Brachial Plexus Injuries in Adults: A Narrative Review of the Literature

Lesiones del plexo braquial en adultos: Una revisión narrativa de la literatura

Joaquín Ananias[1] Paula A. Pino[1,2]

[1] Department of Orthopedics and Traumatology, Facultad de Medicina, Pontificia Universidad Católica de Chile, Santiago, Chile
[2] Equipo Extremidad Superior, Instituto Teletón, Santiago, Chile

Address for correspondence Paula Pino, MD, Departamento de Traumatología y Ortopedia, Facultad de Medicina, Pontificia Universidad Católica de Chile, Diagonal Paraguay n° 362, 3er piso, Santiago, Chile (e-mail: palipinop@gmail.com).

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Abstract

Traumatic brachial plexus injuries comprise a wide spectrum of lesions that are potentially devastating to the functionality of the patients. The aim of the present review is to perform a narrative review of the literature focused on the diagnosis and study of brachial plexus injuries in adults, in addition to providing basic guidelines on the management of this complex pathology.

Level of evidence: Level V.

Keywords
- upper limbs
- nerve grafts
- microsurgery
- brachial plexus
- nerve transfers

Resumen

La patología traumática del plexo braquial comprende un amplio espectro de lesiones potencialmente devastadoras para la funcionalidad de los pacientes. El objetivo del presente trabajo es realizar una revisión narrativa de la literatura enfocada en el diagnóstico y estudio de las lesiones del plexo braquial en adultos, además de entregar nociones básicas sobre el manejo de esta compleja patología.

Nivel de evidencia: Nivel V.

Palabras clave
- extremidades superiores
- injertos nerviosos
- microcirugía
- plexo braquial
- transferencias nerviosas

Introduction

Traumatic brachial plexus injuries are severe lesions resulting in significant impairment in quality of life and function. They occur due to traction of the cervical roots of C5-T1, which form the brachial plexus, and cause motor deficits in muscle groups of the upper limb.

The most common mechanism is closed injuries due to traction in motor vehicle accidents and extreme sports, and they mainly affect men aged between 23 and 34 years.[1] Other highly-suspected lesion mechanisms include shoulder

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dislocations and open injuries, like those caused by bullets or bladed weapons in the path of the plexus.

The treatment of these lesions has evolved significantly in recent years, and it is possible to restore limb function in a significant number of patients. Strategies such as nerve reconstruction with grafts and nerve transfers restore limb innervation and function. These surgeries have a narrow therapeutic window, so timely diagnosis and early referral to a specialized center are critical to improve the prognosis.

The present is a literature review and summary of critical concepts regarding the diagnosis, study, and treatment of traumatic brachial plexus injuries in adults for general traumatologists.

Epidemiology

The estimated incidence of closed brachial plexus injuries in the United States ranges from 0.64 to 3.9/100 thousand people per year, affecting 1.2% of polytraumatized patients.[1] Historically, 70% of brachial plexus injuries in adults were estimated to result from traffic accidents, 70% of which were motorcycle accidents.[2] Recent studies have estimated that 93% of the patients suffered blunt injuries, and 67% of them were due to motorcycle accidents.[1] Open lesions are less frequent, accounting for only 3% of injuries. Among these, lacerations are more common in the infraclavicular plexus, and can compromise blood vessels or the airways. Last, firearm injuries are rare but can be severe, depending on the speed of the projectile; in addition, they involve extensive soft tissue-injuries, contamination, and bone comminution that can further injure the brachial plexus.

Anatomy

The proper diagnosis and treatment of these patients require knowledge of the anatomy of the plexus, which consists of spinal nerves or cervical roots from C5 to T1. Each root is the convergence of the ventral (motor) and dorsal (sensitive) roots emerging from the spinal cord. For the diagnosis, it is important to remember that the motor neuron soma is within the spinal cord (anterior horn), whereas the sensory neuron soma is at the ganglion of the dorsal root, outside the spinal cord.

The brachial plexus consists of five zones:

- Spinal nerve roots;
- Trunks;
- Divisions;
- Cords; and
- Terminal branches.

The nerve roots emerge from the foramina and form three trunks: the upper trunk (C5 and C6), the middle trunk (C7), and the lower trunk (C8 and T1). Each trunk has anterior and posterior divisions. The anterior divisions of the upper and middle trunk form the lateral cord. The posterior divisions of the three trunks form the posterior cord; and the anterior division of the lower trunk form the medial cord. Terminal branches of the lateral cord include the musculocutaneous nerve and lateral contribution to median nerve. The posterior cord forms the axillary and radial nerve, while the medial cord forms the ulnar nerve and medial contribution to the median nerve.

In addition, terminal nerves emerge from different areas of the brachial plexus, as shown in detail in **►Figure 1**. **►Table 1** summarizes the sensory and motor representation (key muscles) for each trunk.

Pathophysiology

There are different classifications for brachial plexus injuries depending on lesion severity, compromised nerve roots, and injury level, among other features. A critical element in assessing a patient with a brachial plexus injury is the distinction of preganglionic or postganglionic lesions (**►Figure 2**), which is based on whether the lesion is proximal or distal to the dorsal root ganglion at the exit level of the root in the cervical spinal cord. Preganglionic lesions occur proximal to the dorsal root ganglion. In these injuries, the root is avulsed from the spinal cord, and repair is not possible because there is no proximal root end connected with the cord. In contrast, postganglionic lesions occur distal to the dorsal root ganglion, damaging the nerve tissue in a range of degrees of severity.

