Adult Brachial Plexus Injuries: Mechanism, Patterns of Injury, and Physical Diagnosis

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Brachial plexus lesions frequently lead to significant physical disability, psychologic distress, and socioeconomic hardship. Adult brachial plexus injuries can be caused by various mechanisms, including penetrating injuries, falls, and motor vehicle trauma. Often the diagnosis is delayed or ignored as the practitioner waits for some recovery. Expedient diagnosis and testing is the best means of maximizing functional return. Evaluators must remember that muscles will begin to undergo atrophy and lose motor end plates as soon as the proximal injury occurs. Thus, early surgical intervention is the best predictor of a successful outcome.

Incidence and cause

The number of brachial plexus injuries that occur each year is difficult to ascertain; however, with the advent of more extreme sporting activities and more powerful motor sports, and the increasing number of survivors of high-speed motor vehicle accidents, the number of plexus injuries continues to rise in many centers throughout the world [1–6]. Most patients with brachial plexus injuries are men and boys aged between 15 and 25 years [5,7–9]. Based on his nearly two decades of work with more than 1000 patients with brachial plexus injuries, Narakas [10] stated that 70% of traumatic brachial plexus injuries occur secondary to motor vehicle accidents and, of these, 70% involve motorcycles or bicycles. In addition, 70% of cycle riders will have other major injuries.

Classification of nerve injury

To understand the requirements for surgery, it is first necessary to review the patterns of nerve injury. Historically, nerve injuries have been described using the Seddon and Sunderland classification scheme. Seddon’s [11] original classification scheme described three possibilities for a dysfunctional nerve: neuropraxia, axonotmesis, or neurotmesis. A neuropraxia is present when there is a conduction block at the site of injury, but no macroscopic injury to the nerve. There may be a demyelinating injury, but Wallerian degeneration does not take place distal to the zone of injury. As soon as the block is resolved, nerve function to the target organ will normalize. The recovery time may extend from hours to months, depending on the extent and severity of the injury to the myelin covering. Physical examination will not show a Tinel’s sign. Electrodiagnostic studies will show no conduction across the area of injury but will show normal conduction distal to the area of injury; this finding is unique to neuropraxias [12]. In axonotmesis, the axon or nerve fibers are ruptured, but the epineurium and perineurium remain intact. Wallerian degeneration will occur distal to the injury, but regeneration in the surviving proximal stump is still possible and should occur at a rate of 1 to 4 mm per day.
In neurotmesis, the entire nerve trunk is ruptured and axonal continuity cannot be restored. Without surgical intervention, this injury pattern will heal as a nonfunctional neuroma.

Sunderland expanded Seddon’s classification scheme into five categories after observing that some patients with axonotmeses recovered, whereas others did not [12]. These new categories were added to better describe the condition of the endoneurium; if the endoneurium remains intact, the nerve has the potential to regenerate to its target organ.

Sunderland’s first-degree injuries are the same as Seddon’s neuropraxia. A second-degree injury involves a rupture of the axon, but the basal lamina or endoneurium remains intact, which allows for the possibility of recovery following Wallerian degeneration. A Tinel’s sign will be noted on examination. The examiner should begin percussion distal to the injury site and note the most distal extent of the Tinel’s sign. This distal Tinel’s sign marks the site of the regenerating nerve cone. The site of the original trauma may show percussive sensitivity for several months, which should not be mistaken for advancing nerve fibers or a nonfunctional neuroma without first testing distally. Recovery from second-degree injuries should be complete unless the injury is so proximal to the target organ that muscle atrophy or motor end-plate degeneration occurs within the period of nerve regrowth. Third-degree lesions involve injury to the endoneurium with preservation of the perineurium. With disruption of the endoneurium, scarring will occur and full recovery is unlikely. Fourth-degree injury involves rupture of the fasciculi with disruption of the perineurium. The nerve is in continuity but scarring will likely prevent regeneration. A Tinel’s sign will be present at the site of injury but will not advance. A fifth-degree injury is defined as complete transection of the nerve and epineurium. Findings are similar to a fourth-degree injury. There will be no advancement of the Tinel’s sign and there will be no evidence of reinnervation with electrodiagnostic testing.

