

Wiertel-Krawczuk A., Wojtysiak M. *The role of neurophysiological studies in evaluating of two different types of traumatic brachial plexus injury. Issue Rehabil. Orthop. Neurophysiol. Sport Promot.* 2017; 21: 67–80. DOI: 10.19271/IRONS-00047-2017-21

THE ROLE OF NEUROPHYSIOLOGICAL STUDIES IN EVALUATING OF TWO DIFFERENT TYPES OF TRAUMATIC BRACHIAL PLEXUS INJURY

Agnieszka Wiertel-Krawczuk

Magdalena Wojtysiak

Department of Pathophysiology of Locomotor Organs, Poznań University of Medical Sciences, Poland

ROLA BADAŃ NEUROFIZJOLOGII KLINICZNEJ W OCENIE DWÓCH ODMIENNYCH PRZYPADKÓW URAZOWEGO USZKODZENIA SPLOTU RAMIENNEGO

Agnieszka Wiertel-Krawczuk

Magdalena Wojtysiak

Zakład Patofizjologii Narządu Ruchu, Uniwersytet Medyczny w Poznaniu, Polska

SUMMARY

Introduction

The anatomical variation and possible combination of sensory and motor shortage makes difficulties to investigate the distribution of the damage and to choose appropriate treatment procedure in traumatic brachial plexus injury. Neurophysiological studies provide valuable information about functional state of brachial plexus.

Aim

The purpose of this paper was to present two significantly different cases of brachial plexus injury both for the lesion mechanism and the results of the clinical and neurophysiological studies.

Material and methods

The assessment of brachial plexus damage was based on clinical studies and neurophysiological studies including electroneurography (ENG), electromyography (EMG) and motor evoked potentials (MEP) performed in two patients after traumatic brachial plexus injury.

Results

Clinical and neurophysiological studies have shown partial postganglionic brachial

STRESZCZENIE

Wstęp

Urazowe uszkodzenie spłotu ramiennego jest poważnym problemem medycznym zarówno diagnostycznym, jak i terapeutycznym. Zróżnicowanie anatomiczne i możliwe komplikacje zaburzeń czuciowych i ruchowych utrudniają zbadanie rozkładu uszkodzenia spłotu ramiennego i dobranie odpowiedniej procedury leczenia. Badania neurofizjologiczne dostarczają cennych informacji odnośnie stanu czynnościowego spłotu ramiennego.

Cel

Celem pracy było przedstawienie dwóch różnych przypadków uszkodzenia spłotu ramiennego zarówno w aspekcie mechanizmu uszkodzenia, jak i wyników badań klinicznych i neurofizjologicznych.

Material i metody

Ocena uszkodzenia spłotu ramiennego dokonywana była na podstawie badań klinicznych oraz neurofizjologii klinicznej w tym electroneurografia (ENG), elektromiografia (EMG) oraz ruchowe potencjały wywołane (MEP). Badania wykonywane były u dwóch chorych po urazowym uszkodzeniu spłotu ramiennego.

Wyniki

Na podstawie badań klinicznych i neurofizjologicznych rozpoznano u pierwszego

plexus injury with axonal-demyelinating features and denervation activity in the examined muscles in the first patient. After a year, spontaneous regeneration of brachial plexus (improvement of conduction parameters and muscle reinnervation) was observed. In the second patient total post-ganglionic damage to the brachial plexus with denervation process in the muscles was observed in the clinical and neurophysiological study, without any symptoms of spontaneous regeneration one year after injury.

Conclusions

MEP does not determine the pre- or post-ganglionic nature of the lesion and has limited use in total brachial plexus damage. MEP can provide valuable information in the assessment of the proximal part of the brachial plexus after reconstruction surgery. The ENG and EMG tests allow to determine the location of the damage and its severity. The above tests are a sensitive tool in evaluating the progression of functional changes of the brachial plexus.