In peripheral nerve injuries, axonal damage triggers a process of distal axonal degeneration called Wallerian degeneration, which occurs 24 to 36 hours after the injury, and has the goal of removing the damaged tissue. The regenerative process begins 2 to 3 weeks after the injury, with the formation of the axonal growth cone at the level of the proximal end. If the internal structure of the nerve is intact, the growth cone advances distally at a rate of 1 mm to 2 mm/day, depending on the patient's age, being lower in elderly subjects. The development of the growth cone can be tracked using the Hoffmann-Tinel sign, which translates neural irritability and results in the "electrical" sensation, pain, or tingling elicited at the affected nerve path by local percussion. At the distal level, Wallerian degeneration of the axons occurs when the nerve stimulus is interrupted, either by avulsion of the cervical root or by a postganglionic lesion with axonal damage. Until nerve conduction is restored, the endplate remains denervated, and muscle atrophy begins. A motor endplate not stimulated for 18 to 24 months suffers atrophy, resulting in permanent muscle fibrosis and inability to restore its function despite reinnervation.[3]

Seddon[4] classified peripheral nerve lesions into three groups: neuropraxia, axonotmesis, and neurotmesis. In neuropraxia-type lesions, the damage results in focal demyelination of the axons. These lesions feature a conduction block that recovers spontaneously within 3 months after remyelination, and they do not show Wallerian degeneration or a Hoffmann-Tinel sign. Axonotmesis-like lesions present axon damage, but the epineurium is intact. If the internal structure of the nerve is spared, the axonal growth cone advances distally, and the damaged nerve regenerates spontaneously. Damage and fibrosis at the internal structure of the nerve block the growth cone, resulting in the development of a

