

Brachial plexus injuries: regeneration timing and prognosis in patients without need for urgent operation. Preliminary results on truncus primarius superior

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Summary

Background. In Italy, and more generally in the industrialised countries, traumatic nerve lesions have become more frequent. It is commonly accepted that it is necessary to wait 6 months after injury to suggest surgery if movement does not appear. In the scientific literature, there is no systematic clinical evidence of nerve regeneration timing after trauma, especially regarding brachial plexus.

Method. We have performed a follow-up study of 15 consecutive patients with traumatic brachial plexus injuries involving truncus primarius superior without need for urgent surgery. In each patient an extensive clinical and neurophysiological evaluation was performed to detect the kind of lesion, level of lesion, severity of lesion and the outcome.

Findings. In our sample, some cases improved within a few weeks. This rapid improvement may be attributed to resolution of neuro-apraxic block, in other cases slower improvement occurred due to rearrangement of motor units and axonal regeneration. In some cases voluntary activity clinically appeared after more than 6 months following injury.

Conclusions. The current study is preliminary, but it provides evidence that a period of 6 months may not be sufficient for the reappearance of clinical movement. Moreover, current results confirm that neurophysiological evaluation may be a highly prognostic tool in traumatic nerve lesions. We hope that our study together with other data may provide us a timetable for expected nerve regeneration.

Keywords: Brachial plexus; injuries; nerve regeneration; timing.

Introduction

In Italy, and more generally in industrialised countries, traumatic nerve lesions have become more frequent for several reasons: in Italy, for example, one of the most important reasons is to make wearing of crash helmets compulsory for motorbikers. This law has reduced mortality but it has subsequently increased incidence of brachial plexus lesions we currently observe [12].

The purpose of the present study was to document recovery of nerves from traumatic injuries and to evaluate timing of regeneration in patients without need for urgent operation. In this paper the preliminary results on truncus primarius superior are reported.

Materials and method

We have performed a follow-up study of 15 consecutive patients with traumatic brachial plexus injuries involving the truncus primarius superior without need for urgent surgery. Patients with at least one of the following features were classified “without need for urgent surgery”:

- partial integrity of nerve fibers (demonstrated by clinical examination or EMG)
- nerve conduction block
- no root avulsion (confirmed by neurophysiological and neuro-imaging evaluations)
- no plexus interruption evidence or haematoma at MRI

In each patient an extensive clinical and neurophysiological evaluation was performed to detect the kind of lesion, level of lesion, severity of lesion and outcome [1–3, 10, 14]. In all patients the following neurophysiological tests (according to procedures recommended by IFCN committee International Federation of Clinical Neurophysiology) [3] were carried out:

1. Sensory nerve conduction studies in digit-wrist segments (radial nerve in the first digit-wrist segment, median nerve in the first and third-wrist segments, ulnar nerve in the fifth digit-wrist segment).
2. Motor nerve conduction studies of ulnar nerve (segments: erb point-axilla, arm, across elbow, forearm, wrist-abductor digit minimi), median (erb point-axilla, elbow-wrist, wrist-thenar eminence) and musculocutaneous nerves (erb-axilla, axilla-biceps brachii)
3. Needle EMG evaluation of the following muscles: deltoid, biceps brachii, extensor digiti communis, abductor digiti minimi
4. F wave response of ulnar nerve through wrist stimulation (recording from abductor digiti minimi)

Table 1. *Clinical neurophysiological mixed scale*

Score mixed scale	
-1	no voluntary activity at EMG (BMRC = 0)
0	BMRC = 0 but presence of voluntary activity
1	BMRC scale: 1
2	BMRC scale: 2
3	BMRC scale: 3
4	BMRC scale: 4
5	BMRC scale: 5

In many cases, these tests were associated with a motor evoked potential study through magnetic stimulation of cortex and cervical roots (recording from biceps brachii, abductor digiti minimi and thenar eminence) and a somatosensory evoked potential evaluation registering from scalp, cervical spine, and Erb point (median and ulnar nerve stimulation).

In case of suspected root avulsion or complete lesion of parts of plexus, neuroimaging was performed; if neuroimaging confirmed occurrence of that kind of lesion, the patient was excluded following the above mentioned criteria.

