Artículo de revisión

Adult Brachial Plexus Injuries. Part 1: Anatomy, Exam and Evaluation

Lesiones del plexo braquial en adultos. Parte 1: Anatomía, examen físico y evaluación

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Resumen

Introducción. El plexo braquial es una red intrincada de nervios que proveen la inervación sensitiva y motora de la cintura escapular y la extremidad superior. Diferentes mecanismos pueden lesionar estos nervios en diferentes grados y localizaciones, y en los casos más severos las raíces se avulsionan de la médula espinal resultando en una pérdida global de la función y la sensibilidad de la extremidad superior.

Objetivos. Revisar la anatomía del plexo braquial y los mecanismos y patrones de lesión; proveer guías para una evaluación estructurada, y revisar el rol de los estudios diagnósticos.

Resultados. El conocimiento sobre la anatomía del plexo braquial es útil para identificar el sitio de la lesión y elegir el tratamiento más adecuado. La tracción cerrada es el mecanismo más común y la mayoría de lesiones son panplexales. El examen físico es fundamental para descartar situaciones que ponen en riesgo la vida, interpretar los estudios diagnósticos y tomar decisiones durante el tratamiento. El mielotac es el estándar de oro de las imágenes diagnósticas. El electrodiagnóstico es la manera más confiable de identificar la lesión nerviosa y documentar los signos más tempranos de reinervación. Aunque existe una gran variedad de opciones para restaurar la función, la elección depende de los nervios lesionados y de los hallazgos al examen físico.

Conclusión. El pronóstico de las lesiones del plexo braquial depende de un diagnóstico correcto y oportununo. Los procesos diagnósticos multifactoriales buscan delimitar las opciones para ayudar al cirujano a decidir el mejor curso de tratamiento y evitar pérdidas de tiempo.

Abstract

Introduction: The Brachial plexus is an intricated network of nerves that provide motor and sensory innervation of the shoulder girdle and the upper extremity. A variety of mechanisms may injure the nerves at different locations to a variable degree. Most severely the nerves are avulsed from spinal cord resulting in global loss of function and sensation of the upper extremity.

Purpose: This paper intends to review the anatomy of the Brachial Plexus, Mechanisms and Patterns of Injury, and to provide guidelines for a structured physical examination. Additionally, the role of diagnostic and supporting studies will be reviewed.

Results: Knowledge of the anatomy of BP assists in identifying location of injury as well as treatment options. Closed traction is the most common mechanism and most of BP injuries are panplexal at presentation. Physical examination is key in ruling out life threatening situations, diagnostic test interpretation and treatment decision making. CT Myelogram remains the gold standard in diagnostic imaging. Nerve electromyologic studies (EMG, NCS, SSEP/MEP) are the most reliable way of identifying nerve injury and document the earliest signs of recovery. While a variety of treatment options are available to restore function, the options depend on which nerves are injured and what the exam findings are.

Conclusion: Prognosis in Brachial plexus injury lies on correct and prompt diagnosis. The multifactorial diagnosis process intends to narrow the options helping the surgeon decide the best course of treatment.

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Anatomy

The Brachial plexus (BP) is an intricate network of nerves that provide motor and sensory innervation of the shoulder girdle and the upper extremity. It originates from the rootlets of the lower four cervical and first thoracic spinal nerves, which form the five roots of the Brachial Plexus. While C4 (prefixed) and T2 (postfixed) may contribute to the BP, this occurrence is not typical. The BP also has communicating branches to the sympathetic cervical chain and paravertebral ganglia nodes of the second and third sympathetic thoracic chains via the Kuntz nerves.¹

The brachial plexus has a well described pattern and can be topographically divided into four portions: interscalene, supracleavicular, retroclavicular and axillary portions. The brachial plexus courses and intertwines in a predictable pattern through these four regions. There are classically five anatomic sections of the brachial plexus: roots, trunks, divisions, cords and terminal branches² (Figure 1).

The five roots are named C5, C6, C7, C8 and T1, because of their anatomic origin. The spinal nerve within the cervical foramen has dorsal rootlets that coalesce into the dorsal root ganglion of the sensory nerves and ventral rootlet carrying the motor fascicles from the anterior horn.