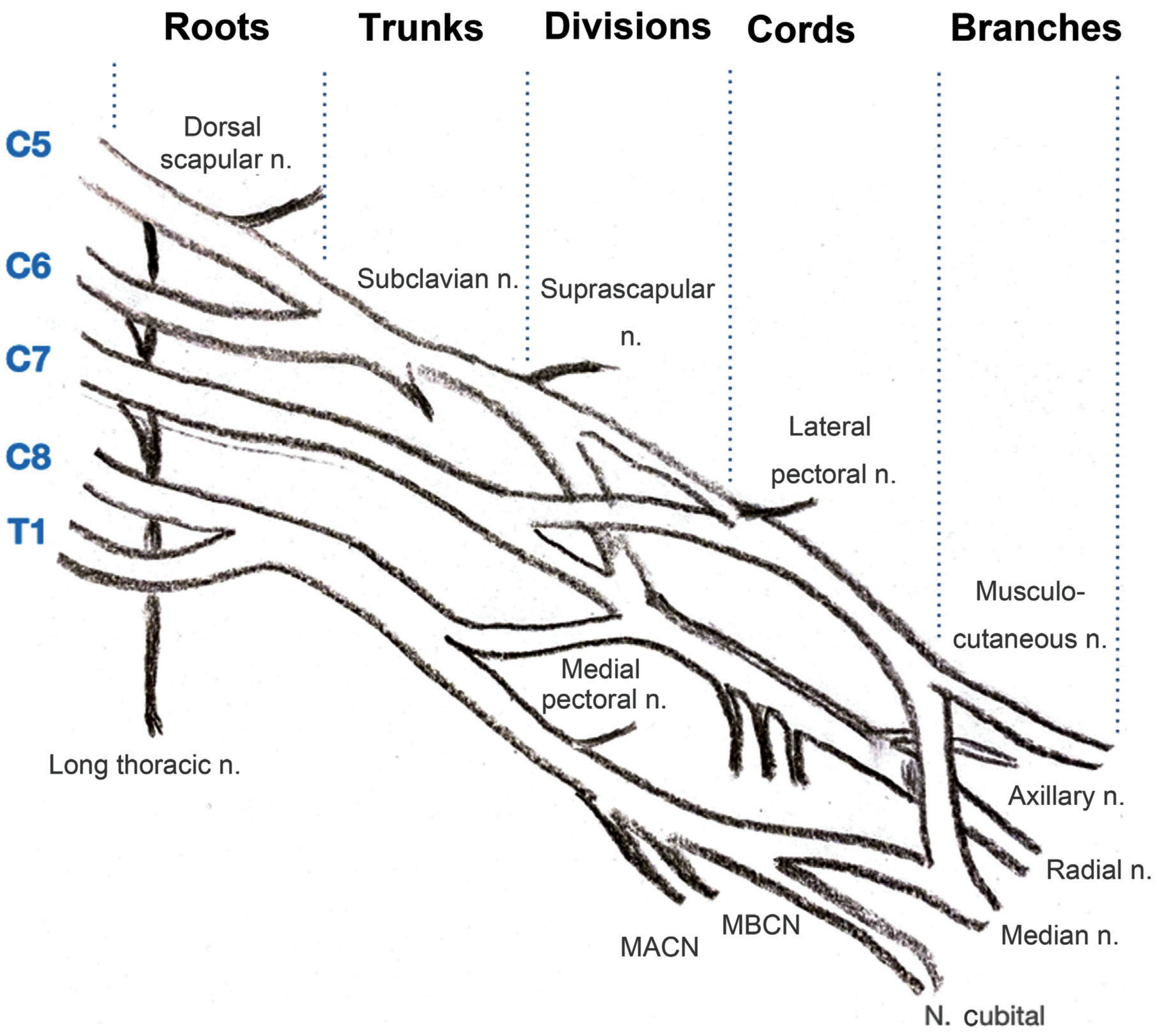

Fig. 1 Schematic illustration of the brachial plexus. Abbreviations: MACN: medial antebrachial cutaneous nerve; MBCN: medial brachial cutaneous nerve; N., nerve.

Table 1 Association of different key muscle groups and sensitive areas and their corresponding trunk

Trunk (roots)	Motor			Sensitive area
	Action	Nerve	Muscle	
Superior (C5 and C6)	Shoulder - external rotation and abduction	Suprascapular	Supraspinatus + infraspinatus	Lateral aspect of the arm (C5); lateral aspect of the forearm (C6); median region of the thumb and index finger (C6)
		Axillary	Deltoid	
	Elbow - flexion	Musculocutaneous	Biceps brachii; brachii	
Median (C7)	Elbow - extension	Radial	Triceps	Median region of the medium finger
	Hand - finger extension	Radial	Extensor digitorum communis	
Inferior (C8 and T1)	Hand - finger flexion; - intrinsic muscles	Median; ulnar	Flexor digitorum profundus; flexor digitorum superficialis; flexor pollicis longus; interosseous	Ulnar region (annular and little finger)

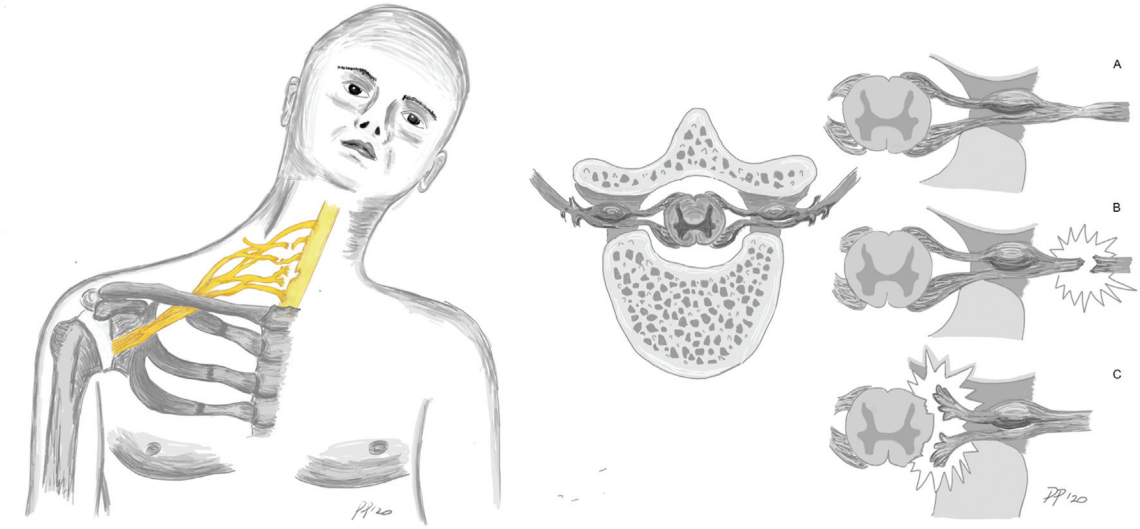

Fig. 2 Diagram representing, on the left, the different types of plexus injuries, and, on the right, the normal anatomy of two roots exiting the spinal cord is observed. (**A**) Postganglionic neuropraxia- or axonotmesis-like lesion, with root elongation alone. (**B**) A postganglionic neurotmesis-like lesion with complete axonal disruption. (**C**) Preganglionic injury with complete disconnection from the spinal cord.

disorganized tissue inside the nerve, called a continuity neuroma. Last, neurotmesis consists of complete section of the nerve.[5] These injuries present a physical distance between the two nerve ends which does not enable axonal regeneration. At the proximal end, the growth cone forms disorganized axon structures called neuromas. Like continuity neuromas, these structures are sensitive to palpation and percussion, which cause pain and a Hoffman-Tinel sign.[6] At first, axonotmesis- and neurotmesis-type lesions are clinically or electrophysiologically undistinguishable. In addition, different types of nerve lesions can coexist in postganglionic lesions, hindering their diagnosis and treatment indication.

Evaluation and Physical Examination

The assessment of a patient with suspected brachial plexus injury aims to locate the level of the lesion, detect the compromised nerves (roots, trunks, cords), determine lesion severity and the need for surgical treatment, and look for undamaged nerves/muscles that can act as sources for nerve or muscle transfers. A team with the technical capability to treat such injuries must perform this assessment as soon as possible. An early diagnosis of lesion severity and of its preganglionic or postganglionic nature is critical because preganglionic lesions must be operated on soon due to the lack of spontaneous recovery and to avoid motor plate atrophy. In contrast, since postganglionic lesions can recover spontaneously, a watchful approach may suffice.

