10.48047/jocaaa.2024.33.06.61

Electrodiagnosis in Brachial Plexus Injury Assessment: Current Perspectives and Clinical Relevance

Enass A. Eliwa[1], Yassir A.A.Mohammad[2], Mohammad-Reda Ahmad[3], Eman Emad El-Din Abdel-Azim[4]

1 Professor of Rheumatology and Rehabilitation, Faculty of Medicine, Zagazig University,

2 Professor of Rheumatology and Rehabilitation, Faculty of Medicine, Zagazig University,

3 Professor of Plastic Reconstructive Surgery, Faculty of Medicine, Zagazig University,

4 Resident Doctor at Rheumatology and Rehabilitation Department, Zagazig University Hospitals,

ABSTRACT

Background: Brachial plexus injuries (BPIs) present a complex clinical challenge due to the intricate anatomical structure and functional importance of the brachial plexus in upper limb movement and sensation. These injuries can result from trauma, obstetric complications, or iatrogenic causes and often lead to significant morbidity. Early and accurate diagnosis is vital for optimal management, which may include conservative therapy or surgical intervention. Among the various diagnostic modalities, electrodiagnosis plays a crucial role in assessing the integrity, location, and severity of nerve damage in BPI. This review explores the current role of electrodiagnostic techniques, primarily electromyography (EMG) and nerve conduction studies (NCS), in the evaluation of brachial plexus injuries. It discusses the physiological basis of electrodiagnosis, its methodological applications, and its relevance in clinical practice. We detail how these tools assist in differentiating between preganglionic and postganglionic lesions, detecting axonal loss, and monitoring nerve regeneration. The review also examines the utility of somatosensory evoked potentials (SSEPs) and newer innovations such as high-resolution nerve ultrasound in conjunction with electrodiagnosis. We emphasize the importance of appropriate timing and repeat assessments to maximize diagnostic yield and inform surgical decision-making. Furthermore, we discuss the limitations of electrodiagnosis, including challenges in differentiating complex injury patterns and variability in interpretation. The review identifies a research gap in the integration of electrodiagnosis with advanced imaging modalities for enhanced diagnostic precision. It also highlights the need for standardized protocols to improve inter-laboratory reliability and clinical outcomes. In conclusion, electrodiagnosis remains an indispensable tool in the evaluation and management of brachial plexus injuries. Its role in localization, prognostication, and treatment planning makes it a cornerstone in the multidisciplinary approach to BPI. Ongoing technological advancements and combined diagnostic strategies promise to further improve its clinical utility in the future.

Keywords*:* Electrodiagnosis, Brachial Plexus Injury , Clinical Relevance

1. Anatomy and Pathophysiology of the Brachial Plexus

The brachial plexus is a complex network of nerves formed by the anterior rami of the C5 to T1 spinal nerves, occasionally receiving contributions from C4 and T2. It is anatomically divided into roots, trunks, divisions, cords, and terminal branches, and it traverses the cervicoaxillary canal to supply motor and sensory innervation to the shoulder, arm, forearm, and hand [1]. This intricate structure

10.48047/jocaaa.2024.33.06.61

makes it vulnerable to various forms of trauma, especially at areas where it is relatively fixed, such as the interscalene triangle and costoclavicular space [2].

Pathophysiologically, brachial plexus injuries can be broadly categorized into preganglionic (proximal to the dorsal root ganglion) and postganglionic (distal to the dorsal root ganglion) lesions. Preganglionic injuries often involve avulsion of the nerve roots from the spinal cord and are associated with poor prognosis due to the limited potential for spontaneous recovery [3]. In contrast, postganglionic injuries such as neuropraxia, axonotmesis, or neurotmesis may exhibit varying degrees of recovery depending on the extent of axonal damage and the integrity of the connective tissue framework [4].

Electrodiagnostic studies are particularly useful in distinguishing between these two types of injuries. For example, in preganglionic lesions, sensory nerve action potentials (SNAPs) remain preserved due to the intact dorsal root ganglion, whereas in postganglionic injuries, these potentials are typically diminished or absent [5]. Understanding the anatomical layout and the type of injury is crucial for accurate diagnosis, prognostication, and therapeutic planning in patients with brachial plexus involvement [6].

