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Review article

# Standard neurophysiological studies and motor evoked potentials in evaluation of traumatic brachial plexus injuries – A brief review of the literature

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ABSTRACT

*Purpose:* Traumatic damage to the brachial plexus is associated with temporary or permanent motor and sensory dysfunction of the upper extremity. It may lead to the severe disability of the patient, often excluded from the daily life activity. The pathomechanism of brachial plexus injury usually results from damage detected in structures taking origin in the rupture, stretching or cervical roots avulsion from the spinal cord. Often the complexity of traumatic brachial plexus injury requires a multidisciplinary diagnostic process including clinical evaluation supplemented with clinical neurophysiology methods assessing the functional state of its structures. Their presentation is the primary goal of this paper.

*Methods:* The basis for the diagnosis of brachial plexus function is a clinical examination and neurophysiology studies: electroneurography (ENG), needle electromyography (EMG), somatosensory evoked potentials (SEPs) and motor evoked potentials (MEPs) assessing the function of individual brachial plexus elements.

*Conclusions:* The ENG and EMG studies clarify the level of brachial plexus damage, its type and severity, mainly using the Seddon clinical classification. In contrast to F-wave studies, the use of the MEPs in the evaluation of traumatic brachial plexus injury provides valuable information about the function of its proximal part. MEPs study may be an additional diagnostic in confirming the location and extent of the lesion, considering the pathomechanism of the damage. Clinical neurophysiology studies are the basis for determining the appropriate therapeutic program, including choice of conservative or reconstructive surgery which results are verified in prospective studies.





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## 1. Introduction

The brachial plexus is one of the most complex structures of the peripheral nervous system. Traumatic damage to the brachial plexus is a serious medical problem, both diagnostic and therapeutic. Sensitivity to injury is mainly due to its surface position and location between two highly mobile structures such as neck and shoulder [1]. Its damage is associated with temporary or permanent motor and sensory dysfunction of the upper extremity [2]. It leads to a severe disability of the patient, often excluding him from the day-life activity [3,4]. Multi-organ damage often coexisting with brachial plexus damage can delay its clinical diagnosis and initiation of the proper management [2]. Traumatic injury to the brachial plexus can be a consequence of sudden traction of the upper extremity, damage to the skeletal structures surrounding the brachial plexus as well as the head, neck and axillary level injuries. Brachial plexus injury often coexists with large arteries damage [3,5]. Lesion to the brachial plexus can be the result of direct traumas related to sports, accidents at work and as an effect of iatrogenic injuries (mastectomy, subclavian-carotid bypass, first rib resection) [2,3,6,7]. The significant increase in motorization contributes to the rise in traffic accidents and related severe multi-organs injuries, including those in peripheral and central nervous system. It is estimated that traumatic damage to the brachial plexus is a consequence of motorbike accidents the most frequently. It usually affects young people in the 2nd–3rd decade of life, especially men [2,3,5]. The pathomechanism of brachial plexus injury often results from the effects of significant force and tissue overloading. It leads to damage to the brachial plexus structures in the rupture mechanism, stretching, and roots avulsion from the spinal cord in severe cases [3,8]. Traumatic injury to the brachial plexus may result from the mentioned – above pathomechanisms coexisting at the same time [5]. Most often it is related to the supraclavicular part, rarely to the middle or subclavian. Therefore, the injury mostly occurs in the roots or trunks than the divisions, cords or peripheral nerves. Upper brachial plexus damage appears when the neck and head are firmly moved away from the ipsilateral shoulder [3,5,9]. The result of this mechanism is a stretch, rupture or even C5–C7 root avulsion without damage to C7–C8 cervical roots. This type of injury takes place mostly in motorcycle accidents. The lower elements of brachial plexus (C8–T1) can be injured by violently abduction and traction of the ipsilateral arm. According to Moran et al. [3], about 70–75% of injuries concern the supraclavicular region, and 75% of these injuries include an injury to the entire plexus (C5–T1). Furthermore, 20–25% of injuries involve damage to the nerve roots of C5 through C7, and 2–35% of injuries have isolated supraclavicular injury patterns to C8 and T1. Panplexal injuries usually engage a C5–C6 rupture with a C7–T1 roots avulsion. The remaining 25% of plexus injuries are subclavicular. The complexity of brachial plexus damage is often difficult to diagnose. It should be remembered that during the passage of time there can appear degenerative changes in motor end-plates causing the muscular atrophy. Hence, proper diagnosis, conservative treatment or surgical intervention may contribute to the efficient regeneration of damaged brachial plexus structures [3,5]. The result of brachial plexus injury depends on multiple factors, and it is closely related to neural plasticity process [10–12]. The basis for the diagnosis of brachial plexus is a clinical examination [2,5,13,14] as well as clinical neurophysiology studies (ENG – electroneurography, EMG – needle electromyography, SSEP – somatosensory evoked potentials, MEP – motor evoked potentials) assessing the function of individual brachial plexus elements. The above studies are aimed at determining the etiology of brachial plexus injury, clarifying its level (proximal or distal to the Dorsal Root Ganglion – DRG), type and severity of injury (mainly using the Sunderland clinical classification) [2,5]. The use of the MEP examination in traumatic brachial plexus injury provides valuable information about the function of its proximal part. MEP study may be additional in confirming the location and extent of the lesion, considering the pathomechanism of the damage (roots avulsion, rupture, stretch). Clinical neurophysiology studies assessing the functional state of brachial plexus structures are the basis for determining the appropriate therapeutic program, including conservative or surgical treatment. The presented work is addressed to clinicians and researchers dealing primarily with the neurophysiological evaluation of patients after brachial plexus damage.

## 2. Clinical examination

A detailed clinical study should include assessment of the motor activity of the shoulder girdle muscles and the upper extremity, assessment of muscle strength, their atrophy and assessment of dysesthesia. It should also include determination of the other possible disorders associated with brachial plexus injuries such as Horner's syndrome indicating the sympathetic ganglion damage at the T1 root level and hyperreflexia determining injury to the upper motor neuron [3,15].

The function of the median, ulnar and radial nerves is assessed by examining the activity of the fingers and wrist. Damage to the lower part of the brachial plexus (C8–T1) leads to paralysis and atrophy of the intrinsic muscles of the hand, disturbances of fingers adduction, abduction and flexion in the phalangeal joints as well as an abnormal sensation on the medial side of the hand. Isolated injury of the middle part of the brachial plexus (C7) is quite rare, usually accompanied by damage to both the upper and lower parts of the plexus. It results in paresis of the extensors muscles of the wrist and fingers as well as the triceps muscle dysfunction. Sensation disturbances of the shoulder girdle and lateral part of the arm, as well as the atrophy of the shoulder girdle muscles, indicate damage to the upper part of the brachial plexus (C5–C6). Impairment of the scapula adduction, disorders of external rotation and shoulder abduction as well as derangement of forearm supination and elbow flexion are observed [2,15]. Clinical symptoms of brachial plexus injury presented above require confirmation in neurophysiological tests. The diagnostic algorithm is summarized in Table 1.



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