;133:71-90.
9. Kline DG, Nulsen FE. The neuroma incontinuity: its preoperative and operative management. *Surg Clin North Am* 1972;52:1189-1209.

10. Hudson AR, Tamner B. Brachial plexus injuries. In: Wilkins RH, Rengachary SS (Eds). *Neurosurgery*. New York: McGraw Hill, 1985:1817-1832.
11. Gilbert A, Tassin JL. Obstetrical palsy: a clinical, pathologic and surgical review. In: Terzis JK (Ed). *Microreconstruction of nerve injuries*. Philadelphia: WB Saunders, 1978:529-553.
12. Brunelli G, Monini L. Neurotization of avulsed roots of brachial plexus by means of anterior nerves of the cervical plexus. *Clin Plast Surg* 1984;11: 149-152.
13. Merle D'Aubigne RN, Deburge A. Etiologic evolution et prognostic des paralysies traumatiques du plexus brachial. *Rev Chir Orthop Reparatrice Appar Mot* 1967;53:23- 42.
14. Alnot JY, Jolly A, Frot B. Direct treatment of nerve lesions in brachial plexus injuries in adults: a series of 100 operated cases. *Int Orthop* 1981;5:151-168.
15. Allieu Y. Exploration et traitement direct des lésions nerveuses dans les paralysies traumatiques par elongation du plexus brachial chez l'adulte. *Rev Surg Orthop* 1975;63:107-122.
16. Samii M, Kahl RI. Clinische resultate der autologen nerven transplantation. *Nelssunger Med Mitteil* 1972;46:197-202.
17. Leffert RD. *Brachial plexus injuries*. New York: Churchill Livingstone, 1985.
18. Terzis JK, Liberson WT, Maragh HA. Motorcycle brachial plexopathy. In: Terzis JK (Ed). *Microreconstruction of nerve injuries*. Philadelphia: WB Saunders, 1987:361-384.
19. Kotani PT, Tushima Y, Matsuda H, Suzuki T, Ishizaki Y. Postoperative results of nerve transposition in brachial plexus injury. *Seikei Geka* 1971; 22:963- 966.
20. Jamieson A, Hugues S. The role of surgery in the management of closed injuries of the brachial plexus. *Clin Orthop Relat Res* 1980;147:210-215.
21. Sedel L. Repair of severe traction lesion of the brachial plexus. *Clin Orthop Relat Res* 1988;237:62-63.
22. Kim DH, Cho YJ, Tiel RL, Kline DG. Outcomes of surgery in 1019 brachial plexus lesions treated at Louisiana State University Health Sciences Center. *J Neurosurg* 2003;98:1005-1016.
23. Samardzic MM, Rasulic LG, Grujicic DM. Gunshot injuries to the brachial plexus. *J Trauma* 1997;43:645-649.
24. McGillicuddy JE. Surgical anatomy and management of brachial plexus injury. In: Tindall GT, Cooper PR, Barrow DL (Eds). *The practice of neurosurgery*. Vol 3. Baltimore: Williams & Wilkins 1996:2859-2877.
25. Sunderland S. A classification of peripheral nerve injuries producing loss of function. *Brain* 1951;74:491-516.
26. Seddon HJ. A classification of nerve injuries. *Br Med J* 1942;2:237-239.
27. Sunderland S. Rate of regeneration of I:sensory nerve fibers and II: motor nerve fibers. *Arch Neurol Psychiatry* 1947;58:1-14.
28. Bufalini C, Pescatore G. Posterior cervical electromyography in the diagnosis and prognosis of brachial plexus injuries. *J Bone Joint Surg* 1969; 51:627-631.
29. Bruxelle J, Travers V, Thiebaut JB. Occurrence and treatment of pain after brachial plexus injury. *Clin Orthop Rel Res* 1988;237:87-95.
30. Warren J, Gutmann L, Figueroa AS Jr, Bloor BM. Electromyographic changes of brachial plexus root avulsion. *J Neurosurg* 1969;31:137-140.
31. Landi A, Copeland SA, Wynn Parry CB, Jones SJ. The role of somatosensory evoked potentials and nerve conduction studies in the surgical management of brachial plexus injuries. *J Bone Joint Surg* 1980;62:492-496.