The outcome was based on a mixed scale we developed by using BMRC score (British Medical Research Council) [9] and EMG findings: we scored -1: muscles where no motor units potential (MUP) were detectable at EMG (and BMRC was 0); score 0: muscles where BMRC was 0 but EMG showed MUP; the other scores were the same as of BMRC scale (see Table 1). Each patient was evaluated with a mean of every 4 months.

Results

Results of this study are summarized on graphs with evolution of the clinical-neurophysiological outcome measure. The figures show the outcome evolution of deltoid, biceps brachii and brachioradialis muscles in patients with brachial plexus damage. Given that in clinical practice it is commonly accepted that it is better to wait for 6 months after injury to suggest surgery if movement does not appear, the graphs include a box to highlight some cases where clinical movement appeared after 6 months [11]. Some cases improved within a few weeks. This rapid improvement may be attributed to resolution of the neuroapraxic block; in other cases there was a slower improvement due to the rearrangement of motor units and axonal regeneration.

Regarding the outcome of deltoid (Fig. 1), excluding cases with rapid improvement due to resolution of neuroapraxic block, in many cases movement appeared after 6 months and complete improvement occurred after about 1.5 years.

Concerning the outcome of biceps brachii and brachioradialis (Figs. 2 and 3, respectively), excluding cases with rapid improvement due to resolution of neuroapraxic block, in many cases movement ap-