The assessment of patients with a brachial plexus injury must be careful. Since a significant number of patients have associated injuries or multiple traumas, the diagnosis in commonly made one the patient is stabilized and is awake. The evaluation of brachial plexus injuries requires a detailed anamnesis of the mechanism of injury, the degree of energy, the associated lesions, age, comorbidities, dominance, and occupation. The mechanism is relevant to the injury pattern and may help define management. For example, open injuries often result in neurotmesis-like lesions, such as wounds by bladed weapons in the path of a nerve. These lesions warrant early exploration. On the other hand, low-speed gunshot wounds have a high probability of spontaneous recovery since the injuries usually result from the shock wave generated by the bullet, and there is transient neuropraxia alone. In contrast, high-speed gunshot wounds generally produce extensive soft-tissue damage and must be explored early.

The degree of energy of the trauma is critical to closed injuries.[1,7] High-energy injuries, especially traffic accidents, have a worse prognosis than that of low-energy injuries, such as falls with shoulder dislocation. The former produce multiple root lesions, mainly of a preganglionic nature, whereas injuries secondary to shoulder dislocation may be only neuropraxia, whose management is expectant. A trauma that forces the shoulder caudally and the cervical spine to the contralateral side mainly affects the superior roots of the plexus. In contrast, shoulder abduction trauma usually injures lower elements of the plexus.

When evaluating the patient at the emergency room, the physical examination must begin with an initial assessment of the trauma (airways, breathing, circulation, disability, and exposure, ABCDE), followed by a process of ruling out associated injuries. One should consider that 55% of the patients with brachial plexus injury also present upper-limb fractures; in addition, 43% have spinal fractures, and 30% present parenchymal injury.[1] Associated vascular injuries occur in 13% of these patients, especially those with scapulothoracic dissociation.[8]

A neurological examination of the limb occurs after ruling out associated injuries. This examination must be systematic and carefully recorded for later comparison during the follow-up visits. As aforementioned, the goals of this examination include the following:[9,10]

1. Identification of the nerves or muscle groups involved;
2. Establishment of the level of the injury: roots, trunks, divisions, cords, or terminal nerves; and
3. Definition of the preganglionic or postganglionic nature of the lesion.

Further evaluations also help define the severity of the injury. Subjects with neuropraxia recover spontaneously within three months. In axonotmesis-type lesions, distal progression of the Hoffmann-Tinel sign may be observed, along with spontaneous recovery depending on the distance between the lesion and the effector muscle. Last, lesions with root avulsion or neurotmesis require surgical treatment for recovery.

The physical examination begins with an inspection of the patient, who must be undressed from the waist up. Note the general appearance of the limb: muscle atrophy (which may not be evident in early stages), trophic skin changes, wounds, and scars. Next, inspect the patient's positioning, and evaluate the height of the shoulders (trapezius paralysis results in a drooping shoulder), glenohumeral subdislocation (deltoid and rotator cuff denervation), and hand posture. At this moment, evaluate for Horner syndrome: ptosis, miosis, anhidrosis, and enophthalmos on the affected side of the face.

Next, assess the passive range of motion of the compromised joints and perform a motor examination, which consists of the documentation of the strength of key muscle groups, recorded using the British Medical Research Council (BMRC) score, which ranges from 0 to 5 (**►Table 2**).

The main goal is to recognize the muscle groups affected, which indicate the site of the injury. A patient with an upper-trunk injury, for instance, presents a motor deficit in shoulder abduction and external rotation, as well as in elbow flexion. A C5-C6-C7 lesion also results in reduced active elbow extension. Patients with a lower-trunk injury present severe hand compromise, with an inability to flex the fingers and activate the intrinsic musculature (**►Table 1**). Many charts show the muscle groups examined to diagnose and locate a brachial plexus injury. **►Table 3** shows one of them, and it systematizes the initial assessment and subsequent follow-up.

A dermatome evaluation and two-point discrimination on the fingers determine sensitivity. One should look for the Hoffmann-Tinel sign at the cervical level (paresthesia on percussion with the fingers or reflex hammer). This sign is absent in preganglionic and postganglionic lesions of the neuropraxia type. As aforementioned, the Hoffmann-Tinel sign helps to assess reinnervation in axonotmesis-like or surgically-repaired lesions.

Avulsion-Related Signs

Some clinical signs suggest avulsion of the nerve roots from the cervical spine (**►Table 4**). The evaluation of branches that originate proximally in the brachial plexus, directly at the level of the nerve roots, reveal these signs; in addition, they are associated with Horner syndrome.

1) Winged scapula: the rhomboid parascapular and levator scapulae muscles maintain proper coordination between the glenohumeral and scapulothoracic movements. Their innervation is provided by nerves originating proximally at the level of the C5 root, in the case of the dorsal scapular nerve (rhomboid muscles), and at the level of the C5-C6-C7 roots, in the case of the long thoracic nerve (serratus anterior muscle). Clinically, a lesion in any of these nerves may be observed as a winged scapula, which involves damage to the roots of the proximal plexus (before the exit of these nerves) and is highly suggestive of root avulsion. To examine these muscles, ask the patient to push the wall with their hand and observe if there is an elevation of the inferomedial angle of the scapula (medial winged scapula).