Keywords: traumatic injury, brachial plexus, neurophysiological study

Date received: 23th October 2017

Date accepted: 17th November 2017

Introduction

Traumatic damage to the brachial plexus is a serious medical problem, both diagnostic and therapeutic. Sensitivity to injury results mainly from its surface position and location between two highly mobile structures such as neck and shoulder (Ferrante 2012). Its damage is associated with temporary or permanent motor and sensory dysfunction of the upper limb (Gregory *et al.* 2009). There are many causes of traumatic injury to the brachial plexus,

pacjenta częściowe zazwojowe uszkodzenie splotu ramiennego (typ Erba) o charakterze mieszanym aksonalno-demielinizacyjnym z cechami odnerwienia w badanych mięśniach. Po roku w drugim badaniu zarówno klinicznym jak i neurofizjologicznym (poprawa parametrów przewodzenia oraz obecne cechy reinerwacji w mięśniach) obserwowano samoistną regenerację splotu ramiennego. U drugiego pacjenta w badaniu klinicznym i neurofizjologicznym rozpoznano zazwojowe całkowite uszkodzenie splotu ramiennego z cechami odnerwienia w badanych mięśniach, bez cech spontanicznej regeneracji po roku od uszkodzenia.

Wnioski

Badanie ruchowych potencjałów wywołanych nie rozstrzyga o przed- czy za zwojowym charakterze uszkodzenia i ma ograniczone zastosowanie w całkowitym uszkodzeniu splotu ramiennego. Może wносить cenne informacje w ocenie części proksymalnej splotu ramiennego po zabiegach rekonstrukcyjnych. Badanie ENG oraz EMG pozwalają na określenie lokalizacji uszkodzenia oraz jego ciężkości. Są czułym narzędziem w ocenie progresji zmian czynnościowych splotu ramiennego.

Słowa kluczowe: urazowe uszkodzenie, splot ramienny, badania neurofizjologiczne

Data otrzymania: 23 października 2017

Data zaakceptowania: 17 listopada 2017

mainly due to the sudden traction of the upper limb as well as the damage of the skeletal structures surrounding the plexus, head and neck injuries, axillary fossa lesion, which is also often accompanied by damage to large arteries (Moran *et al.* 2005, Ghany *et al.* 2011). Lesion to the brachial plexus can be the result of, among other things, direct injuries related to sports, workplace accidents, falls from high (Moran *et al.* 2005, Gregory

et al. 2009). Significant increase in motorization, especially in Western countries, contributes significantly to the increase in traffic accidents and related serious multiorgan trauma, including peripheral and central nervous system injuries. Traumatic damage to the brachial plexus is a consequence mainly of motorcycle accidents. This usually applies to young people in 2–3 decade of life, for the vast majority of men (Moran *et al.* 2005, Gregory *et al.* 2009, Ghany *et al.* 2011). The pathomechanism of brachial plexus injury usually results from the effects of significant forces and tissue overload, leading to damage neural structures in the mechanism of rupture, stretching and in severe cases of cervical roots avulsion from the spinal cord (Moran *et al.* 2005). The most common is supraclavicular brachial plexus injury, less common is infraclavicular injury or damage directly on clavicular level. Standard diagnostic procedures for brachial plexus include clinical examination, imaging studies (USG, CT / myelography or MRI) (Ghany *et al.* 2011, Rankine 2004, Gregory *et al.* 2009) and neurophysiological studies like electroneurography (ENG), electromyography (EMG) as well as somatosensory evoked potentials (SSEP) and motor evoked potentials (MEP). The above studies aim to determine the etiology of injury, to clarify its level (proximal or distal to the Dorsal Root Ganglion), type and severity of the lesion (using mainly Sunderland classification of nerve damage) (Ghany *et al.* 2011, Gregory *et al.* 2009). The management of brachial plexus depends on the degree of damage and the site of injury, these are the most important factors. Treatment of brachial plexus injury is either conservative or surgical. Surgical procedures include neurolysis, nerve grafting, nerve transfer, and other reconstructive methods other such as transplantation of different structures (Ghany *et al.* 2011).

The purpose of this paper was to present two significantly different cases of brachial

plexus injury both for the lesion mechanism and the results of the clinical and neurophysiological studies.

Materials and methods

Clinical examination included the evaluation of the motor function of the shoulder and upper limb, the assessment of muscle strength, their atrophy, and the evaluation of sensory disturbances. In addition, other possible disorders associated with brachial plexus injury such as the Horner syndrome showing T1 sympathetic injury, excessive reflexes that impair upper motor neuron damage were included.