Mackinnon [12] has popularized an additional “sixth-degree” injury pattern, which represents a mixed pattern of nerve injury encompassing all degrees of injury: neuropraxia, axonotmesis, and neurotmesis. Some fascicles within the zone of injury may recover, whereas others will not. This finding complicates the diagnosis. This injury pattern is often seen in cases of traction. Traction forces on the nerve initially lengthen the epineurium and perineurium. The fasciculi are then stretched, reducing the nerves’ cross-sectional area, which raises intrafascicular pressure. Before axonal rupture, the epineurium and perineurium rupture and strip off the nerve. Axons tend to rupture over several centimeters, with the larger fascicles breaking first [13]. Long areas of fibrosis and scarring are produced, which inhibit nerve regeneration. The successful repair of these types of injuries often involves intraoperative nerve stimulation to identify which portions of the nerve are capable of spontaneous recovery and those portions of the nerve which require resection, grafting, or neurotization. In these cases, definitive surgery is often delayed up to 3 months until the physician can determine the potential for nerve recovery.

Mechanism and pathoanatomy

Most adult brachial plexus pathology is caused by closed trauma. Nerve injury in these cases is from traction and compression, with traction accounting for 95% of injuries [14]. Following a traction injury, the nerves may rupture, be avulsed at the level of the spinal cord, or be significantly stretched but remain intact (Fig. 1). Following are five possible levels where the nerve can be injured (Fig. 2):

1. The root
2. The anterior branches of the spinal nerves
3. The trunk
4. The cord
5. The peripheral nerve

Root injuries may be further localized with respect to the dorsal root ganglion (DRG). Postganglionic (infraganglionic) injuries are located distal to the DRG, whereas preganglionic (supraganglionic) lesions are located proximal to the DRG. With both types of lesions, patients present with loss of muscle function. In preganglionic injuries, the nerve has been avulsed from the spinal cord separating the motor nerve fibers from the motor cell bodies in the anterior horn cells. The sensory fibers and cell bodies are still connected at the DRG; however, the efferent fibers entering the dorsal spinal column have been disrupted. Thus, sensory nerve action potentials (SNAPs) are preserved in patients with supraganglionic injuries. In postganglionic injuries, both the motor and sensory nerve cells have been disrupted so there will be abnormalities in both motor action potentials and SNAPs (see Fig. 1) [15]. At present, the repair of preganglionic...
injuries requires a neurotization procedure. Post-ganglionic injuries may be amenable to surgical repair or grafting.

Root avulsions

Root avulsions are present in 75% of cases of supraclavicular lesions; multiple root avulsions have become more frequent over the past 25 years. There are two mechanisms for avulsion injuries: peripheral and central. Peripheral avulsion injuries are more common, whereas central avulsion injuries are rare and usually the result of direct cervical trauma (Figs. 3 and 4).

The peripheral mechanism occurs when traction forces on the arm overcome the fibrous...
Fig. 2. Anatomy of the brachial plexus. (A) The brachial plexus has five major segments: roots, trunks, divisions, cords, and branches. The clavicle overlies the divisions. The roots and trunks are considered the supraclavicular plexus, whereas the cords and branches are considered the infraclavicular plexus. (B) The relationship between the axillary artery and the cords is shown. The cords are named for their anatomic relationship to the axillary artery: medial, lateral, and posterior. LC, lateral cord; LSS, lower sub scapular nerve; MABC, medial antibrachial cutaneous nerve; MBC, medial brachial cutaneous nerve; MC, medial cord; PC, posterior cord; TD, thoracodorsal; USS, upper sub scapular nerve. (Courtesy of the Mayo Foundation, Rochester, MN; with permission.)
supports around the rootlets. Anterior roots may be avulsed with or without the posterior rootlets. The epidural sac may tear without complete avulsion of the rootlets. Different injury types account for the different patterns seen on myelography. The epidural cone moves into the foraminal canal with peripheral avulsions (Fig. 5). Nagano et al [16,17] have classified avulsion and partial avulsion patterns based on findings at the time of myelogram. The nerve roots of C5 and C6 have strong fascial attachments at the spine and are less commonly avulsed in comparison to the nerve roots of C7 through T1.