2) Diaphragmatic paralysis: the phrenic nerve originates from the contribution of the C3 to C5 roots before trunk formation. Diaphragmatic paralysis is also a sign of a proximal upper root injury.

3) Horner syndrome: a set of clinical signs, including ptosis, miosis, anhidrosis, and enophthalmos, on the hemiface ipsilateral to the lesion. It results from a disruption of the sympathetic chain at the level of T1, and it usually indicates a T1 root avulsion.

Additional Studies

Imaging and electrodiagnostic studies supplement the physical examination, but do not replace it. Current evidence suggests that imaging and electrodiagnostic methods still lack precision in diagnosing nerve root conditions.[11–14] To date, the gold standard for the diagnosis of brachial plexus injuries is surgical exploration.[12,13]

Radiography: after ruling out associated injuries, request a chest radiograph to assess the elevation of the hemidiaphragm

Table 2 British Medical Research Council muscle strength score

Score	Strength	Description
0	No contraction	None
1	Contraction present, but no generation of movement	Trace
2	Partial movement with no gravity	No gravity
3	Complete range of motion against gravity	Against gravity
4	Complete range of motion against gravity + resistance	Almost normal
5	Normal strength	Normal

Table 3 Muscle groups assessed to establish the diagnosis, locate the brachial plexus injuries, and perform the follow-up

Movement	Peripheral nerve – muscle	Nerve roots
Shoulder abduction	Axillary nerve – deltoid muscles	C5, C6
	Suprascapular nerve – supraspinatus muscle	C5, C6
Shoulder: external rotation	Suprascapular nerve – infraspinatus muscle	C5, C6
	Axillary nerve – teres minor muscle	C5, C6
Shoulder: internal rotation	Subscapular nerve – subscapular muscle	C5, C6, C7
Elbow flexion	Musculocutaneous nerve – biceps, brachii muscles	C6
	Radial nerve – brachioradialis muscle	C6
Elbow extension	Radial nerve – triceps muscle	C7
Forearm supination	Musculocutaneous nerve – biceps muscle	C6
	Radial nerve – supinator muscle	C6
Forearm pronation	Median nerve – pronator teres muscle	C7
	Median nerve (anterior interosseous nerve) – pronator quadratus muscle	C8, T1
Wrist extension	Radial nerve – extensor carpi radialis longus muscle	C6
	Radial nerve – extensor carpi radialis brevis muscle	C7
Wrist flexion	Median nerve – flexor carpi radialis muscle	C7
	Ulnar nerve – flexor carpi ulnaris muscle	C8
Long finger flexion	Median and ulnar nerves – flexor digitorum profundus muscle	C8, T1
Thumb flexion	Median nerve – flexor pollicis longus muscle	C8, T1
Extension of the metacarpophalangeal joint of the fingers	Radial nerve – extensor digitorum communis muscle	C7
Extension of the interphalangeal joint of the fingers	Ulnar nerve – dorsal and palmar interosseous muscles	C8, T1
Fingers: abduction and adduction	Ulnar nerve – dorsal and palmar interosseous muscles (respectively)	C8, T1
Thumb: abduction and adduction	Median nerve – thumb abductor muscle	C8, T1
	Ulnar nerve – thumb adductor muscle	C8, T1

Table 4 Signs suggestive of preganglionic lesion

Rhomboid atrophy (dorsal scapular nerve)
Medial winged scapula (long thoracic nerve)
Horner syndrome (stellate ganglion injury)
Negative Hoffman-Tinel sign
Hemidiaphragm elevation (phrenic nerve injury)
Pseudomeningoceles in cervical spine computed tomography with myelography or magnetic resonance imaging
Sensorial loss with preserved sensory nerve action potential and conduction velocity

resulting from potential damage to the phrenic nerve and detect any rib fractures, which can influence the choice of nerves for transfer. In addition to ruling out an associated spinal injury, cervical spine radiographs may show a fracture of the transverse process, which is suggestive of root avulsion.

Computed tomography (CT) of the cervical spine with myelography: it is an invasive technique, with a contrast medium injected into the intrathecal space followed by a CT scan of the cervical spine. It reveals pseudomeningoceles suggestive of root avulsion, which develop within 3 to 4 weeks. Its sensitivity to detect pseudomeningoceles ranges from 79% to 86%, with a specificity ranging from 50% to 97%.[15,16] Bordalo-Rodrigues et al.[17] report a higher interobserver correlation in the root evaluation using CT with myelography compared with magnetic resonance imaging (MRI). However, a major limitation is its invasiveness and the potential toxicity of the contrast medium; in addition, patients with pseudomeningoceles may not present root avulsions, and avulsed roots may not result in pseudomeningoceles.[13,18]

Cervical spine MRI: a cervical spine MRI has the advantage of being a non-invasive method for root visualization; in addition, it enables the observation of pseudomeningoceles, adjacent soft tissues, and the postganglionic plexus. Its sensitivity to diagnose root avulsion ranges from 90% to 96%, with 75% to 95% of specificity.[12,13,19] Different studies reveal heterogeneous results when assessing postganglionic lesions. Acharya et al.[12] report 87% of sensitivity and 26% of specificity, whereas Leigheb et al.[11] report 90% of sensitivity and specificity. The major limitation of this technique is that it relies on the quality of the equipment and protocols used in order to obtain images with adequate definition. The studies that report[20] better sensitivity and specificity rates have been carried out using equipments with at least 1.5 Tesla.