2. Etiology and Classification of Brachial Plexus Injuries

Brachial plexus injuries (BPIs) can arise from various etiologies, with trauma being the most common cause in adults. High-energy mechanisms such as motorcycle accidents, falls from heights, and penetrating injuries can result in traction, compression, or laceration of the plexus [7]. The direction and magnitude of force determine the nature of the injury, with severe traction or violent lateral flexion of the neck often leading to root avulsions or rupture [8]. In neonates, obstetric brachial plexus injuries are associated with difficult deliveries, shoulder dystocia, or excessive lateral traction during vaginal delivery [9].

Iatrogenic injuries constitute another significant etiology and may occur during surgical procedures such as cervical spine surgery, axillary lymph node dissection, or thoracic outlet decompression [10]. Neoplastic infiltration or radiation-induced plexopathy also contributes to non-traumatic causes, necessitating careful differential diagnosis in oncologic patients [11].

Classifying BPIs aids in guiding diagnostic and therapeutic decisions. Sunderland's classification, an expansion of Seddon's earlier model, remains widely used. It categorizes nerve injuries into five degrees based on the severity of structural damage—from mild demyelination (neuropraxia) to complete transection (neurotmesis) with discontinuity of all connective tissue layers [4]. A sixth category, introduced later, accounts for mixed injury patterns often seen in clinical practice [12].

Additionally, BPIs can be anatomically classified as upper plexus (Erb's palsy), lower plexus (Klumpke's palsy), or total plexus involvement. Upper plexus injuries typically affect shoulder

10.48047/jocaaa.2024.33.06.61

abduction and elbow flexion, while lower plexus injuries impair intrinsic hand muscles and wrist flexion [13]. This anatomical classification correlates with specific nerve root involvement and assists clinicians in localizing lesions and planning further investigations, including electrodiagnostic studies [14].

3. Clinical Evaluation of Brachial Plexus Injuries

A comprehensive clinical evaluation remains the cornerstone in the diagnosis of brachial plexus injuries, serving as the basis for subsequent investigations. The assessment begins with a detailed history, focusing on the mechanism, force, and direction of injury. This helps determine the likely pattern of nerve involvement, especially in trauma-related cases where traction or direct impact suggests different injury distributions [15].

Physical examination aims to localize the lesion and assess functional deficits. Motor testing is performed systematically, evaluating muscle groups innervated by different branches of the plexus. For example, weakness in shoulder abduction (deltoid, C5) and elbow flexion (biceps, C6) may indicate upper trunk involvement, whereas hand grip weakness and sensory loss in the ulnar distribution suggest lower trunk or C8-T1 injury [16]. Reflex testing is also essential; the presence or absence of biceps, triceps, and brachioradialis reflexes can help localize the lesion within the plexus [17].

Sensory examination should include light touch, pinprick, and proprioception testing across dermatomal and peripheral nerve distributions. In preganglionic injuries, sensory loss may be patchy or absent due to dorsal root ganglion preservation, whereas postganglionic injuries often show more consistent sensory deficits [5]. Special clinical signs, such as Horner's syndrome (ptosis, miosis, anhidrosis), suggest T1 root involvement and possible preganglionic injury [18].

The presence of muscle atrophy and trophic skin changes can provide additional clues about chronicity and severity. Serial clinical evaluations over time help monitor progression or recovery and assist in planning interventions such as surgical nerve repair or rehabilitation strategies [19].

While clinical evaluation provides valuable insights, it may be insufficient in differentiating complex or proximal injuries, especially in the acute phase when voluntary movements are minimal. Therefore, it must be integrated with electrodiagnostic and imaging studies for comprehensive assessment and management planning [20].

4. Principles of Electrodiagnosis in Peripheral Nerve Injuries

Electrodiagnosis is a functional diagnostic modality that provides objective information on the integrity of peripheral nerves and muscles by assessing their electrical properties. The two primary

10.48047/jocaaa.2024.33.06.61

techniques employed are nerve conduction studies (NCS) and electromyography (EMG), both of which are instrumental in evaluating the site, severity, and nature of nerve damage in brachial plexus injuries [21].