The roots of the brachial plexus arise between the anterior and middle scalene muscles in the neck, and merge to form the upper (or superior), middle and lower trunks. The trunks are located in the posterior triangular space, C5 and C6 merge to form the upper trunk, C8 and T1 form the lower trunk, and C7 continues as the middle trunk. Each trunk gives an anterior and posterior division in the retroclavicular space and converge in the axilla, beneath the Pectoralis minor muscle. All the posterior divisions combine to form the Posterior Cord, the anterior division of the upper and middle trunks form the Lateral Cord and the Anterior Division of the lower trunk forms the Medial cord. The Cords are named according to their relationship with the Axillary Artery (Figure 2).

Figure 1. Brachial Plexus Anatomy.
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Figure 2. Retroclavicular Plexus and anatomic relations to Axillary vascular bundle.
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The cords further divide to form the terminal branches of the Brachial plexus. The Lateral Cord divides to form the Musculocutaneous nerve and the lateral cord contribution of the Median nerve. The Posterior cord terminates as the Axillary and Radial nerves and the Medial Cord forms the Ulnar nerve and the medial cord contribution to the Median Nerve. Medial Brachial cutaneous and medial antebrachial cutaneous nerves are two sensory terminal branches originating from the medial cord, providing sensation to the skin in the medial aspect of arm and forearm, respectively.

In general, C5 and C6 contribute to shoulder and elbow motion, through the axillary, suprascapular and musculocutaneous nerves. C7 contributes to elbow, wrist and finger extension (MP joint) through the radial nerve. Wrist extension is shared between C6 and C7 through the Radial Nerve. C8 takes care of finger flexion through Median and ulnar nerves and T1 motorizes the intrinsic muscles of the hand (Figure 1).

The Dorsal Scapular Nerve is the first collateral branch of the Brachial plexus, it originates directly from C5, and innervates the levator and rhomboid muscles. The Long thoracic nerve combines branches from C5, C6 and C7, innervates the serratus anterior muscle and is also known as Bell’s nerve. The suprascapular nerve arises from the upper trunk where C5 and C6 converge (Erb’s Point), it passes dorsally through the suprascapular notch below the transverse ligament and innervates the supraspinatus and infraspinatus muscles. The Lateral and Medial Pectoral nerves from the lateral and medial cords, innervate Pectoralis Major and Minor, respectively. At the infraclavicular level, arising from the posterior cord, two or three subscapular nerves arise, they innervate the Subscapularis and Teres major muscles. Between the subscapular nerves originates the thoracoacromial plexus. A high velocity impact is more likely to cause nerve disruption. The Nerves and surrounding tissues sustain a two-step injury, the crush that causes the bullet and a stretching injury secondary to the effect of temporary cavitation. This type of injury may involve arterial damage, acute or even delayed because of formation of pseudoaneurysms that may compress the brachial plexus with time. High velocity impacts are more likely to cause nerve disruption.

Compression Mechanism

This mechanism is less common and consists of a traumatic force against the shoulder on a cephalocaudal direction, compressing the infraclavicular plexus between the clavicle and first rib. Fractures of the coracoid process may compress the Musculocutaneous nerve and Lateral Cord. Humeral head and scapular neck fractures and shoulder dislocation can injure the axillary nerve and posterior cord, and fractures of the spine of the scapula may sever the Suprascapular nerve.

Gunshot wounds

The Nerves and surrounding tissues sustain a two-step injury, the crush that causes the bullet and a stretching injury secondary to the effect of temporary cavitation. This type of injury may involve arterial damage, acute or even delayed because of formation of pseudoaneurysms that may compress the brachial plexus with time. High velocity impacts are more likely to cause nerve disruption.

Penetrating Trauma

Although less common, sharp or blunt direct injury to the BP can be sustained, partial or complete disruption of the plexus is often encountered, as well as vascular and intrathoracic injuries. Immediate exploration is advised and there is generally a good prognosis of recovery.

Nerve Injury Classification

Injury location refers to the relationship of the site of injury to the Dorsal Root Ganglion, which contain the sensory cell body. Preganglionic injuries are root avulsions occurring proximal to the dorsal root ganglion, they are not amenable to repair with grafting and the-

Figure 3. A) Motorbike accidents are one of the main causes of traumatic Brachial Plexus injury; B) Combined avulsion, rupture and stretching of roots are shown.