Neurophysiological tests were recorded with Keypoint system (Medtronic A/S, Skovlunde, Denmark). During ENG examination standard (AgCl) 5 mm² surface electrodes were used for recording compound muscle action potentials (CMAP). The active electrode (cathode) was placed on muscle belly, reference electrode was positioned on muscle's tendon. Stimulating bipolar electrode was applied along the anatomical passage of each examined nerve (Lee and DeLisa 2005). Time base was set on 5 ms/D, recordings sensitivity on 2 mV/D, 20 Hz upper and 10 kHz lower filters of recorder amplifier were used during ENG. Intensity of single electrical stimuli (with 0.2 ms duration and 1 Hz frequency) was increased from 20 mA to value evoking the potential with the largest amplitude (supramaximal stimulus). Values of amplitude (Di Bella *et al.* 1997), latency and conduction time or conduction velocity (Kimura 2001, Preston and Shapiro 2005, Daube and Rubin 2009) were analysed in CMAP recordings. Confirmation of axonal changes in brachial plexus nerves required needle EMG examination from upper extremity muscles (Lee and DeLisa 2005) showing the presence of denervation potentials or reinnervation process. The study included the analysis of muscle's spontaneous activity at rest and evaluation of twenty motor unit action potentials (MUAPs) parameters

(amplitude in mV, duration in ms and Size Index in mV/ms) recorded during weak voluntary contraction (Daube and Rubin 2009). The last stage of EMG examination included measurements of amplitude and frequency of motor unit recruitment during maximal voluntary contraction (Stålberg and Falck 1997). Conducting the two last stages of EMG examination was possible only in muscles presenting the voluntary activity.

To confirm the function of the proximal part of the brachial plexus, MEPs induced by the magnetic field were used. For stimulation, a 110mm rounded induction coil connected to MagPro X-100 pulse generator (Medtronic) was applied at both the Erb's point and the cervical spine, 1 cm lateral to the spinous process of four root levels (C5-C8). At both excitation points, a magnetic impulse of 100% (1.9T) of the stimulator output was used. Time base was set on 10 ms/D, recordings sensitivity on 2 mV/D, 20 Hz upper and 10 kHz lower filters of recorder amplifier and two-phase pulses with a duration of 5ms were used during MEP study. MEP's were recorded from specific upper limb muscles, which are the effectors of the corresponding peripheral nerves. The amplitude, latency and the latency difference between the stimulation points corresponding to the conduction time from the cervical spine level to Erb's point were analyzed in MEP study.

Case Report 1

History and Examination

The patient was a 59-year-old who fell to the ground from height of 4 meters. He suffered clavicular fracture (closed trauma) with significant hematoma present at this level and within the proximal part of the left upper limb. Conservative treatment consisting of clavicular reductino and immobilization of the upper limb was applied. Within 6 weeks after accident, progressive left upper limb paresis was observed, and hematoma decompression has been applied. After four months, a clinical examination

revealed lesion of the upper part of the brachial plexus (Erb palsy) with apparent muscle atrophy mostly of the deltoid muscle and biceps brachii muscle. On motor examination, he had reduced strength in arm abduction, rotation and elbow flexion. The clinical study did not show the presence of Horner's syndrome and excessive reflexes in the left upper limb.

Neurophysiological Examination

In the first ENG study, a decrease in amplitude of CMAP and prolongation of the conduction time were observed in the motor fibers of suprascapular nerve, axillary nerve and the musculocutaneous nerve as well as in sensory fibers of median nerve after stimulation of first, second and third finger. In EMG examination of the deltoid and biceps brachii muscles, denervation potentials and neurogenic MUAPs were recorded.

After one year, second clinical examination showed symptoms of spontaneous regeneration of the left upper extremity but with still demonstrated lesion of the upper trunk of brachial plexus and visible asymmetry and slight muscle mass loss (Figure 1). On motor examination the range of arm abduction, rotation and elbow flexion were higher than in first examination.



Figure 1. Example of injury to upper brachial plexus with apparent atrophy of shoulder muscles (A) and example of abnormal clavicle adhesion after its fracture (B).

The results of neurophysiological examination, ENG and MEP studies of the left brachial plexus nerves performed one year after the previous study are presented on Figures 2 and 3 respectively.