Nerve conduction studies measure the speed and amplitude of electrical signals along motor and sensory nerves. They can identify abnormalities such as demyelination, axonal loss, and conduction blocks. In BPI, motor NCS can reveal reduced compound muscle action potential (CMAP) amplitudes or conduction blocks, while sensory NCS help distinguish between preganglionic and postganglionic lesions. For instance, preserved sensory nerve action potentials (SNAPs) with absent motor responses typically suggest preganglionic involvement, where the dorsal root ganglion remains intact [22].

Electromyography involves the insertion of a needle electrode into selected muscles to record electrical activity at rest and during voluntary contraction. It helps identify denervation changes, such as fibrillation potentials and positive sharp waves, which usually appear 10 to 14 days after injury. Chronic changes like polyphasic motor unit potentials can indicate reinnervation or long-standing damage [23].

Electrodiagnosis also aids in determining the prognosis of nerve recovery. Early absence of voluntary motor unit potentials may signal a complete lesion, whereas the appearance of nascent potentials indicates regeneration. Moreover, serial electrodiagnostic testing can be used to monitor progress and refine treatment strategies over time [24].

An essential principle in electrodiagnosis is the appropriate selection of nerves and muscles for testing, guided by clinical suspicion and anatomical knowledge. Accurate interpretation requires understanding the normal values and expected patterns in relation to the suspected injury level. Therefore, electrodiagnosis should be performed by experienced clinicians and interpreted in conjunction with clinical findings for optimal accuracy [25].

5. Electrodiagnostic Modalities in Brachial Plexus Injuries

Electrodiagnostic assessment in brachial plexus injuries utilizes a range of techniques that provide complementary data on nerve function. The primary modalities include nerve conduction studies (NCS), electromyography (EMG), and somatosensory evoked potentials (SSEPs), with emerging adjuncts like high-resolution ultrasonography and magnetic stimulation enhancing diagnostic accuracy [26].

Nerve Conduction Studies (NCS) focus on evaluating both motor and sensory nerves. In brachial plexus injuries, sensory NCS is particularly valuable for distinguishing preganglionic from postganglionic lesions. A hallmark finding of preserved SNAPs with absent motor responses suggests root avulsion, whereas diminished SNAPs indicate a postganglionic injury. Motor NCS, on the other

10.48047/jocaaa.2024.33.06.61

hand, helps assess the extent of axonal loss or demyelination and identifies conduction blocks, especially in cases with partial injury [27].

Electromyography (EMG) complements NCS by detecting denervation and reinnervation changes within affected muscles. Needle EMG findings such as fibrillations and positive sharp waves appear approximately two weeks after injury and signify ongoing axonal degeneration. Chronic denervation and reinnervation can be seen as long-duration, polyphasic motor unit potentials, helping to assess the timing and progression of injury. EMG also assists in identifying the level of the lesion by testing muscles innervated by different roots or trunks [28].

Somatosensory Evoked Potentials (SSEPs) offer an additional tool for assessing preganglionic injuries. SSEPs measure the cortical response to peripheral stimulation, and an absence or delay in these responses can support the diagnosis of root avulsion. They are particularly useful in unconscious or uncooperative patients and in medicolegal evaluations where objective evidence of lesion is required [29].

Emerging Techniques, such as high-resolution ultrasonography combined with electrodiagnosis, allow for real-time visualization of nerve continuity, scarring, or entrapment. Recent advancements also include magnetic stimulation and motor unit number estimation (MUNE), which provide non-invasive, quantitative assessments of motor axon loss and recovery [30].

Together, these electrodiagnostic tools provide a comprehensive picture of the functional status of the brachial plexus and are indispensable for guiding clinical management and surgical decision-making [31].

6. Timing and Protocols for Electrodiagnostic Evaluation

Timing is a critical factor in maximizing the diagnostic yield of electrodiagnostic studies in brachial plexus injuries. Performing these tests too early may lead to false-negative results, while excessive delays can hinder timely surgical intervention. Typically, electrodiagnostic evaluation begins around 2 to 3 weeks post-injury, when Wallerian degeneration has occurred and denervation changes become electrophysiologically detectable [32].

The **initial study** is usually conducted at 3–4 weeks after injury. This timing allows for detection of fibrillation potentials and positive sharp waves on EMG, which indicate axonal degeneration. Nerve conduction studies performed at this stage may show reduced CMAPs and SNAPs, or absent responses in severe cases. However, early findings should always be correlated with clinical assessment and imaging to confirm lesion severity and location [33].