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Roots are held together only by loose endoneurium making them more prone to avulsion, the anterior rootlets are especially more susceptible, because of their less compact and thinner constitution, and Dural sheath being thicker in the back. Upper roots (C5, C6, C7) have a stronger attachment at the entrance to the foramen making them more resistant to avulsion from the spinal cord.
referred require nerve transfers. Postganglionic, are located distal to the dorsal Root Ganglion and provide a feasible root donor to graft. They are not exclusive and are often found combined along the root levels\(^3\) (Figure 4).

**Figure 4.** Types and location of root injury. A) Pre and post ganglionic injuries are defined according to the relationship to the dorsal root ganglion; B, C and D) Roots may present with avulsion from the medulla, and stretch or complete rupture after foramen emergence. Usually different injury types may concur in one patient.

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Postganglionic nerve injury can be classified according to the type of damage to the nerve fiber. Seddon described three well-known groups of nerve injury: Neurapraxia, Axonotmesis and Neurotmesis. Sunderland, emphasized in further categorization of Axonotmesis, according to epineurium, perineurium and endoneurium integrity. Sunderland Grade 1 is equivalent to Seddon’s Neurapraxia and grade 5 is equivalent to Neurotmesis (Table 1).

| Class | Name          | Description                                           |
|-------|---------------|-------------------------------------------------------|
| I     | Neurapraxia   | Temporary nerve conduction interruption without loss of axonal continuity |
| II    | Axonotmesis   | Loss of axonal continuity with relative preservation of nerve connective tissue |
| III   | Neurotmesis   | Total severance of the nerve |

| Grade | Seddon counterpart | Description                                           |
|-------|--------------------|-------------------------------------------------------|
| I     | I                  | Same as Seddon Neurapraxia                           |
| II    | II                 | Seddon Axonotmesis, all connective tissue of the nerve remains intact. |
| III   | II                 | Loss of axonal continuity plus endoneurium disruption. Perineurium and Epineurium remain intact. |
| IV    | II                 | Loss of axonal continuity plus Endoneurium and Perineurium disruption. Epineurium remains intact. |
| V     | III                | Total disruption of the Nerve.                        |

Source: Own elaboration.

Mixed types of injury at different levels is the usual presentation in BP palsies and clinical utility of Seddon and Sunderland classifications is debated. Macroscopically we may find stretch or rupture in postganglionic type lesion.
Clinical patterns

Barnes described four patterns of BP injury in the 1940’s when conservative management was the rule. These injury patterns continue to be relevant.

C5-C6 injury

These injuries account for approximately 15% of all lesions, it is usually referred to as Erb-Duchene paralysis and shows absence of: shoulder abduction, elevation and external rotation. Elbow flexion and Forearm supination. Sensibility is affected in the skin of the lateral aspect of the shoulder and lateral aspect of thumb, middle and index finger. Level of injury may be at the roots or their coalescence at Erb’s point where they form the upper trunk.

C5-C7 injury

This pattern occurs in 20 to 35% of patients. In addition to C5-C6 deficits, weakness of elbow, wrist and finger extension occurs in variable degrees. Sensory disturbance is located additionally in the pulp of middle finger. It is known as Erb Plus Pattern.

C8-T1 injury

In 10% of Brachial Plexus Palsy, only C8 and T1 are injured. Patients show paralysis of hand intrinsics, wrist and finger flexors and variable weakness of hand extrinsics and finger extensors. Sensory loss is located in the medial aspect of arm and forearm, and palmar aspect of ulnar fingers. It is called Dejerine - Klumpke Palsy.

Pan Plexus injury

It is the most common pattern of injury, accounting for 50% to 75% of cases. These patients show a flail arm and insensate hand. Combination of pre and postganglionic injuries are occasionally present and during follow up, partial recovery may be seen.

Other types

The ramifications of combination of patterns of injury make description of all injury patterns nearly impossible. Occasionally, C5-8 is injured and T1 is preserved. Also known as the T1 Hand, it results in muscle paralysis of shoulder, elbow and wrist with preservation of finger and thumb extension and weak thumb and finger flexion.

Physical examination

Clinical exam must be thorough, systematic and periodical. It should be focused on establishing location and extent of the initial injury and also set a baseline to compare recovery to.

The accuracy of clinical findings alone in BP injuries, has been found to be around 65%, every muscle of the upper extremity must be examined and motor strength recorded, when and if possible. A global neurological exam of the upper and lower extremity is necessary to rule out spinal cord lesions and examination of potential donor nerves should be taken into account.