The prolongation of conduction time in axillary nerve and musculocutaneous nerve was recorded. The Carpal Tunnel Syndrome was also noted in ENG studies (also in the previous study), which may be the basis

ENG study of left upper limb

MNCS						
Nerves - left upper extremity	Lat		Amp		CV	
	ms	Ref.Dev	mV	Ref.Dev	m/s	Ref.Dev
Axillaris Motor						
Erb - Deltoid	4.77		14.1			
Medianus Motor						
Wrist - APB	4.71	3.6	8.0	-0.29		
Elbow-Wrist	8.85		7.9	3.7	50.7	-2.1
Ab. elbow-Elbow	10.8		7.7		53.8	
Erb-Ab. elbow	16.0		0.76		57.7	
Musculocutaneous Motor						
Erb - Biceps	5.21	1.81	11.7	7.7		
Radialis Motor						
Erb-Ab. elbow	7.85		8.6		51.8	
Radialis r. brevis Motor						
Erb - m. triceps brachii	5.04		9.0			
Ulnaris Motor						
Wrist - ADM	3.00	-0.30	7.9	1.90		
Bl. elbow-Wrist	6.65		8.0		54.8	5.8
Ab. elbow-Bl. elbow	9.25		7.1		51.9	2.9
Erb-Ab. elbow	15.2		1.02		50.4	

F wave	M	F min	F max	Ilość
	ms	ms	ms	%
Medianus				
Wrist - APB	5.2	19.8	33.4	95.0
Ulnaris				
Wrist - ADM	1.71	25.2	30.9	100

SNCS						
Nerves - left upper extremity	Peak Lat		Amp		CV	
	ms	Ref.Dev	uV	Ref.Dev	m/s	Ref.Dev
Medianus						
Dig I - Wrist	4.03		0.35		34.8	
Dig II - Wrist	4.25	3.0	1.82	-4.8	34.1	-5.0
Dig III - Wrist	4.08		8.3		37.9	
Radialis						
Forearm - Wrist	2.90	0	20.3		67.7	
Ulnaris						
Dig V - Wrist	2.40	-0.70	6.3		51.1	

Figure 2. The results motor (MNCS) and sensory nerve conduction studies (SNCS) and F wave examination of short and long branches of the left brachial plexus performed one year after the previous study.

The MNCS study highlights the reduced CMAP amplitude after electrical stimulation from the Erb's point of the median nerve and the ulnar nerve, which would suggest a conduction block at this level. However, the correct frequency of F wave in both examined nerves and proper conduction velocity at this level precludes this suspicion.

for a hypothesis regarding the coexistence of a double crash syndrome in this case of patient.

In the SNCS study of median nerve, there was a considerable slowing of conduction velocity and reduction in SNAP amplitude especially after first and second finger stimulation. Conduction of nerve impulses in

the sensory fibers in ulnar and radial nerve was within the normal range.

The results of the ENG study were not compared with the asymptomatic side. Such an analysis was made in the MEP study.

voluntary contraction were normal except those recorded from the deltoid muscle and the biceps brachii muscle (reinnervation symptoms), the interference pattern was recorded while maximum muscle contraction.