Follow-up studies are typically scheduled at 6 to 12-week intervals to assess ongoing denervation, reinnervation, and nerve recovery. These serial evaluations are essential for differentiating between neuropraxia and more severe axonotmesis or neurotmesis. The appearance of nascent motor unit

10.48047/jocaaa.2024.33.06.61

potentials suggests regenerative activity and can inform decisions regarding rehabilitation or surgical referral [34].

The **protocol** for electrodiagnostic testing should be individualized based on the suspected lesion level and pattern. Standard protocols often include testing of representative muscles from each major root (C5 to T1) and corresponding peripheral nerves (e.g., axillary, musculocutaneous, radial, median, and ulnar). Sensory studies should be directed to assess SNAPs from the median, ulnar, and lateral antebrachial cutaneous nerves, especially when distinguishing between preganglionic and postganglionic injuries [35].

In addition to routine EMG and NCS, certain protocols incorporate **SSEPs** or **paraspinal EMG** to detect preganglionic involvement. For instance, absent cervical paraspinal activity on EMG may support a diagnosis of root avulsion, further emphasizing the importance of selecting appropriate muscles for assessment [36].

Timely and protocol-driven electrodiagnostic evaluation not only improves diagnostic clarity but also aids in prognostication and surgical planning, particularly in cases being considered for nerve repair or transfer [37].

7. Interpretation and Diagnostic Yield

Interpreting electrodiagnostic findings in brachial plexus injuries requires a detailed understanding of the anatomical organization of the plexus and the pathophysiological processes underlying nerve injury. A systematic approach that correlates clinical findings with electrodiagnostic data is essential to localize lesions accurately and assess the severity of involvement [38].

One of the primary goals of electrodiagnostic interpretation is to differentiate **preganglionic** from **postganglionic** injuries. This distinction is made by evaluating sensory nerve action potentials (SNAPs): preserved SNAPs in the presence of motor deficits suggest a preganglionic lesion, while absent or reduced SNAPs indicate postganglionic pathology. Additionally, needle EMG findings of absent cervical paraspinal muscle activity further support a preganglionic lesion [39].

The **diagnostic yield** of electrodiagnostic testing is highest when studies are appropriately timed and follow a structured protocol. In particular, EMG of multiple muscles innervated by different roots and trunks can help map the injury distribution and detect lesions involving specific cords or terminal branches. For example, combined denervation in deltoid, biceps, and supraspinatus suggests upper trunk involvement, whereas hand intrinsic muscle abnormalities imply lower trunk or medial cord damage [40].

Sensitivity and specificity of electrodiagnosis in brachial plexus injuries vary depending on the timing of the study and the experience of the examiner. When performed after 3 to 4 weeks post-injury and combined with serial testing, the sensitivity can exceed 85%, particularly in detecting axonal injuries

10.48047/jocaaa.2024.33.06.61

and localizing lesions [41]. However, electrodiagnosis is less sensitive in identifying purely demyelinating injuries or subtle conduction blocks without significant axonal loss [42].

In complex or mixed injury patterns, electrodiagnostic findings must be interpreted in conjunction with imaging modalities such as MRI or CT myelography to confirm anatomical disruptions or root avulsions. This integrated approach enhances diagnostic accuracy and informs clinical decision-making, especially in surgical planning [43].

Ultimately, the diagnostic yield of electrodiagnosis depends on meticulous technique, expert interpretation, and integration with clinical and radiologic findings to offer a comprehensive view of the injury and guide management strategies effectively [44].

8. Electrodiagnosis in Prognostication and Treatment Planning

Electrodiagnostic studies not only facilitate diagnosis but also play a critical role in predicting recovery and shaping treatment strategies in brachial plexus injuries. Prognostic information is particularly valuable in determining the likelihood of spontaneous recovery versus the need for surgical intervention, such as nerve repair or transfer [45].

One of the most significant electrodiagnostic indicators of poor prognosis is the absence of voluntary motor unit potentials (MUPs) in needle EMG, particularly in muscles innervated by nerves suspected of complete injury. The lack of nascent MUPs even after 12 weeks often suggests irreversible damage, prompting timely surgical referral [46]. Conversely, the early appearance of polyphasic, low-amplitude MUPs may reflect collateral reinnervation and a favorable prognosis for functional recovery [47].