32. Kline DG, Happel LT. A quarter century's experience with intraoperative nerve action potential recording. *Can J Neurol Sci* 1993;20:3-10.
33. Carvalho GA, Nikkiah G, Matthies C, Penkert G, Samii M. Diagnosis of root avulsions in traumatic brachial plexus injuries: value of computerized tomography myelography and magnetic resonance imaging. *J Neurosurg* 1997;86:69-76.
34. Walker AT, Chaloupka JC, de Lotbiniere AC, Wolfe SW, Goldman R, Kier EL. Detection of nerve rootlet avulsion on CT myelography in patients with birth palsy and brachial plexus injury after trauma. *AJR Am J Roentgenol* 1996;167:1283-1287.
35. Nakamura T, Yabe Y, Horiuchi Y, Takayama S. Magnetic resonance myelography in brachial plexus injury. *J Bone Joint Surg* 1997; 79:764-769.
36. Doi K, Otsuka K, Okamoto Y, Fujii H, Hattori Y, Baliarsing AS. Cervical nerve root avulsion in brachial plexus injuries: magnetic resonance imaging classification and comparison with myelography and computerized tomography myelography. *J Neurosurg* 2002;96:277-284.
37. Terzis JK, Vekris MD, Soucacos PN. Outcomes of brachial plexus reconstruction in 204 patients with devastating paralysis. *Plast Reconstr Surg* 1999; 104:1221-1240.
38. Nagano A. Treatment of brachial plexus injury. *J Orthop Sci* 1998;3:71-80.
39. Kline DG, Hudson A, Kim DH. *Atlas of peripheral nerve surgery*. Philadelphia: WB Saunders, 2001.
40. Kim DH, Murovic JA, Kline DG. Brachial plexus injury: mechanisms, surgical treatment and outcomes. *J Korean Neurosurg Soc* 2004;36:177-185.
41. Songcharoen P, Mahaisavariya B, Chotigavanich C. Spinal accessory neurotization for restoration of elbow flexion in avulsion injuries of the brachial plexus. *J Hand Surg* 1996;21:387-390.
42. Yeoman P, Seddon HJ. Brachial plexus injuries: treatment of the flail arm. *J Bone Joint Surg* 1961;43:493-500.
43. Narakas AO. Thoughts on neurotization or nerve transfers in irreparable nerve lesions. *Clin Plast Surg* 1984;11:153-159.
44. Oberlin C, Beal D, Leechavengvongs S, Salon A, Dauge MC, Sarcy JJ. Nerve transfer to biceps muscle using a part of ulnar nerve for C5-C6 avulsion of the brachial plexus: Anatomical study and report of four cases. *J Hand Surg* 1994;19A:232-237.
45. Hou Z, Xu Z. Nerve transfer for treatment of brachial plexus injury: Comparison study between the transfer of partial median and ulnar nerves and that of phrenic and spinal accessory nerves. *Chin J Traumatol* 2002;263-266.
46. Weber RV, Mackinnon SE. Nerve transfers in the upper extremity. *J Am Soc Surg Hand* 2004;4:200-213.
47. Leechavengvongs S, Witoonchart K, Uerpairojkit C, Thuvasethakul P. Nerve transfer to deltoid muscle using the nerve to the long head of the triceps, part 2: a report of 7 cases. *J Hand Surg* 2003;28:633-638.
48. Gu YD, Ma MK. Use of the phrenic nerve for brachial plexus reconstruction. *Clin Orthop* 1996;323:119-121.
49. Gu YD. Cervical nerve root transfer from the healthy side in the treatment of brachial plexus root avulsion. *Natl Med J Chin* 1989;69:563-565.
50. Chuang DC. Neurotization procedures for brachial plexus injuries. *Hand Clin* 1995;11:633-645.
51. Leffert R. *Brachial plexus injuries*. Edinburgh: Churchill Livingstone, 1986.
52. Narakas AO. Brachial plexus injuries. In: McCarthy (Ed). *Plastic surgery, the hand, Vol.7, Part 1*. Philadelphia: WB Saunders, 1990:4776-4815.