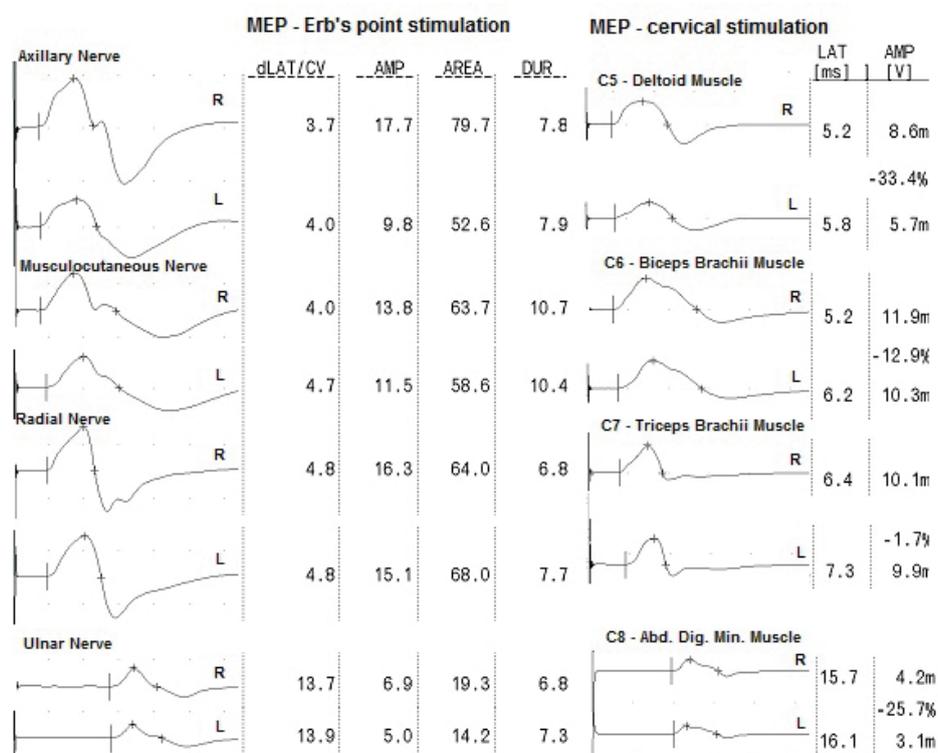


Figure 3. The results of MEP examination of the left brachial plexus after magnetic stimulation from the Erb's point and from the cervical spine performed one year after the previous study.

The MEP studies showed asymmetry in CMAP amplitude mainly due to axillary nerve magnetic stimulation from the Erb's point (44%) and to a lesser extent after C5 level of the cervical spine (34%) as compared to the asymptomatic side. Latency values were comparable on both sides after stimulation alike from the Erb's point and from the cervical spine level (C5-C8) except for the axillary nerve and the musculocutaneous nerve, the latencies were prolonged in comparison with the right side.

In the EMG study of the left deltoid muscle, biceps brachii muscle, triceps brachii muscle, first interosseous dorsal muscle and abductor pollicis brevis muscle at rest, no denervation potentials were recorded. The MUAP parameters recorded during

Clinical neurophysiology studies have confirmed the progressive spontaneous brachial plexus regeneration, showing still coexisting exponents of upper trunk damage, but with symptoms of reinnervation in the examined muscles. The axonal-demyelinating mixed lesion in the first study changed its character to a primarily demyelinating nature of lesion. The outcome of neurophysiological studies is consistent with the clinical examination of the patient at the same time.

Case Report 2

History and Examination

The patient was 39-year-old man who has suffered a multi-organ injury because of a motor vehicle accident. In the early period

after the accident, the right upper extremity treatment (close trauma) was limited to skin transplants instead of tissue defect at arm and forearm level. Due to the severity of the illness and the associated multi-organ trauma, the treatment and full diagnosis of brachial plexus was implemented one year after the injury. Magnetic resonance imaging revealed damage to brachial plexus structures without cervical root injury. Clinical examination of the right upper limb demonstrated a complete brachial plexus lesion with upper limb muscular atrophy and no signs of clinical recovery. On motor examination, the patient had complete absence of strength in right arm abduction, elbow flexion, elbow extension, forearm supination, wrist extension, and finger extension (Figure 4).

In needle EMG study denervation potentials (Figure 7) and lack of voluntary contraction of right upper extremity muscles were recorded confirming the results of EMG and MEP studies.

The results of the neurophysiological studies were consistent with the results of clinical trials. Based on the results a further therapeutic regimen has been established, including the reconstructive surgery of the brachial plexus.

Discussion

Diagnostic electrophysiological criteria evaluation of brachial plexus injury include ENG (nerve conduction test) and EMG needle test, which is one of the most useful neurophysiological methods used in confirming the severity and the level of injury