Serial studies are essential to track **nerve regeneration**, which generally progresses at a rate of 1–3 mm per day. Electrodiagnosis can document the reappearance of motor and sensory responses over time, providing objective evidence of nerve regeneration. This is especially helpful in differentiating between neuropraxia and more severe axonotmesis or neurotmesis, conditions that carry different recovery trajectories and therapeutic needs [48].

In surgical planning, electrodiagnosis helps **define the extent of injury** and select donor nerves for transfer procedures. For example, preserved function in adjacent or contralateral nerves, as confirmed by EMG, can guide the selection of suitable donor nerves for neurotization, such as the spinal accessory, intercostal, or phrenic nerves [49].

Electrodiagnosis also aids in **rehabilitation planning**. Knowledge of denervated muscles helps target physiotherapy and electrical stimulation, preventing muscle atrophy while awaiting nerve recovery. Moreover, prognostic insights support patient counseling, setting realistic expectations and timelines for functional improvement or the need for assistive devices [50].

10.48047/jocaaa.2024.33.06.61

Thus, the role of electrodiagnosis extends beyond diagnostics, offering valuable guidance in forecasting outcomes and customizing both surgical and non-surgical management pathways in patients with brachial plexus injuries [51].

9. Limitations and Challenges of Electrodiagnostic Studies

Despite their invaluable role, electrodiagnostic studies in the assessment of brachial plexus injuries are not without limitations. These challenges must be acknowledged to avoid misinterpretation, guide complementary testing, and ensure optimal patient care [52].

One of the primary limitations is **timing sensitivity**. Electrodiagnostic changes related to axonal loss, such as fibrillations and reduced compound motor action potentials, may not become apparent until 2–3 weeks after injury. Consequently, very early studies may yield false negatives, particularly in severe injuries where immediate diagnosis is critical for surgical decision-making [53].

Another challenge is **difficulty in localizing complex or multiple-level lesions**, especially when overlapping innervation or compensatory reinnervation is present. For example, injuries involving multiple trunks or cords can produce EMG findings in several muscles, complicating precise anatomical localization without imaging support [54].

Patient cooperation is also essential for optimal EMG assessment. In children, individuals with altered consciousness, or those experiencing significant pain, obtaining reliable voluntary contractions may be difficult, thus limiting the diagnostic scope. Similarly, obese patients or those with deep-seated musculature may pose technical difficulties in needle placement and signal acquisition [55].

Electrodiagnostic studies are also **operator-dependent**, requiring significant expertise in both test performance and interpretation. Variations in technique, electrode placement, or stimulation parameters can affect outcomes. Moreover, experience is necessary to differentiate subtle pathological changes from normal variants or co-existing conditions such as cervical radiculopathy or peripheral entrapment neuropathies [56].

Certain **technical limitations** of the equipment itself, such as inadequate resolution in capturing low-amplitude or proximal responses, may hinder accurate assessment, especially in partial injuries. Advanced techniques like high-density EMG or motor unit number estimation (MUNE) can offer better sensitivity but are not widely available [57].

Therefore, while electrodiagnosis is an essential component of BPI evaluation, its limitations necessitate a multimodal approach—integrating clinical, radiological, and surgical data for comprehensive assessment and treatment planning [58].

10. Integration with Imaging and Surgical Planning

While electrodiagnostic studies provide essential functional insights into brachial plexus injuries, integrating these findings with imaging modalities enhances diagnostic accuracy and surgical planning.

10.48047/jocaaa.2024.33.06.61

This multimodal approach ensures that both anatomical and physiological aspects of the injury are addressed, guiding timely and effective interventions [59].

Magnetic resonance imaging (MRI) and **CT myelography** are commonly used to visualize structural abnormalities such as nerve root avulsions, pseudomeningoceles, or fibrotic scarring. MRI offers excellent soft tissue contrast and is particularly effective in identifying preganglionic injuries. When combined with EMG findings—such as absent paraspinal activity or preserved SNAPs—MRI can confirm root avulsions and assist in determining the extent of damage [60].