Clinical exam findings suggestive of preganglionic avulsion are evaluated: Horner syndrome manifested as pupil miosis, eyelid ptosis and anhidrosis (T1 avulsion), absence of Tinel sign, paraspinal muscle weakness and loss of dorsal neck sensation (spinal cervical root’s dorsal rami), winged scapula (C5-C7) and atrophy of parascapular muscles (C5).

Sensory examination is evaluated by light touch of the dermatomes of the roots and specific nerves.

Motor examination includes (Table 2):

- Observation of muscle atrophy.
- Measuring passive and active motion (shoulder, elbow, wrist, thumb and fingers) in degrees.
- Manual Motor Testing of affected muscles and possible donors using the British Medical Research Council (BMRC) grading system or one of its many variations by the same examiner over several periods of time.

Table 2. Brachial Plexus Record Tool, for assessing muscle and corresponding nerve / root examination. It is very helpful in identifying level of injury and recovery during follow-up, as well as possible donors for nerve transfers.

The grading system must be clearly understood by the examiner and all the muscles need to be tested independently in every visit. BMRC grading system while simple, tends to be misinterpreted. One variation of the grading that we have found useful is to have the convention that in order to be a grade III, motion has to be within the arc of passive motion against gravity and a higher grade cannot be obtained unless the criteria for the lower grade is met (Table 3).
Paralysis of some muscles suggest level of injury, i.e., Serratus anterior muscle paralysis in the setting of a total or upper trunk palsy, suggests root C5-C6 avulsion; diaphragmatic palsy associated to elbow and shoulder paralysis, suggests avulsion or injury close to the vertebral foramen of C4-C5 and C6.¹

Severe neuropathic pain is usually associated with avulsion injuries, secondary to spinal cord injury. Dry skin on an anesthetic limb suggests a postganglionic injury.¹

Ruling out compromise of the spinal cord is important; suspicion arises if lower limbs are affected. Brown-Sequard syndrome, shows spasticity and loss of tactile discrimination, vibration and position sense on the ipsilateral lower limb and loss of pain and temperature sensation on the contralateral limb.¹

In the emergency setting, vascular damage, fractures and associated life-threatening injuries should be addressed. Afterwards, BP deficit can be established. If there is a vascular injury that demands exploration, Brachial plexus may be explored at the same time. Physical examination regarding vascular integrity might be unimpressive at first, 5% to 15% of patients with vascular injury may have a normal pulse. Hard and soft signs of vascular injury must be evaluated, and if there are hard signs present there is a 90% probability that vascular injury is present.¹³

Scapulothoracic dissociation is a severe injury to the shoulder girdle that consists of a lateral displacement of the scapula, disruption of the upper extremity by either an acromioclavicular or sternoclavicular joint dislocation or clavicle fracture associated to brachial plexus and subclavian vessels rupture with uncompromised overlying skin. Limb threatening ischemia happens in 10% of cases and 41% of them concur with ipsilateral fractures of humerus, radius, ulna and hand.

Diagnosis is suspected with a chest radiograph, and suspicion must be followed by angiography tests.

**Imaging Studies**

**Chest Radiographs**

Paralysis of hemidiaphragm suggests injury of phrenic nerve, even though isolated elevation of hemidiaphragm on a chest radiograph is of little value, the condition is unlikely in the absence of elevation.¹⁴

A sniff test monitors diaphragms excursion under fluoroscopy while the patient breathes in and out. The affected side usually does not move or is paradoxically pulled up during inspiration. The diagnostic ability of a sniff test is good in unilateral diaphragm paralysis but suboptimal in bilateral diaphragm paralysis¹⁵,¹⁶ (Figure 5).

![Figure 5. Left dome diaphragm elevation secondary to Phrenic nerve paralysis. Unilateral diaphragmatic paralysis is defined as the right dome of the diaphragm sitting >2 cm higher than its left counterpart or the left hemidiaphragm sitting equally high or higher than its right counterpart. Source: Own elaboration.](image)

**CT Myelogram vs MRI**

Preoperative imaging contributes to evaluation of the level of the injury, traditionally myelogram and CT myelogram have been the mainstay of diagnosis, based on the formation of a deformity of the subarachnoid space or pseudomeningocele, three to four weeks after injury once the dura mater has healed. Studies have shown a significant amount of root avulsions with no sign of meningocele or pseudomeningocele formation¹⁷ (Figure 6).