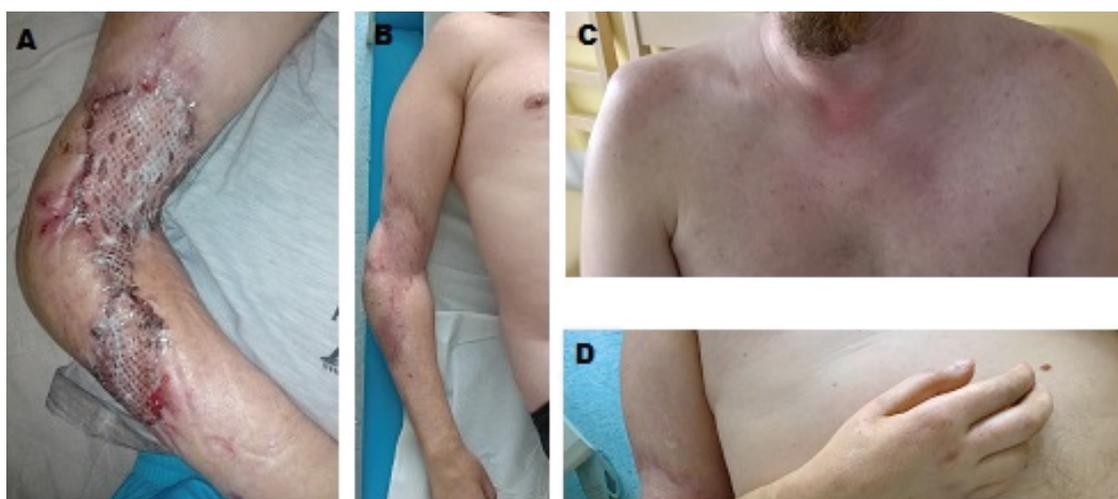


Figure 4. Photographs show the location of the skin graft (A) and upper limb muscles atrophy (B-D, one year after injury) in the patient after complete damage to the right brachial plexus.

Neurophysiological Examination

The following figure (Figure 5) shows the results of the ENG study (MNCS and SNCS). The potentials of motor and sensory fibers of brachial plexus have not been recorded, which confirms total postganglionic damage to the brachial plexus. No motor evoked potentials (MEPs) have been recorded after magnetic stimulation from the cervical spine (C5-C8) (figure 6). Based on this study, the type of injury between axonotmesis and neurotmesis cannot be distinguished.

(Ciaramitaro *et al.* 2016, Mansukhani 2013). An essential part of assessing brachial plexus damage is the analysis of sensory fibers based on SNCS and SSEP studies, which is helpful in proving pre- or post-ganglionic nature of brachial plexus damage as well as distinguishing possible pathologies of individual brachial plexus structures such as trunks or cords (Ferrante 2012, Ferrante and Wilbourn 1995). SSEP analysis, as it is helpful in determining the integrity of afferent pathways and damage at pre-or

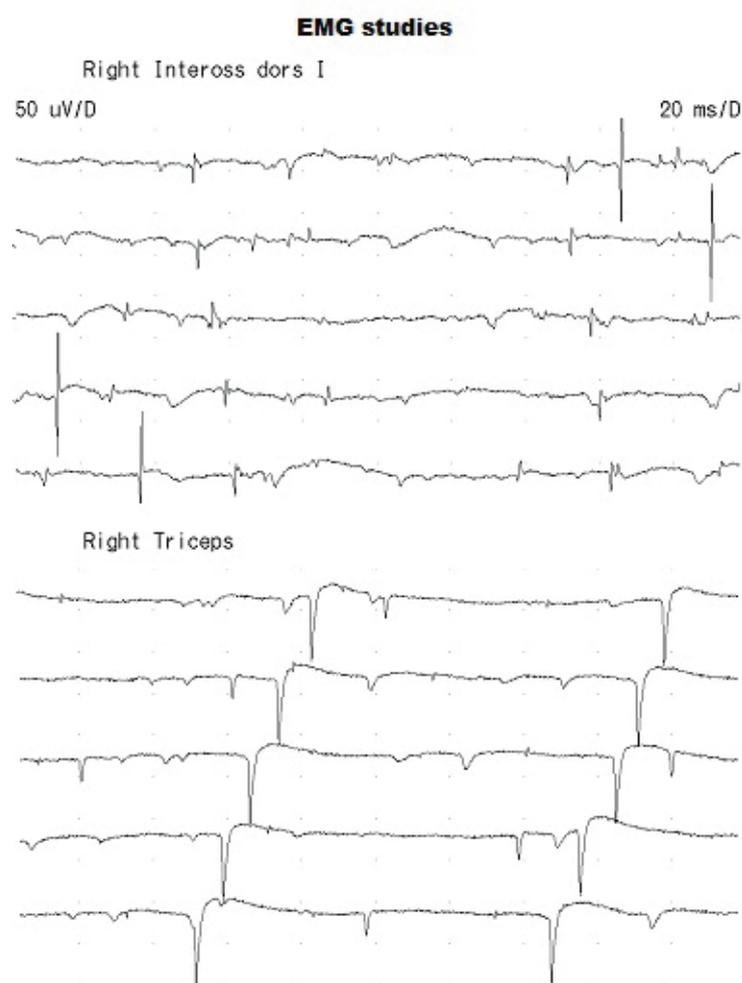


Figure 7. An example of denervation activity (positive sharp waves and fibrillation potentials) recorded from the first interosseous dorsal and triceps brachii muscles in EMG study.