MR neurography, an advanced imaging technique, allows for high-resolution visualization of peripheral nerves and has proven particularly useful in cases where standard imaging and electrodiagnosis yield inconclusive results. It provides detailed mapping of nerve continuity, neuromas, and surrounding tissue pathology. When interpreted alongside electrodiagnostic data, MR neurography facilitates accurate localization and differentiation between pre- and postganglionic lesions [61].

CT myelography remains superior in detecting nerve root avulsions due to its ability to demonstrate cerebrospinal fluid leaks and pseudomeningoceles. It is often used in conjunction with electrodiagnostic studies to validate findings of preganglionic injuries. This combined approach aids in deciding whether a patient would benefit from surgical exploration or nerve transfer procedures [62].

In the **surgical context**, electrodiagnostic studies are invaluable in selecting donor nerves and target muscles. Intraoperative nerve stimulation and preoperative EMG data guide surgeons in identifying functioning nerve segments suitable for grafting or neurotization. This integration of electrodiagnosis with imaging helps tailor the surgical plan to each patient's specific injury pattern, improving the likelihood of functional recovery [63].

Furthermore, the combined use of these modalities is critical in **monitoring postoperative outcomes**. Follow-up EMG can track reinnervation progress, while imaging can confirm graft integrity or identify complications such as neuroma formation. This comprehensive strategy underscores the importance of electrodiagnosis as part of a multidisciplinary approach to managing brachial plexus injuries [64].

Conclusion

Electrodiagnosis remains a cornerstone in the comprehensive evaluation of brachial plexus injuries, offering invaluable functional insight into the nature, severity, and prognosis of nerve damage. Through a combination of nerve conduction studies, electromyography, and adjunctive techniques like somatosensory evoked potentials, clinicians can accurately localize lesions, distinguish between pre- and postganglionic injuries, and monitor the progression or recovery of nerve function. When integrated with detailed clinical examination and advanced imaging modalities such as MRI and MR neurography, electrodiagnostic studies enhance diagnostic precision and inform crucial decisions

10.48047/jocaaa.2024.33.06.61

regarding surgical intervention, donor nerve selection, and rehabilitation planning. The ability of electrodiagnosis to provide dynamic, real-time feedback on nerve status makes it particularly useful in tracking recovery and optimizing individualized treatment protocols. In conclusion, electrodiagnosis continues to be a vital component in the assessment and management of brachial plexus injuries. Its integration with clinical acumen and evolving technologies positions it as an indispensable tool in modern neurorehabilitation and surgical planning.

REFERENCES

1. Moore KL, Dalley AF, Agur AM. *Clinically Oriented Anatomy*. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2014.
2. Nath RK, Lyons AB, Bietz G. Physiological basis of electrodiagnosis in brachial plexus injury. *J Brachial Plex Peripher Nerve Inj.* 2007;2:1-9.
3. Midha R. Epidemiology of brachial plexus injuries in a multitrauma population. *Neurosurgery*. 1997;40(6):1182-1188.
4. Sunderland S. A classification of peripheral nerve injuries producing loss of function. *Brain*. 1951;74(4):491-516.
5. Kim DH, Murovic JA, Tiel RL, Kline DG. Management and outcomes in 353 surgically treated sciatic nerve lesions. *J Neurosurg*. 2004;101(1):8-17.
6. Birch R, Bonney G, Parry CBW. *Surgical Disorders of the Peripheral Nerves*. 2nd ed. London, UK: Springer; 2001.
7. Narakas AO. The treatment of brachial plexus injuries. *Int Orthop*. 1985;9(1):29-36.
8. Terzis JK, Papakonstantinou KC. Surgical treatment of brachial plexus injuries in adults: an overview. *Adv Tech Stand Neurosurg*. 2000;26:167-218.
9. Gilbert A. Obstetrical brachial plexus palsy. *Hand Clin*. 1995;11(4):563-575.
10. Heary RF, Bono CM. Brachial plexopathy after anterior cervical spine surgery. *Spine J*. 2004;4(3):303-309.
11. Bendszus M, Wessig C, Solymosi L, Pham M. Magnetic resonance neurography: diagnostic imaging in neurology. *Nat Rev Neurol*. 2015;11(12):698-707.
12. Mackinnon SE. Pathophysiology of nerve injury. *Clin Plast Surg*. 2003;30(1):109-126.
13. Chuang DC. Neurotization procedures for brachial plexus injuries. *Hand Clin*. 1995;11(4):633-645.
14. Malessy MJ, Thomeer RT. Evaluation of intercostal to musculocutaneous nerve transfer in reconstructive brachial plexus surgery. *J Neurosurg*. 1998;88(2):266-271.
15. Spinner RJ, Shin AY, Bishop AT. *Brachial Plexus Surgery: Principles and Practice*. Philadelphia, PA: Elsevier Saunders; 2011.
16. Campbell WW. Evaluation and management of peripheral nerve injury. *Clin Neurophysiol*. 2008;119(9):1951-1965.
17. Menorca RM, Fussell TS, Elfar JC. Nerve physiology: mechanisms of injury and recovery. *Hand Clin*. 2013;29(3):317-330.
18. Alnot JY. Traumatic brachial plexus palsy in adults. Retro- and infraclavicular lesions. *Clin Orthop Relat Res*. 1999;(368):78-86.