![Figure 6. Image of a Pseudomeningocele in CTM Source: Own elaboration.](image)

Compared to intradural exploration of the cervical roots, accuracy of high-resolution CT myelogram is 85%, as opposed to 52% with 1.5-tesla Magnetic Resonance Imaging (MRI).
The disadvantages of CTM include its invasive nature, exposure of patients to high doses of ionizing radiation, potential for allergic reaction to contrast material and that it doesn’t provide information of lesions beyond the intervertebral foramina. In spite these disadvantages, it is still very useful in preoperative diagnosis and surgical planning.

Axial CT images are the standard in preoperative evaluation, they allow to establish presence or absence of rootlets and differentiation between ventral and dorsal rootlets, although as the spinal roots run obliquely, they may be difficult to follow. Coronal and coronal oblique views raises diagnostic accuracy of intact roots on CT myelogram from 90% to 98%, when compared to axial views. In 5 to 15% of cases, it is not possible to obtain good quality images, sometimes due to epidural injection of the contrast material.

Some authors believe CTM should be reserved to situations where MRI is not available, considering current quality in this type of imaging. Van der Linde et al, using CTM as gold standard, found sensitivity of 1.5T MRI in the detection of nerve root avulsion and pseudomeningocele was 82% and specificity 100%. Interobserver agreement for root avulsion was 81.25% and for pseudomeningocele was 87.5, with kappa index of 0.77 and 0.84, respectively.

**Recommendations for CTM: it should be taken at least one month after injury to allow for the dura matter to heal avoiding contrast to dye surrounding spaces and blood clots to dissolve, minimizing artifact formation consequently producing high quality images that lead to correct interpretation.**

**Role of Brachial Plexus MRI**

With advances in MRI sequences and 3Tesla MRI units, evaluation of cervical brachial plexus as well as supra and infraclavicular injuries, is becoming possible. Signs for preganglionic nerve injury include interruption of root continuity, meningeal cysts, thickening of nerve roots and dura matter, morphological abnormalities of root sleeve. Manifestation of postganglionic injuries are nerve thickening and elevation of MRI signals. Visualization of pseudo neuromas have also been described. Sensitivity has been reported between 82% and 91% in detecting nerve root integrity and pseudomeningocele. Specificity of 92-95% for nerve root injury and 100% for pseudomeningocele detection (Figure 7).

**Figure 7.** This patient with complete avulsion of C8 had both a CT myelogram and an MRI. MRI can demonstrate pseudomeningoceles just as well as CT myelogram. Complete absence of the ventral and dorsal rootlets is also demonstrated, compared to the intact rootlets on the right side (yellow arrows), but one can see that the rootlets are not as conspicuously seen on MRI compared to CT myelogram.

Source: Own elaboration.

However, it is important to select the correct imaging sequence to obtain clear images, new protocols according to the level of injury that even suppress fat tissue and blood vessels have been described. Using these sequences and high-resolution MRI units, overall accuracy is 89.5% for preganglionic injury detection and 85.7% for postganglionic injuries.

**Recommendation:** While MRI may surpass the role of CTM in the future, at the present time, CTM remains the gold standard. Metal implant artifact, soft tissue edema and concomitant injuries with metal implants, often make MRI evaluation not possible.

**Electrodiagnostic Studies**

The studies that play a role in Brachial Plexus injury are Sensory and motor conductions, needle electromyography, somatosensory and motor evoked potentials (SSEP and MEP) and Nerve Action Potentials (NAP). These studies help confirm diagnosis, assess severity and location of the injury, and they provide information about reinnervation during follow-up.