post-ganglionic levels, cannot be conclusive in determining damage at a specific root level (Abbruzzese *et al.* 1993, Ferrante 2012, Serror 1994). An alternative method for electrical stimulation of the brachial plexus structure is magnetic stimulation. Application of magnetic stimulation at Erb's point and at the level of cervical spine, which reflects ventral roots at the level of intervertebral foramen, may be used to evaluate the proximal part of the lower motor neuron (Abbruzzese *et al.* 1993). The use of magnetic stimulation is considered by some authors to be less objective, mainly because there is no evidence that the intensity of magnetic stimulation is supramaximal, so that it stimulates the proper number of axons within the nerve domain. Hence, the

determination of the conduction block in the proximal part of the nerve after magnetic stimulation is not objective. However, as Öge *et al.* (Öge *et al.* 1997) and other authors (Seror 1994, Veltsista and Chroni 2015) pointed that CMAP amplitude induced by magnetic stimulation from the neck level may be reduced in comparison to CMAP induced by electrical or magnetic stimulation at distal site, and form the basis for suspicion of proximal conduction block. Magnetic stimulation brings important information mainly in the aspect of segmental demyelination in the proximal part of the examined nerves. According to the authors, magnetic stimulation included in standard neurophysiological tests may provide information on localization of lesions, segmental

demyelination or axonal loss of the brachial plexus primarily affected by compression. In our study, similarly, magnetic stimulation applied from Erb's point as well as over the cervical spine was an additional method attached to the diagnostic standard including the ENG and EMG studies. In patient with brachial plexus compression (Patient No. 1), magnetic stimulation demonstrated a slight prolongation of conduction time on the cervical spine C5-C6 – Erb's point segment and reduced amplitude, especially in the axillary nerve, compared to the healthy side. In the case of patients with complete brachial plexus injury (Patient No. 2), in the absence of motor (ENG, MEP) and sensory evoked potentials, it is possible to conclude that the lesion was in the post-ganglionic part of the brachial plexus. In this case, neurophysiological studies were not able to clarify the nature of the lesion (axonotmesis vs neurotmesis). The absence of motor and sensory potentials and the denervation activity in muscles may be appropriate for both mechanisms of injury. In this case, the imaging study is crucial in determining the location and nature of the lesion, as indicated by many authors (Rankine 2004, Gregory *et al.* 2009, Ghany *et al.* 2012). In patients with severe brachial plexus injury, appropriate reconstructive procedures should be taken prior to irreversible muscle damage, which will determine the effectiveness of the treatment (Martins *et al.* 2013). EMG study was therefore essential to examine the dynamics of both degenerative and regenerative processes (Moran *et al.* 2005, Gregory *et al.* 2009), which was also confirmed by our study. In a patient with a mixed type of lesion, the EMG test showed an acute neurogenic process in the first study. One year after the reinnervation process and interference pattern during maximal muscles contraction were registered. As Gregory *et al.* (Gregory *et al.* 2009) pointed, in the process of regeneration of motor fibers the results of EMG studies will still not be completely correct,

the size of motor units will be greater than before damage and firing pattern of motor units will be different, with increased firing rates to maintain force production.

Conclusion

MEP does not determine the pre- or post-ganglionic nature of the lesion and has limited use in total brachial plexus damage. MEP can provide valuable information in the assessment of the proximal part of the brachial plexus after reconstruction surgery. Compilation of clinical and neurophysiological studies brings important information about the functional state of brachial plexus. It is the basis for the application of therapeutic procedures appropriate to the degree of brachial plexus injury.