Today, the study of choice for these lesions is cervical spine MRI.[11,19] Its main advantages over CT myelography include its non-invasive nature and the fact that it enables the visualization of adjacent soft structures and postganglionic lesions; in addition, recent studies[20] have shown its higher sensitivity and specificity.

Electrodiagnostic studies: electromyography and conduction velocity studies help in the diagnosis of preganglionic lesions. Moreover, they can detect subclinical recovery, identifying persistent reinnervation or denervation. It is important to be familiar with the components of this study for a correct interpretation of its findings and limitations. Ideally, an electrodiagnostic study must be performed 4 to 6 weeks after the injury, not before 2 to 3 weeks, to allow time for the Wallerian degeneration to end in patients with axonotmesis or neurotmesis.[14] This study can be repeated serially after a couple of months to assess reinnervation.

Electromyography (EMG): it analyzes the electrical activity of the muscle during activity and rest, and it can help distinguish between pre- and postganglionic lesions by exploring muscles innervated by motor branches at the level of the lesion that are difficult to examine (such as the serratus anterior and the rhomboid). A denervated muscle generates fibrillations and positive sharp waves (PSWs) at rest. Activity reduces or abolishes the motor unit potentials (MUPs). In cases with demyelination (neuropraxia) alone, a block in conduction occurs with no signs of denervation signs. The signs of reinnervation include polyphasic MUPs, which indicate disorganized muscle activity during early recovery.

Conduction velocity: it enables an assessment of both the conduction speed at the motor and sensory fibers, as well as the determination of the amplitude of the electrical signal. Sensory conduction velocity directly evaluates the sensory nerve action potential (SNAP), which is the most important part of the study to diagnose preganglionic lesions. In these injuries, the dorsal root ganglion is in continuity with the remaining part of the nerve, preventing its Wallerian degeneration and sustaining SNAPs despite the lack of a clinical correlation (absence of sensitivity in the area of the avulsed root).

Diagnosis

There are different classifications for plexus injuries. According to location, they are divided into supraclavicular (roots and trunks), retroclavicular (divisions), and infraclavicular (fascicles and terminal branches) injuries. A total of 90% of patients present supraclavicular injuries or a combination of supra- and infraclavicular injuries, while only 10% have infraclavicular lesions.[1]

The most common root or trunk injuries in the supraclavicular region include global plexus injuries (C5-T1), which affect 53% of the patients, followed by upper trunk injuries (C5-C6), which are observed in 39%, and lower trunk lesions, which are infrequent and affect 6% of the patients.[1]

In addition, after the physical examination and supplementary tests, the lesions can be classified as preganglionic or postganglionic. The C8 and T1 roots are most susceptible to preganglionic lesions due to the bone and soft-tissue arrangement in the area. Higher roots usually present postganglionic lesions.

Treatment

Timely diagnosis and early referral to a multidisciplinary team specialized in brachial plexus injuries are essential for a good prognosis. The early recognition of preganglionic and postganglionic lesions with surgical indication provides patients with a full range of available surgeries.[9,21] On the other hand, the conservative treatment requires an adequate rehabilitation scheme, careful follow-up, including standardized physical exams to determine if the patient would benefit from surgery.

Whether and when to perform the surgical treatment is a difficult decision, and it must be supported by the following: history, physical examination, imaging, electrodiagnosis, and patient evolution. Surgery is indicated in open brachial plexus injuries, especially those resulting from sharp objects, those with high suspicion of root avulsion, and postganglionic lesions with no clinical or electrodiagnostic recovery within six months.[10,21,22]

Conservative Treatment

The percentage of patients with spontaneous recovery from traumatic brachial plexus injuries is not well reported.[20] Serial follow-up is indicated for patients with partial injuries, which present preserved, although reduced, motor and sensory activity, as well as in cases of closed low-energy lesions, and in patients who present improvement in the serial follow-up.

Lim et al.[21] evaluated the factors associated with the conservative treatment of brachial plexus injuries. In this study, 40% of incomplete injuries and 20% of complete injuries recovered without surgery, whereas 60% of infraclavicular injuries and only 29% of supraclavicular injuries did so. Although the authors[20] did not find an association between the potential for spontaneous recovery and the severity of the initial injury, they suggest that incomplete, closed, and infraclavicular injuries have a good prognosis.

The conservative treatment of brachial plexus injuries involves close follow-up with serial physical examinations, electrodiagnostic studies depending on the reinnervation rate, and rehabilitation until full function is restored.

Rehabilitation plays a fundamental role in maintaining passive joint mobility and, subsequently, in strengthening reinnervated s.