The central mechanism of root avulsion is the result of the spinal cord moving longitudinally or transversely following significant cervical trauma. Spinal bending within the medullary canal induces avulsion of the rootlets [18]. The root remains fixed in the foramen and the epidural sleeve is not ruptured (see Fig. 4).

Injury patterns

Any combination of avulsion, rupture, or stretch may occur following a brachial plexus injury; however, certain patterns are more
Brachial plexus lesions most frequently affect the supraclavicular region rather than the retroclavicular or infraclavicular levels. The roots and trunks are more commonly injured in comparison to the divisions, cords, or terminal branches. Double-level injuries can occur and should be included in the differential. In the supraclavicular region, traction injuries occur when the head and neck are violently moved away from the ipsilateral shoulder, often resulting in an injury to the C5, C6 roots or upper trunk (Fig. 6). Traction to the brachial plexus can also occur with violent arm motion. When the arm is abducted over the head with significant force, traction will occur within the lower elements of the brachial plexus (C8–T1 roots or lower trunk; Fig. 7).

Distal infraclavicular lesions are usually caused by violent injury to the shoulder girdle. These lesions can be associated with axillary arterial rupture. Biomechanically for a cord to rupture, it must be firmly fixed at both ends. The two major mechanisms for rupture are the anterior medial dislocation of the glenohumeral joint and traction of the upper arm with forced abduction [13]. The nerves become injured between two points at which the nerve is either fixed, restrained by surrounding structures, or where it changes direction. Suprascapular, axillary, and musculocutaneous nerves are susceptible to rupture because they are tethered within the glenohumeral area at the scapular notch and coracobrachialis. Physicians must always consider the possibility of a double crush at the scapular notch and the musculocutaneous nerve at the coracobrachialis. Rupture of the ulnar nerve at the level of the humerus or elbow and median nerve rupture at the level of the elbow are also possible.

Approximately 70% to 75% of injuries are found in the supraclavicular region. Approximately 75% of these injuries involve an injury to the entire plexus (C5–T1); in addition, 20% to 25% of injuries involve damage to the nerve roots of C5 through C7 and 2% to 35% of injuries have isolated supraclavicular injury patterns to C8 and T1. Panplexal injuries usually involve a C5-C6 rupture with a C7-T1 root avulsion. The remaining 25% of plexus injuries are infraclavicular.

Other mechanisms

Though less common than closed injuries, open injuries do occur. If the mechanism results in sharp division (eg, by means of a knife), direct repair may be possible. Iatrogenic injuries have been reported from multiple surgical procedures, including mastectomy, first rib resection, and subclavian carotid bypass [19–21]. Emergency exploration for open trauma is usually only warranted in cases of vascular injury or sharp laceration. Lower truck injuries are more likely to have concomitant vascular injuries. Open injuries caused by gunshot wounds are best managed conservatively because the nerve in these injuries is rarely transected [9].
Physical examination

A patient with a brachial plexus injury is often seen in conjunction with significant trauma. This additional trauma can potentially delay diagnosis of any existing nerve injury until the patient is stabilized and resuscitated. A high index of suspicion for a brachial plexus injury should be maintained when examining a patient in the emergency department who has a significant shoulder girdle injury, first rib injuries, or axillary arterial injuries. Often, the patient is obtunded or sedated in the emergency setting and careful observation therefore is necessary as the patient becomes more coherent.

A detailed examination of the brachial plexus and its terminal branches can be performed in a few minutes on an awake and cooperative patient. The median, ulnar, and radial nerves can be evaluated by examining finger and wrist motion. Further up the arm, elbow flexion and extension can be examined to determine musculocutaneous and high radial nerve function. An examination of shoulder abduction can determine the function of the axillary nerve, a branch of the posterior cord. An injury to the posterior cord may affect deltoid function and the muscles innervated by the radial nerve. Thus, examination of wrist extension, elbow extension, and shoulder abduction can help determine the condition of the posterior cord.