REFERENCES

- Abbruzzese, G., Morena, M., Caponnetto, C., Trompetto, M., Abbruzzese, M., Favale, E.** (1993) 'Motor evoked potentials following cervical electrical stimulation in brachial plexus lesion.' 241, pp. 63–67.
- Ciaramitaro, P., Mondelli, M., Rota, E., Battiston, B., Sard, A., Pontini, I., Faciani, G., Migliaretti, G., Merola, A., Cocito, D., Italian Network for Traumatic Neuropathies.** (2016) 'Electrophysiological Predictors of Clinical Outcome in Traumatic Neuropathies: A Multicenter Prospective Study.' *Neurol Res Int.*, 2016:4619631, doi: 10.1155/2016/4619631.
- Daube, J.R., Rubin, D.I.** (2009) *Clinical neurophysiology.* Oxford University Press.
- Di Bella, P., Logullo, F., Lagalla, G., Sirolla, C., Provinciali, L.** (1997) 'Reproducibility of normal facial motor nerve conduction studies and their relevance in the electrophysiological assessment of peripheral facial paralysis.' *Neurophysiol Clin.*, 27, pp. 300–308.
- Ferrante, M.A.** (2012) 'Electrodiagnostic Assessment of the Brachial Plexus.' *Neurol Clin.*, 30, pp. 551–580.
- Ferrante, M.A., Wilbourn, A.J.** (1995) 'The utility of various sensory nerve conduction responses in assessing brachial plexopathies.' *Muscle & Nerve*, 18, pp. 879–889.
- Ghany, A.F.A., Rabie, N.M.O., El-Shazly, A.A.R.** (2011) 'Brachial Plexus Injury: Diagnosis of Nerve Root Avulsion Using Multislice CT Myelography.' *Egypt J Neurol Psychiat Neurosurg.*, 48 (1), pp. 63–69.
- Gregory, J., Cowey, A., Jones, M., Pickard, S., Ford, D.** (2009) 'The anatomy, investigations and management of adult brachial plexus injuries.' *Orthopaedics and Trauma*, 23 (6), pp. 420–432.
- Kimura, J.** (2001) *Electrodiagnosis in diseases of nerve and muscle.* Oxford University Press.
- Lee, H.J., DeLisa, J.A.** (2005) *Manual of nerve conduction study and surface anatomy for needle electromyography.* Philadelphia, Lippincott Williams & Wilkins.
- Mansukhani, K.A.** (2013) 'Electrodiagnosis in traumatic brachial plexus injury.' *Ann Indian Acad Neurol.*, 16 (1), pp. 19–25.
- Martins., R.S., Bastos, D., Siqueria, M.G., Heise, C.O, Teixeira, M.J.** (2013) 'Traumatic injuries of peripheral nerves: a review with emphasis on surgical indication.' *Arq Neuropsiquiatr.*, 71 (10), pp. 811–814.
- Moran, S.L, Steinmann, S.P, Shin, A.Y.** (2005) 'Adult Brachial Plexus Injuries: Mechanism, Patterns of Injury, and Physical Diagnosis.' *Hand Clin.*, 21, pp. 13–24.
- Öge, A.E., Boyaciyan, A., Gürvit, H., Yazici, J., Değirmenci, M., Kantemir, E.** (1997) 'Magnetic nerve root stimulation in two types of brachial plexus injury: segmental demyelination and axonal degeneration.' *Muscle & Nerve*, 20, pp. 823–832.
- Preston, D.C., Shapiro, B.E.** (2005) *Electromyography and neuromuscular disorders. Clinical-electrophysiologic correlations.* 2nd Ed. Elsevier, Butterworth & Heinemann.
- Stålberg, E., Falck, B.** (1997) 'The role of electromyography in neurology.' *Electroencephalogr Clin Neurophysiol.*, 103, pp. 579–598.
- Rankine, J.J.** (2004) 'Adult traumatic brachial plexus injury.' *Clinical Radiology.*, 59, pp. 767–774.
- Serror, P.** (1994) 'Study of proximal conduction of the arms using SEP and MEP' *Neurophysiol Clin.*, 24(4), pp. 325–336.
- Veltsista, D., Chroni, E.** (2015) 'Usefulness of cervical root magnetic stimulation in assessing proximal motor nerve conduction.' *Journal of Electromyography and Kinesiology*, 25, pp. 742–748.