Surgical Treatment

The indication for early surgery is clear in open lesions and highly-suspicious preganglionic lesions. Immediate or early intervention (within two to six weeks) is indicated in open injuries (stab wounds, high-energy firearm injuries) and highly-suspicious preganglionic lesions.[9,23]

In closed injuries due to traction mechanisms, the time of surgery is controversial. Initially, expect spontaneous lesion recovery; balance the time for postoperative muscle self-reinnervation with the delayed atrophy of the motor endplate not to lose the therapeutic window. Global lesions warrant special consideration because of their poor prognosis and the long reinnervation time up to the last hand effector muscles, which are very distant. The absence of spontaneous recovery within six months usually leads to surgical exploration.[24,25] Late surgeries ($>$ 9 or 12 months) have worse outcomes.[1,9,26]

The treatment of a brachial plexus injuries is technically demanding, and the outcomes of nerve reconstructions will be apparent approximately 1 to 2 years after surgery. Informing the patient about the anticipated treatment outcomes and recovery times is critical in order not to create false expectations. Depending on the patient's age, preference, and occupation, it is possible to consider a secondary reconstruction with tendon transfers, osteotomies, and arthrodesis, with short-term inferior but predictable results.

Next, we discuss the different types of treatment available today, providing an overview of the alternatives for the patients.

Treatment Priorities

When planning the reconstruction of a brachial plexus injury, specific functions that require restoration to optimize limb function have been defined. The first reconstruction priority is elbow flexion.[9] The next priority is shoulder stability to enhance limb function and avoid pain due to shoulder subluxation. Restauration of shoulder abduction and external rotation is also a priority. Restoration of wrist and hand mobility is more difficult given the great distance from the injury to the motor endplate of the effectors. Hand and wrist function are the third priority, as proper hand function requires a stable, elbow-flexing limb. Last, we attempt to restore the protective sensitivity of the hand.

Types of Surgery

There are two groups of surgeries to treat these injuries. Primary brachial plexus reconstruction includes graft reconstructions and nerve transfers; they are time-dependent because their goal is to restore the function of denervated muscles through motor endplate reinnervation. These surgeries maintain muscular anatomy and biomechanics. Secondary procedures include tendon transfers, free muscle transfers, arthrodesis, and osteotomies to restore limb function. They do not depend on the time of the injury. Type of injury, time, nerves or muscles available for transfers, and the patient's preference determine the performed surgical procedure.

Today, depending on the type of injury, it is possible to combine these techniques for reconstruction. In patients with early diagnosis (within six months), it is consensual that nerve reconstructions are prioritized over secondary surgery. There is no consensus for the management of late-diagnosed injuries (after 12 months), and nerve reconstructions or secondary surgeries, such as free muscle transfers, are indicated.[27]

Primary Reconstruction

1. **Reconstruction using grafts**: this surgical technique reconstitutes the continuity of the affected nerves using a nerve graft. Only postganglionic injuries are suitable to this technique because there is a nerve root in continuity with the spinal cord. This surgery consists of neurolysis of the site of the injury, identifying the proximal and distal ends of the damaged nerves, trunks, or cords. A successful reconstruction requires scar tissue or neuroma resection in viable nerve tissue (bleeding and visible fascicles). Most patients still present a defect of variable size, which is solved with a nerve graft. Traditionally, this technique has been used for the reconstruction of brachial plexus injury. It has the advantage of recovering both motor and sensory functions. Its main disadvantage is the worse outcomes with increasing reinnervation distance and graft size, along with inferior outcomes compared with those of certain nerve transfers.[24,28–30] However, some authors[9] still recommend grafting viable roots and associating this surgery with distal nerve transfers.
2. **Nerve transfers**: they aim to use a healthy, redundant, or "non-critical" nerve to reinnervate a muscle with a critical functional affected by the original injury. The advantage of nerve transfers is that neurorrhaphy is distal to the lesion and closer to the effector muscle, reducing the time required for reinnervation. The outcomes of these surgeries are superior compared to those of graft reconstruction to restore elbow flexion.[24,28–30] They also play a fundamental role in preganglionic injuries, which present no root to graft, as well as in lower trunk injuries, and late surgeries. They can use intraplexal or extraplexal nerves, especially the following:
    a) **Intraplexal nerves**: ulnar nerve (flexor carpal muscle bundle), median nerve (flexor carpi radialis muscle bundle), radial nerve (triceps muscle bundle, supinator branch, extensor carpi radialis brevis bundle), pectoral nerve.
    b) **Extraplexal nerves**: accessory nerve, intercostal nerves, contralateral C7 nerve.

Common nerve transfers include the Oberlin transfer, in which an ulnar nerve bundle to the flexor carpi ulnar muscle reinnervates the biceps brachii muscle when transferred to the biceps branch arising from the musculocutaneous nerve.[31] Another usual transfer in upper trunk injuries is the Leechavengvongs transfer, in which one of the heads of the triceps muscle receives radial nerve branches for deltoid reinnervation.[32] Different combinations of nerve transfers restore functionality depending on the degree of the injury. As the severity of the brachial plexus injury increases, the number of donor nerves for transfer decreases along with the potential restoration of limb functionality. One of the main advantages of nerve transfers is that they do not require more physical therapy, unlike some tendon transfers that require intense rehabilitation for retraining and to prevent adhesions.