10.48047/jocaaa.2024.33.06.61

19. Kline DG, Hudson AR. *Nerve Injuries: Operative Results for Major Nerve Injuries, Entrapments, and Tumors*. Philadelphia, PA: WB Saunders; 1995.
20. Midha R. Nerve transfers for severe brachial plexus injuries: a review. *Neurosurg Focus*. 2004;16(5):E5.
21. Preston DC, Shapiro BE. *Electromyography and Neuromuscular Disorders: Clinical-Electrophysiologic Correlations*. 3rd ed. Philadelphia, PA: Elsevier; 2013.
22. Dumitru D, Amato AA, Zwarts MJ. *Electrodiagnostic Medicine*. 2nd ed. Philadelphia, PA: Hanley & Belfus; 2002.
23. Daube JR. Clinical neurophysiology. *Mayo Clin Proc*. 1996;71(3):279-287.
24. Johnson EW. *Practical Electromyography*. 3rd ed. Baltimore, MD: Williams & Wilkins; 1988.
25. Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice*. 4th ed. New York, NY: Oxford University Press; 2013.
26. Buschbacher RM, Prahlow ND. *Manual of Nerve Conduction Studies*. 2nd ed. New York, NY: Demos Medical Publishing; 2006.
27. Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice*. 4th ed. New York, NY: Oxford University Press; 2013.
28. Dumitru D, Amato AA, Zwarts MJ. *Electrodiagnostic Medicine*. 2nd ed. Philadelphia, PA: Hanley & Belfus; 2002.
29. Nuwer MR. Somatosensory evoked potentials. *Muscle Nerve*. 1999;22(8):1007-1023.
30. Boe SG, Dalton BH, Harwood B, Doherty TJ, Rice CL. Estimating the number of motor units in human muscles using the spike-triggered averaging technique. *J Vis Exp*. 2010;(43):2163.
31. Bhadra N, Peckham PH, Kilgore KL. Neuromuscular stimulation for restoration of hand function after spinal cord injury and stroke. *Neurol Res*. 2001;23(6):653-661.
32. Robinson LR. Traumatic injury to peripheral nerves. *Muscle Nerve*. 2000;23(6):863-873.
33. Shahani BT. Electrophysiological evaluation of brachial plexus lesions. *Neurol Clin*. 1999;17(3):529-547.
34. Wilbourn AJ. Electrodiagnostic testing of the brachial plexus. *Neurol Clin*. 1999;17(3):525-548.
35. Lee HJ, Wolfe SW. Diagnostic and therapeutic approach to brachial plexus injuries. *J Am Acad Orthop Surg*. 2009;17(10):590-600.
36. Nardin RA, Patel MR, Gabel B, Putz D, Rutkove SB. Electromyography and nerve conduction studies in preganglionic lesions. *Muscle Nerve*. 1999;22(5):682-690.
37. Midha R. Surgical management of brachial plexus injuries. *Neurosurg Clin N Am*. 2001;12(2):343-359.
38. Preston DC, Shapiro BE. *Electromyography and Neuromuscular Disorders*. 3rd ed. Philadelphia, PA: Elsevier; 2013.
39. Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle*. 4th ed. New York, NY: Oxford University Press; 2013.
40. Shahani BT, Young RR. Human electromyography in normal subjects and in patients with motor neuron disease. *Arch Neurol*. 1973;29(2):111-116.
41. Daube JR. Needle electromyography. *Muscle Nerve*. 2000;23(4):517-539.
42. Wilbourn AJ. Electrodiagnostic studies in brachial plexopathies. *Neurol Clin*. 1999;17(3):525-548.
43. Chhabra A, Williams EH, Wang KC, Dellon AL, Carrino JA. MR neurography of neuromas related to nerve injury and entrapment with surgical correlation. *AJNR Am J Neuroradiol*. 2010;31(8):1363-1368.
44. Robinson LR. Role of neurophysiologic evaluation in diagnosis. *Phys Med Rehabil Clin N Am*. 2003;14(1):33-60.
45. Midha R. Management of nerve injuries. *Clin Plast Surg*. 2003;30(2):203-221.