The latissimus dorsi is innervated by the thoracodorsal nerve, which is also a branch of the posterior cord. This area can be palpated in the posterior axillary fold and can be felt to contract when a patient is asked to cough, or to press his or her hands against his or her hip. The pectoralis major is innervated by the medial and lateral pectoral nerves, each a branch, respectively, of the medial and lateral cords. The lateral pectoral nerve innervates the clavicular head and the medial pectoral nerve innervates the sternal head of the pectoralis major. The entire pectoralis major can be palpated from superior to inferior as the patient adducts his or her arm against resistance.

Proximal to the cord level, the suprascapular nerve is a terminal branch at the trunk level. This area can be examined by assessing shoulder external rotation and elevation. Often in a chronic situation, the posterior aspect of the shoulder will show significant atrophy in the area of the infraspinatus muscle. Supraspinatus muscle atrophy is harder to detect clinically, because the trapezius muscle covers most of the muscle. The loss of shoulder flexion, rotation, and abduction can also

Fig. 6. Upper brachial plexus injuries occur when the head and neck are violently moved away from the ipsilateral shoulder. The shoulder is forced downward whereas the head is forced to the opposite side. The result is a stretch, avulsion, or rupture of the upper roots (C5, C6, C7), with preservation of the lower roots (C8, T1). (Courtesy of the Mayo Foundation, Rochester, MN; with permission.)
be from a significant rotator cuff injury or deltoid injury. Axillary nerve function and rotator cuff integrity should be evaluated when testing shoulder function in addition to suprascapular nerve function.

Certain findings suggest preganglionic injury on clinical examination. For example, an injury to the long thoracic nerve or dorsal scapular nerve suggests a higher (more proximal) level of injury because both nerves originate at the root level (see Fig. 2). The long thoracic nerve is formed from the roots of C5-C7 and innervates the serratus anterior. The length of the nerve is more than 20 cm, and it is vulnerable to injury as it descends along the chest wall. Injury to this nerve with resultant serratus anterior dysfunction results in significant scapular winging as the patient attempts to forward elevate the arm. The dorsal scapular nerve is derived from C4-C5 and innervates the rhomboid muscles, often at a foraminal level. Careful examination will show atrophy of the rhomboids and parascapular muscles if this nerve is injured. The patient must be observed from the posterior to be able to fully evaluate the serratus anterior and rhomboid muscles.

Patients should also be examined for the presence of Horner’s syndrome (Fig. 8). The sympathetic ganglion for T1 lies in close proximity to the T1 root and provides sympathetic outflow to the head and neck. The avulsion of the T1 root results in interruption of the T1 sympathetic ganglion and in Horner’s syndrome, which consists of miosis (small pupil), enophthalmos (sinking of the orbit), ptosis (lid droop), and anhydrosis (dry eyes).

Motor testing must consider neighboring cranial nerves. For example, the spinal accessory nerve that innervates the trapezius muscle can occasionally be injured with neck or shoulder trauma that affects the brachial plexus. Its integrity is important because of the increasing use of the spinal accessory as a nerve transfer. Trapezial paralysis or partial paralysis results in a rotation of the scapula in addition to an inability to abduct the shoulder beyond 90°.

Careful sensory (or autonomic) examination should check various nerve distributions (especially autonomous zones). The sensation of root level dermatomes can be unreliable because of overlap from other nerves or anatomic variation.
Active and passive range of motion should be recorded. Reflexes should be assessed. The physician should ensure that there is no evidence of concomitant spinal cord injury by examining for lower limb strength, sensory levels, increased reflexes, or pathologic reflexes. Percussing the nerve is especially helpful. Acutely, pain over a nerve suggests a rupture. An avulsion may be present when there is no percussion tenderness over the brachial plexus. An advancing Tinel’s sign suggests a recovering lesion (as described previously).