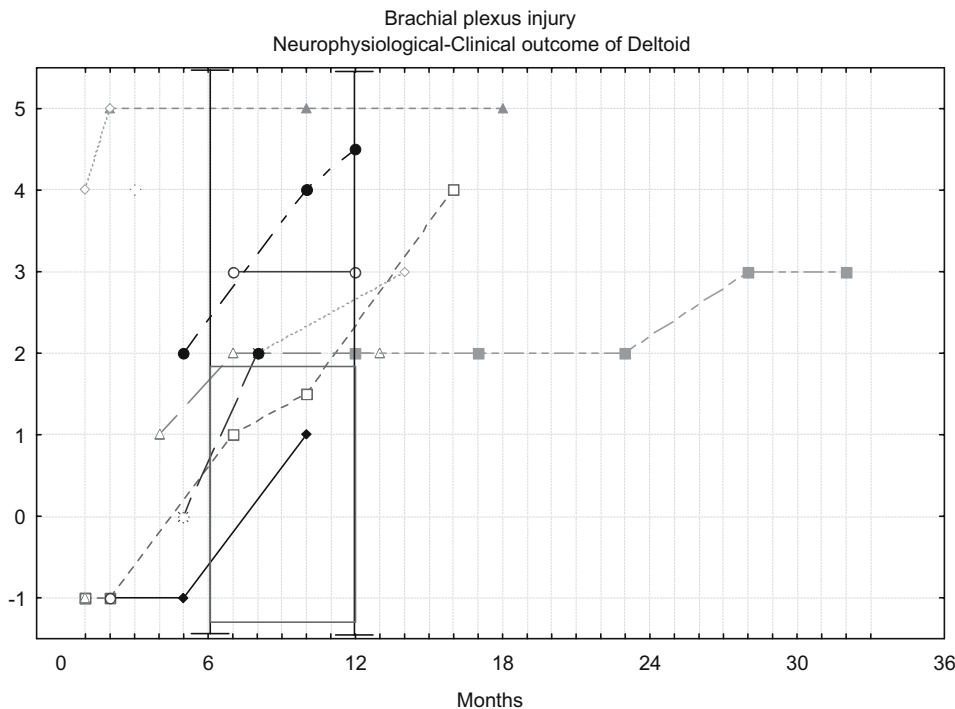


Fig. 1. Shows the neurophysiological-clinical outcome of Deltoid

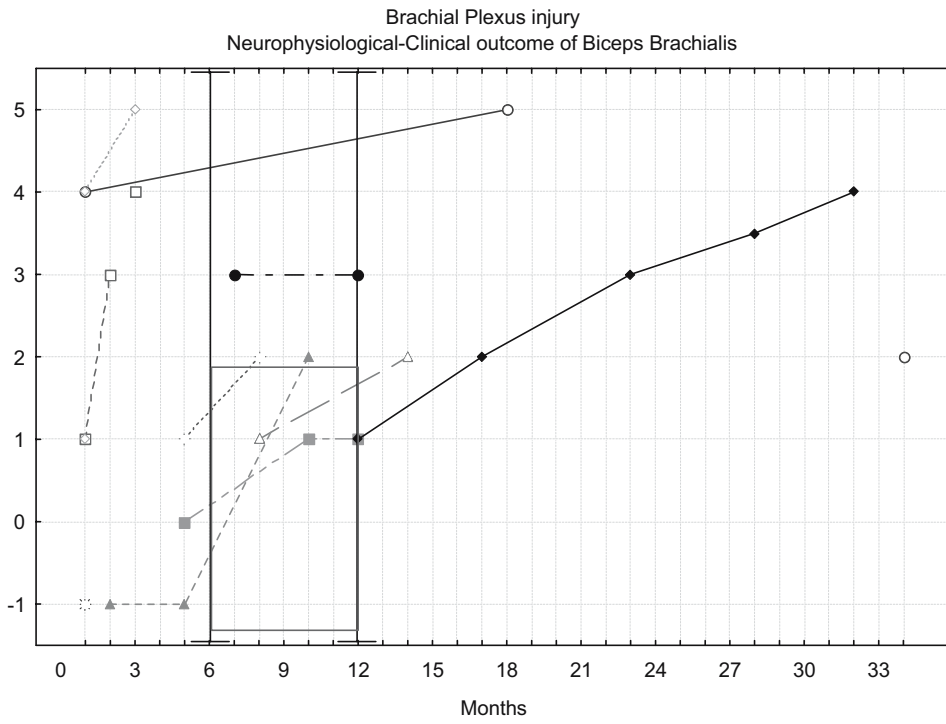


Fig. 2. Shows the neurophysiological-clinical outcome of biceps brachii

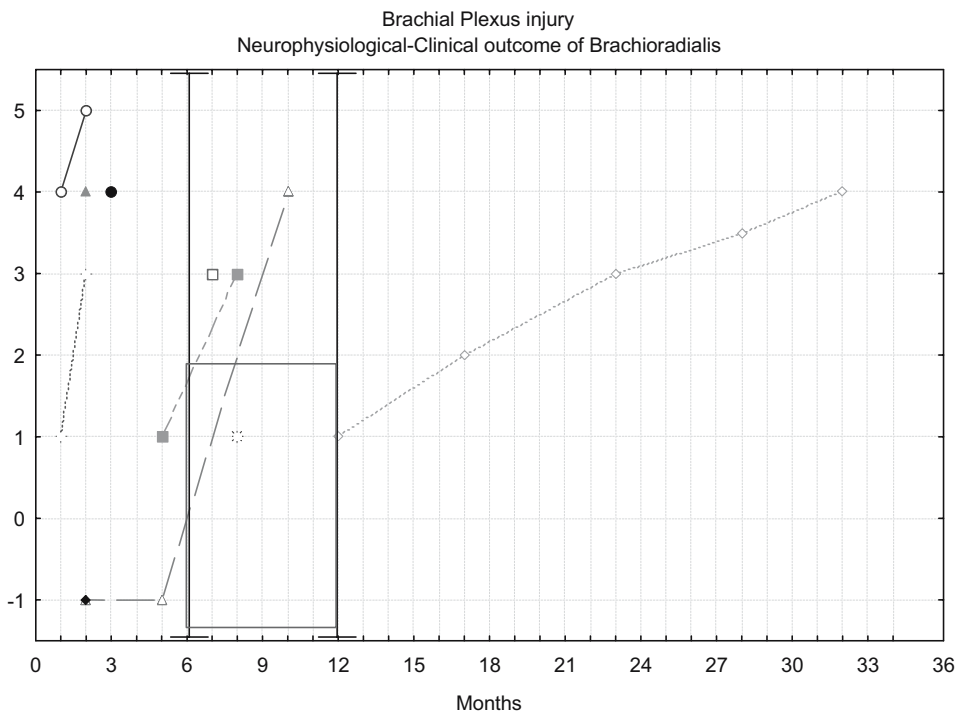


Fig. 3. Shows the neurophysiological-clinical outcome of brachioradialis

peared after 6 months and complete improvement occurred after about 2 years.