10.48047/jocaaa.2024.33.06.61

46. Kim DH, Murovic JA, Tiel RL, Kline DG. Surgical management and outcomes in 273 brachial plexus lesions. *J Neurosurg*. 2004;100(4 Suppl Spine):365-376.
47. Robinson LR. Predictive value of electrodiagnostic testing in peripheral nerve injuries. *Phys Med Rehabil Clin N Am*. 1998;9(4):791-808.
48. Nardin RA, Rutkove SB. Clinical utility of EMG in evaluating neuromuscular diseases. *Neurologist*. 2001;7(6):309-323.
49. Terzis JK, Kostopoulos VK. The surgical treatment of brachial plexus injuries in adults. *Plast Reconstr Surg*. 2007;119(4):73e-92e.
50. Green DP, Hotchkiss RN, Pederson WC, Wolfe SW. *Green's Operative Hand Surgery*. 5th ed. Philadelphia, PA: Elsevier Churchill Livingstone; 2005.
51. Bhandari PS, Deb P, Bhatti TS. Role of electromyography in brachial plexus injury. *Indian J Plast Surg*. 2011;44(1):21-27.
52. Preston DC, Shapiro BE. Limitations of electrodiagnosis. In: *Electromyography and Neuromuscular Disorders*. 3rd ed. Philadelphia, PA: Elsevier; 2013.
53. Robinson LR. Role of electrodiagnostic studies in early nerve injury evaluation. *Muscle Nerve*. 2004;30(6):683-694.
54. Shahani BT. Electrodiagnostic challenges in brachial plexopathy. *Clin Neurophysiol*. 1997;104(5):407-412.
55. Wilbourn AJ. Pitfalls in the electrodiagnosis of peripheral nerve lesions. *Neurol Clin*. 2002;20(1):179-206.
56. Daube JR. Common errors in clinical electromyography. *Muscle Nerve*. 1996;19(3):234-244.
57. Rutkove SB. Electrical impedance myography: background, current state, and future directions. *Muscle Nerve*. 2009;40(6):936-946.
58. Chhabra A, Subhawong TK, Carrino JA. MR neurography: current perspectives and literature review. *AJNR Am J Neuroradiol*. 2011;32(11):1957-1966.
59. Birch R. Brachial plexus injury: diagnosis and investigation. *J Hand Surg Br*. 2001;26(2):99-102.
60. Chhabra A, Madhuranthakam AJ, Andreisek G. Magnetic resonance neurography: current perspectives and literature review. *Eur Radiol*. 2018;28(2):698-707.
61. Zhang Z, Song L, Meng Q, Zhang Y. Application of MR neurography in traumatic brachial plexus injury: correlation with intraoperative findings. *Eur J Radiol*. 2020;125:108875.
62. Spinner RJ, Amrami KK, Dyck PJ. Brachial plexus imaging: current concepts, techniques, and diagnostic findings. *Clin Neurophysiol*. 2008;119(10):2380-2391.
63. Terzis JK, Kostopoulos VK. Outcomes of primary nerve reconstruction in preganglionic brachial plexus injuries. *Plast Reconstr Surg*. 2007;120(5):1341-1359.
64. Kim DH, Murovic JA, Tiel RL, Kline DG. Surgical treatment outcomes of 546 brachial plexus lesions. *J Neurosurg*. 2003;98(5):1005-1016.