A vascular examination should also be performed. This examination should include feeling distal pulses, feeling for thrills, or listening for bruits. It is possible to rupture the axillary artery in a significant brachial plexus injury. Vascular injuries are not an infrequent finding with infraclavicular lesions or with even more severe injuries, such as scapulothoracic dissociations, and should be evaluated and managed by a vascular surgeon either before or concomitantly with surgical intervention to the brachial plexus. In the acute setting, major concomitant injuries are present in 60% of patients, with most of these injuries being long bone fractures, followed by head injuries, chest injuries, and spinal fractures [9].

Radiographic evaluation

After a traumatic injury to the neck or shoulder girdle region, radiographic evaluation can give clues to the existence of associated neurologic injury. Standard radiographs should include cervical spine views, shoulder views (anteroposterior, axillary views), and a chest X ray. The cervical spine films should be examined for any associated cervical fractures, which could put the spinal cord at risk. In addition, the existence of transverse process fractures of the cervical vertebrae might indicate root avulsion at the same level. A fracture of the clavicle may also be an indicator of trauma to the brachial plexus. A chest X ray may show rib fractures (first or second ribs), suggesting damage to the overlying brachial plexus. Careful review of chest radiographs may give information regarding old rib fractures, which may become important should intercostal nerves be considered for nerve transfers (because rib fractures often injure the associated intercostal nerves). In addition, if the phrenic nerve is injured, there will be associated paralysis and elevation of the hemidiaphragm. Arteriography may be indicated in cases where vascular injury is suspected. Magnetic resonance angiography also may be useful to confirm the patency of a previous vascular repair or reconstruction.

CT combined with myelography has been instrumental in helping to define the level of nerve root injury [16,22,23]. When there is an avulsion of a cervical root, the dural sheath heals with development of a pseudomeningocele. Immediately after injury, a blood clot is often in the area of the nerve root avulsion and can displace dye from the myelogram. Therefore, a CT/myelogram should be performed 3 to 4 weeks after injury to allow time for any blood clots to dissipate and for pseudomeningocele to fully form. If a pseudomeningocele is seen on CT/myelogram, a root avulsion is likely (see Fig. 5).

MRI has improved over the past several years and can be helpful in evaluating the patient with a suspected nerve root avulsion [24–26]. It has some advantages over CT/myelogram because it is noninvasive and can visualize much of the brachial plexus, whereas CT/myelography shows only nerve root injury. MRI can reveal large neuromas after trauma and associated inflammation or edema. MRI of the brachial plexus can be helpful in evaluating mass lesions in the spontaneous nontraumatic neuropathy affecting the brachial plexus or its terminal branches. In acute trauma, however, CT/myelography remains the gold standard of radiographic evaluation for nerve root avulsion. MRI continues to improve, however, and may someday eliminate the need for the more invasive myelography.

Electrodiagnostic studies

Electrodiagnostic studies are an integral component of preoperative and intraoperative decision making when used appropriately and
Electrodiagnostic studies can help confirm a diagnosis, localize lesions, define the severity of axon loss and the completeness of a lesion, eliminate other conditions from the differential diagnosis, and reveal subclinical recovery or unrecognized subclinical disorders. Electrodiagnostic studies therefore serve as an important adjunct to a thorough history, physical examination, and imaging studies, not as a substitute for them. When they are considered together, a decision can be made whether to proceed with operative intervention.

For closed injuries, baseline electromyography (EMG) and nerve conduction studies (NCSs) can best be performed 3 to 4 weeks after the injury because Wallerian degeneration will occur by this time. Serial electrodiagnostic studies can be performed in conjunction with a repeat physical examination every few months to document and quantify ongoing reinnervation or denervation.

EMG tests muscles at rest and with activity. Denervation changes (ie, fibrillation potentials) in different muscles can be seen in proximal muscles as early as 10 to 14 days after the injury and 3 to 6 weeks post injury in more distal muscles. Reduced motor unit potential (MUP) recruitment can be shown immediately after weakness from lower motor neuron injury occurs. The presence of active motor units with voluntary effort and few fibrillations at rest has a good prognosis compared with the absence of motor units and many fibrillations. EMG can help distinguish preganglionic from postganglionic lesions by needle examination of proximally innervated muscles that are innervated by root level motor branches (eg, cervical paraspinals, rhomboids, serratus anterior).