Sensory Nerve Conduction or Sensory Nerve action potentials (SNAPs) differentiates between pre and postganglionic lesion, as
their normal in almost all preganglionic injuries, because the sensitive cell body lies within the dorsal ganglion and maintains continuity with the avulsed root. SNAPs start to lose amplitude after 7 to 11 days after injury in postganglionic lesions, hence the study should not be done earlier than this time frame because a postganglionic injury could be taken as preganglionic.22

Motor Nerve Conduction or Compound Motor Nerve action potentials (CMAPs) drop between day 3 and 7, therefore a well preserved CMAP after the first week in an affected muscle, suggests neuropraxia. It also assesses severity of the lesion, as it indicates proportion of axon loss when compared to the contralateral limb.23

Needle electromyography (EMG) is the most reliable way of identifying nerve injury.21 it appraises abnormal spontaneous activity, analyses motor unit behavior and recruitment patterns. EMG confirms axon injury when fibrillations potentials are present. Three weeks and document the earliest signs of recovery if unstable polyphasic units are seen. When voluntary muscle contraction is examined, if motor units are seen, it means there are surviving axons and reinnervation by collateral sprouting may be expected.24

Electrodiagnostic Studies also have limitations, the initial study cannot differentiate between a Sunderland 4 and 5 injuries, and it is imperative that the axon gets to the muscle to see reinnervation signs.

Recommendation: Baseline EDX studies should be ordered after 3 to 4 weeks so Wallerian degeneration has occurred.22 Frequent follow-up studies are not recommended because they tend to discourage the patient. True axonal reinnervation takes 3 to 4 months to show on EMG, depending on the length of the nerve injured. Thus, interval studies every 6–8 months to follow reinnervation is recommended.

Intraoperative Studies

SSEP and MEP help to determine whether there is continuity between the spinal cord and the cervical roots. An electrode placed on the scalp over the parietal cortex is placed, and it can act as a stimulator or a receptor.7

MEPs are motor compound action potentials elicited by noninvasive stimulation of the motor cortex through the scalp, and are used to evaluate roots that are being considered for grafting.26 SSEPs are very low amplitude potentials recorded on the scalp after stimulating a contralateral peripheral nerve, they allow for evaluation of functional continuity of sensory fibers through the dorsal root to the spinal cord.26 They can be altered by the depth of anesthesia. SSEPs and MEPs require a well-trained team.7

Nerve Action Potentials (NAPs) are useful in evaluating neuro-mas-in-continuity, if a good NAP is present across it, there is a high chance of self-recovery, so neurolysis is indicated. In the opposite scenario, neurona should be resected and grafted.27

Vascular Studies

In a cohort of 36 patients over a 10-year period,24 around 40% of all admitted Brachial Plexus Trauma patients, had an associated vascular injury. Within this group the most common mechanism was penetrating or gunshot injury, 22% of patients had a blunt trauma.

Shoulder dislocations that concur with BP injury, has been associated with damage of axillary artery in 27 to 44% of cases.29 Cases of BP compression due to an axillary artery pseudoaneurysm or expanding hematomas have been described as well.30–32

Arteriography is considered the Gold Standard with a published sensitivity of 95% to 100% and a specificity around 90%, with an overall accuracy of 92%,33 and it is diagnostic as well as therapeutic. However, has well known limitations, being invasive and time consuming, and it has been associated to complications such as contrast nephropathy, allergic reaction and local vessel injury.

Other alternatives include Doppler, which is inexpensive with an overall accuracy of 98% for arterial occlusion diagnosis,34 it is operator dependent and the presence of open wounds, large hematomas and fractures turn its application difficult. CT angiogram with a sensitivity of 95.1% and a specificity of 98.7%, is available in emergency departments and evaluates multiple body segments.35 Magnetic Resonance Angiogram, provides noninvasive imaging without ionizing radiation and contrast agent, however monitoring of neurologically impaired patients inside the magnet is difficult.36

Recommendation: Currently, CTA is the imaging test of choice for trauma patients with suspected vascular injuries. If there is a life-threatening situation associated to hard signs of vascular injury, imaging can be left out and immediate surgical intervention may proceed.

Conclusion

Brachial Plexus injury is a catastrophic condition which leads to severe impairment of work related, recreational and daily living activities, with economic, psychosocial and emotional consequences for the patients, their family and society. Although complete recovery is not possible, correct and prompt diagnosis lead to higher probabilities of better results. Final outcomes rely not only on initial severity but also in location of the injury, time elapsed and concomitant lesions.

Diagnosis is based on multiple factors, including mechanism of injury, findings on physical examination, imaging and nerve conduction studies. Even though it is not possible to clearly identify avulsion and nerve ruptures from neuropraxia in the acute setting and observation is still fundamental, the multifactorial diagnosis process intends to narrow the options helping the surgeon decide the best course of treatment, avoiding time loss.

Conflicts of interest

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