Secondary Reconstruction

1. Tendon transfers: they follow the same principle as those of nerve transfers, in which a healthy, redundant, or "non-critical" muscle is used to replace the function of the affected muscle. These transfers require muscles with normal strength, ideally functionally synergistic to the denervated muscle; in addition, this muscle must go in a straight line from its origin to the new attachment and cross a single joint. One of the most common tendon transfers is that of the pronator teres muscle to the extensor carpi radialis brevis (ECRB) for wrist extension. In this example, the pronator teres muscle acts in the same phase as the ECRB when extending the wrist to perform a movement that requires fist strength. The disadvantage of these surgeries compared to nerve transfers is the use of another muscle with a different attachment site and strength vector; as such, both muscle and tendon must have a proper excursion, and fixation must be performed under adequate tension. These procedures also require intensive rehabilitation for function retraining and to avoid postoperative adhesions. Their main advantage is that they can be performed at any time, since they do not depend on the vitality of the endplate for function. Therefore, they are usually performed in patients with late presentations.

2. Functional free muscle transfer: it involves transferring a healthy muscle from another body segment to the injured limb. Free muscle is reinserted proximally and distally to replace poor muscle function, followed by proper arterial and venous anastomosis. Subsequently, a donor nerve (such as the intercostal nerves or the spinal accessory nerve) is transferred to the grafted muscle. They are a good option in patients with chronic injuries outside the therapeutic window for a nerve transfer. The most widely-used free muscle transfer involves the gracilis muscle, with excellent outcomes in terms of restoration of elbow flexion in late upper trunk injuries.[27]

3. Arthrodesis and osteotomy: they are used to improve the posture of the limb in space and enhance its function. In case of a wrist extension deficit with no muscle available for a tendon transfer, for example, wrist arthrodesis positions the hand in slight extension, improving the strength generated by the digital flexor muscles.

The following are examples of procedures according to the level and type of the injury:

1) *Avulsion lesions of the upper trunk (C5-C6)*: the patients present flaccid shoulder, abduction deficit, external rotation, and loss of elbow flexion. In these injuries nerve transfers are ideally performed within six months to restore lost functions. The outcomes of nerve transfers are better than those of graft reconstruction in this group of patients.[28,30] The following nerve transfers are performed: transposition of the spinal accessory nerve to the suprascapular nerve (which innervates the supraspinatus and infraspinatus muscles), transposition of the branch to the long tricipital head (radial nerve) to the axillary nerve (deltoid muscle), and Oberlin transposition (biceps brachii muscle). This combination results in rates of elbow flexion $>$ or $=$ to M3 of 93.1% to 96% and 74% to 82.5% for shoulder abduction of $>$ or $=$ to M3.[24,28,30,33,34]

2) *Lower trunk injuries (C8-T1)*: the functional outcomes in the hand are significantly less successful compared with those in the shoulder and elbow, because hand recovery is more complex. The treatment aims to restore a functional grip and fist either with nerve or tendon transfers. Recent studies[35,36] have shown good outcomes after transfers of the branch to the brachialis (musculocutaneous nerve) to the anterior interosseous nerve and pronator teres branch or from the supinator to the posterior interosseous nerve if necessary.

3) *Global preganglionic plexus lesion*: these traumatic plexus lesions have the worst prognosis. The greatest challenge for their treatment is the limited availability of donor nerves or muscles to restore limb functionality. In these patients, the aforementioned treatment priorities are critical. First, the aim is to reestablish elbow flexion; then, shoulder stability, and, if possible, shoulder abduction and external rotation; and, finally, to reinnervate the hand, with restoration of grip strength. These patients present only extraplexal nerves for nerve transfers, so secondary reconstruction techniques such as arthrodesis and free gracilis flap are frequently required. The literature reports different treatment options. A common strategy is a spinal accessory nerve transfer to the suprascapular nerve,[37] transfer of intercostal nerves to the musculocutaneous nerve, and a free gracilis flap for hand function.[22,38]

Rehabilitation

Rehabilitation in brachial plexus injuries plays a fundamental role in different stages of the treatment. Initially, for patients with surgical or conservative management indications, it is critical to maintain passive ranges of motion of the affected joints to avoid stiffness until reinnervation occurs. On the other hand, some surgical strategies require limb mobilization and retraining of the restored function.[22]

Broadly speaking, the goals of rehabilitation in this group of patients include the following:

- To maintain joint mobility;
- To strengthen synergistic muscles;
- Motor retraining of muscles reinnervated by nerve transfers;

- Occupational therapy, retraining for activities of daily life, and eventually change of dominant limb laterality; and
- Management of denervation pain.

Functional recovery from brachial plexus reconstructions can take up to three years. Physical therapy lasts from 6 to 12 months, depending on the evolution and improvement in strength and range of motion.

Conclusion

Traumatic brachial plexus injuries are a complex entity that can significantly compromise function and quality of life. Currently, several surgical techniques have been demonstrated to successfully restore limb function. To do so, the different professionals caring for these patients must know these treatment options; in addition, a timely diagnosis and early referral to teams specialized in the treatment of brachial plexus injuries are critical.

Conflict of Interests

The authors have no conflict of interests to declare.

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