Discussion

Brachial plexus lesion is a complex diagnosis. The role of neurophysiological evaluation is crucial in the diagnosis and prognosis of brachial plexus lesion [4–8, 13]. To know the time period needed for recovery after injury is fundamental for the clinical (and surgical) approach, for the rehabilitation program and obviously for the patients. In the scientific literature, there is no systematic clinical evidence of timing of nerve regeneration after trauma, especially regarding brachial plexus. We followed up patients with non-operated brachial plexus injury.

Our results confirm that timing of regeneration is different according to the length of the damaged nerve segment (of course recovery of a short segment is faster than that of a longer one). The current study is preliminary but it provides evidence that a period of 6 months may not be sufficient for reappearance of clinical movement. Moreover, current results confirm that neurophysiological evaluation may be a highly prognostic tool in traumatic nerve lesions. In several cases, even 6 months after injury no clinical movement was present but EMG showed voluntary activity; in all these cases we observed improvement with clinical appearance of voluntary activity.

We hope that our study, together with other data, may provide us a timetable for expected nerve regeneration and possibly criteria for surgical indication. In our opinion this study may represent a preliminary step toward an evidence based therapeutic approach for brachial plexus injury, but further fundamental steps should be taken. When comprehensive information on therapeutic effects and natural evolution of this lesion is available and brought together, an evidence-based standardization of the therapeutic approach to brachial plexus injury will be at hand. Our study does not question the importance and necessity of therapy. In fact, until further data are available, the therapeutic

decision must be taken case by case, on the basis of the clinical picture. We hope our data spurs on more studies, possibly multicentre, about the natural course and the evolution of this disease after surgery.

References

1. Aminoff MJ, Olney RK, Parry GJ, Raskin NH (1988) Relative utility of different electrophysiologic techniques in the evaluation of brachial plexopathies. *Neurology* 38: 546–550
2. Chaudhry V, Cornblath DR (1992) Wallerian degeneration in human nerves: serial electrophysiological studies. *Muscle Nerve* 15: 687–693
3. Deuschl G, Eisen A (1999) Recommendations for the practice of clinical neurophysiology: guidelines of the international federation of clinical neurophysiology, 2nd edn. *Electroencephalography and clinical neurophysiology supplement* n. 52
4. Emre Oge A, Boyaciyani A, Gurvit H, Yazici J, Degirmenci M, Kantemir E (1997) Magnetic nerve root stimulation in two types of brachial plexus injury: segmental demyelination and axonal degeneration. *Muscle Nerve* 20: 823–832
5. Ferrante MA, Wilbourn AJ (1995) The utility of various sensory nerve conduction responses in assessing brachial plexopathies. *Muscle Nerve* 18: 879–889
6. Jones SJ (1979) Investigation of brachial plexus traction lesion by peripheral and spinal somatosensory evoked potentials. *J Neurol Neurosurg Psychiatry* 43: 495–503
7. Jones SJ, Wynn Parry CB, Landi A (1981) Diagnosis of brachial plexus traction lesion by sensory nerve action potentials and somatosensory evoked potentials. *Injury* 12: 376–382
8. Kimura J (1974) F-wave velocity in the central segment of the median and ulnar nerves. *Neurology* 539–546
9. Medical Research Council (1976) Aids to examination of the peripheral nervous system. HMSO, London
10. Miller RG (1987) AAEM minimonograph 32: injury to the peripheral motor nerves. *Muscle Nerve* 10: 698–710
11. Millesi H (1982) Peripheral nerve injuries. Nerve sutures and nerve grafting. *Scand J Plast Reconstr Surg [Suppl]* 19: 25–37
12. Rowland J, Rivara F, Salzberg P, Soderberg R, Maier R, Koepsell T (1996) Motorcycle helmet use and injury outcome and hospitalization costs from crashes in Washington state. *Am J Public Health* Jan 86(1): 41–45
13. Taylor PE (1962) Traumatic intradural avulsion of the nerve roots of the brachial plexus. *Brain* 85: 579–604
14. Wilbourn AJ (1993) Brachial plexus disorders. In: Dick PJ, Thomas PK (eds) *Peripheral neuropathy*, 3rd edn. Saunders, Philadelphia, pp 911–950

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