NCSs are also performed with the EMG. In post-traumatic brachial plexus lesions, the amplitudes of compound muscle action potentials (CMAPs) are generally low. SNAPs are important in localizing a lesion as preganglionic or postganglionic. SNAPs are preserved in lesions proximal to the DRG. Because the sensory nerve cell body is intact and within the DRG, NCSs will often show that the SNAP is normal and the motor conduction is absent, when clinically, the patient is insensate in the associated dermatome. SNAPs are absent in a postganglionic or combined pre- and postganglionic lesion. Because of overlapping sensory innervation, especially in the index finger, the physician needs to be cautious about the localization of the specific nerve with a preganglionic injury based on the SNAP alone.

There are obvious limitations to electrodiagnostic studies. The EMG/NCS is only as good as the experience of the physician in performing the study and interpreting the result. Certainly, EMG can show evidence of early recovery in muscles (ie, emergence of nascent potentials, a decreased number of fibrillation potentials, or the appearance or increased number of MUPs); these findings may predate clinically apparent recovery by weeks to months. EMG recovery does not always equate with clinically relevant recovery, however, either in terms of quality of regeneration or extent of recovery. It merely indicates that some unknown number of fibers has reached muscles and established motor end-plate connections. Conversely, EMG evidence of reinnervation may not be detected in complete lesions despite ongoing regeneration, when target end organs are further distal.

The current authors believe that intraoperative electrodiagnostic studies are a necessary part of brachial plexus surgery in helping guide decision making. They use a combination of intraoperative electrodiagnostic techniques to maximize the information gathered before making a surgical decision. They use nerve action potentials (NAPs) and somatosensory evoked potentials (SEPs) routinely, and occasionally use CMAPs. The use of NAPs allows a surgeon to test a nerve directly across a lesion, which allows the surgeon to detect reinnervation months before conventional EMG techniques and determine whether a lesion is neurapraxic (negative NAP) or axonotmetic (positive NAP). The presence of an NAP across a lesion indicates preserved axons or significant regeneration. Primate studies have suggested that the presence of an NAP indicates the viability of thousands of axons rather than hundreds as seen with other techniques [27]. The presence of an NAP bodes well for recovery after neurolysis alone, without the need for additional treatment (eg, neuroma resection and grafting). More than 90% of patients with a preserved NAP will gain clinically useful recovery. NAPs can indirectly help distinguish between pre- and postganglionic injury. A faster conduction velocity with large amplitude and short latency (with severe neurologic loss) indicates a preganglionic injury. A flat tracing suggests that adequate regeneration is not occurring; this finding would be consistent either with a postganglionic lesion that is reparable or a combined pre- and postganglionic lesion (irreparable). In the latter situation, sectioning the nerve back to an intraforaminal level would not reveal good fascicular structure [20].
Intraoperative SEPs are also used. The presence of an SEP suggests continuity between the peripheral nervous system and the central nervous system by means of a dorsal root. A positive response is determined by the integrity of few hundred intact fibers. The actual state of the ventral root is not tested directly by this technique; instead, it is inferred from the state of the sensory nerve rootlets, although there is not always perfect correlation between dorsal and ventral root avulsions. SEPs are absent in post-ganglionic or combined pre- and postganglionic lesions. Motor evoked potentials can assess the integrity of the motor pathway by means of the lesions. Motor evoked potentials can assess the extent of nerve injury to allow for expedient surgical intervention when necessary.

Summary

Most brachial plexus injuries involve the entire plexus. An injury to major cords or branches often contains a mixed injury pattern, with portions of the nerve being avulsed, ruptured, or stretched. An advancing Tinel’s sign implies the possibility of neurologic recovery; however, the surgeon should combine this physical finding with that of electrodagnostic studies to assess the extent of nerve injury to allow for expedient surgical intervention when necessary.

References


