Journal of Electromyography and Kinesiology 24 (2014) 910–916

Contents lists available at ScienceDirect

Journal of Electromyography and Kinesiology

journal homepage: www.elsevier.com/locate/jelekin

Motor unit number estimation and quantitative needle electromyography in stroke patients

Ioanna Kouzi [a], Eftichia Trachani [a], Evangelos Anagnostou [a], Christina-Anastasia Rapidi [b], John Ellul [a], George C. Sakellaropoulos [c], Elisabeth Chroni [a,*]

[a] Departments of Neurology, School of Medicine, University of Patras, Patras, Greece
[b] Departments of Spinal Cord Lesions Rehabilitation, School of Medicine, University of Patras, Patras, Greece
[c] Departments of Medical Physics, School of Medicine, University of Patras, Patras, Greece

ARTICLE INFO

Article history:
Received 21 May 2014
Received in revised form 6 September 2014
Accepted 8 September 2014

Keywords:
Motor units number estimation
Electromyography
Motor unit potentials
Stroke
Hemiparesis
Upper and lower motor neuron

ABSTRACT

Objective: To evaluate the effect of upper motor neuron damage upon motor units' function by means of two separate and supplementary electrophysiological methods.
Methods: The abductor digiti minimi muscle of the non-paretic and the paretic side was studied in forty-six stroke patients with (a) motor unit number estimation (MUNE) – adapted multiple point stimulation method and (b) computerized quantitative needle electromyography (EMG) assessing the configuration of voluntary recruited motor unit potentials. Main outcome comparisons were focused on differences between non-paretic and paretic side.
Results: On the affected hands mean MUNE value was significantly lower and mean area of the surface recorded single motor unit potentials was significantly larger than the corresponding ones on the non-paretic hands. EMG findings did not reveal remarkable differences between the two sides. Neither severity nor chronicity of stroke was related to MUNE or EMG parameters.
Discussion: MUNE results, which suggested reduced motor unit numbers in stroke patients, in conjunction with the normal EMG features in these same muscles has given rise to different interpretations. In a clinical setting, reinnervation type changes in the EMG similar to that occurring in neuronopathies or axonal neuropathies should not be expected in muscles with central neurogenic lesion.

1. Introduction

The terms "plastic changes" or "plasticity" are collectively used to describe the numerous biological adaptations that follow a focal injury to nervous tissue. In the central nervous system, plasticity emerges as a result of many sub-processes such as increased activity in cortical areas adjacent to injured ones, recruitment of neurons not ordinarily involved in an activity, expansion of cortical representation maps and activation of subcortical pattern generators (Cramer, 2008; Dobkin, 2012). In the peripheral nervous system, functional recovery takes place either via remyelination and regrowth of the severed axons, or through sprouting of axonal branches from neighboring intact neurons that reinnervate some of the denervated muscle fibers (Brown et al., 2013). Plastic changes at the interface between central and peripheral nervous system, as is the case with anterior horn motoneurons, are less well understood. At this point, where a single pyramidal neuron projects to spinal motoneurons for several muscles, the complex and overlapping corticospinal innervation (Graziano et al., 2002), has to be taken into account as a mechanism of neural redundancy able to support plastic recovery. Moreover, it has been convincingly demonstrated that membrane properties of spinal motoneurons undergo profound changes after disconnection from the primary motor cortex (Jankelowitz et al., 2007). The role of this phenomenon in plastic processes remains speculative.

From distant to recent past various neurophysiological approaches have been used to investigate the functional state of lower motor neurons and their motor units in patients with hemiparesis or hemiplegia due to stroke, which is a suitable model to study the effect of upper motor neuron damage upon locomotion (Johnson et al., 1975; Chang, 1998; Lukacs, 2005; Arasaki et al., 2006). Mc Comas et al. (1971) reported 50% decrease of the functioning motor units on the hemiplegic side of stroke patients. More recently, Hara et al. (2000, 2004) have found a decreased number

* Corresponding author at: Department of Neurology, School of Medicine, University of Patras, Rio Patras 26504, Greece. Tel.: +30 2610 999485; fax: +30 2610 993949.
E-mail address: echroni@yahoo.com (E. Chroni).

http://dx.doi.org/10.1016/j.jelekin.2014.09.006
1050-6411/

of functional motor units already in the second week after stroke by means of F-wave Motor Unit Number Estimation (MUNE) method. Alternatively, electromyographic (EMG) examination of muscles in the hemiparetic side has repeatedly demonstrated fibrillation potentials and positive waves at the early phase of a cerebrovascular accident (Johnson et al., 1975; Benecke et al., 1983) Specific EMG techniques such as single-fiber have been employed by other researchers to explore the post stroke rearrangement of the lower motor neuron pool (Chang, 1998; Lukacs et al., 2009) and their findings supported the notion of reinnervation process of paretic muscles. On the whole, clinical studies using diverse approaches have showed functional and even structural alterations of the neuromuscular apparatus secondary to upper motor neuron damage. However, interpretation of the results of one study cannot be advocated by those of others since direct comparison between studies is not feasible due to differences in methodology applied and in the clinical features of participants.

The aim of this study was to provide an approximate estimation of electrically evoked motor unit using the modified MUNE method (Doherty and Brown, 1993; Albrecht and Kuntzer, 2004) and connect these calculations with the findings of EMG signal analysis of voluntary activated motor units in a group of stroke patients with variable severity of muscle weakness and variable time lapse from the symptoms' onset. To the best of our knowledge, no combined MUNE and EMG study in a homogenous post-stroke patient group has been reported.

2. Subjects and methods

2.1. Subjects

The study group consisted of 46 right-handed patients (32 men, 14 women) aged 35–82 years (mean ± SD, 59.2 ± 12.6 years) who suffered from unilateral motor weakness related to a cerebrovascular accident. Exclusion criteria were known peripheral nervous system disorders or risk factors for peripheral neuropathy, severe cognitive impairment, multiple strokes in the past history or bilateral weakness of any cause. According to the medical records, all patients upon stroke manifestations were hospitalized and underwent brain computerized tomography or/and magnetic resonance imaging which confirmed the existence of a recent, unilateral and unique lesion that corresponded to the patients' acute clinical deficit. Forty-two patients had an ischemic and 4 a hemorrhagic cerebral infarction; in 25 patients the lesion was cortical and in 21 capsular; it was located in the right hemisphere in 25 and in the left in 21 patients. All subjects gave written informed consent for participation in the study which was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the Department of Neurology of the University of Patras (no of approval 25, 17.6.2008).

2.2. Methods

2.2.1. Clinical examination

On the recruitment day, a brief medical history was taken and the duration of hemiparesis was noted in all patients. They were clinically examined by a neurologist (I.K.) to determine Scandinavian Stroke Scale (SSS) long term scores (total score ranged from 0 to 58, with the lower score indicating more severe motor disability) (Group, 1985). Explicitly, maximal voluntary strength of hypothenar musculature on both sides was graded on the Medical Research Council (MRC)-scale (0 = no visible muscle contraction, 5 = normal strength) and expressed in kg using a handgrip dynamometer.

2.2.2. Routine electrophysiological examination

For all studies, a two-channel Keypoint ver. 3.25 electromyographic apparatus (Medtronic-Dantec Electronics, Sakovlunde, Denmark) was employed. Patients were instructed to remain relaxed in the recumbent position throughout the examination. Prior to electrode placement, the skin of the examined regions was cleaned by abrasive paste and the limb temperature was maintained at 33 °C ± 0.5 °C throughout the recording procedure. Firstly, the possibility of superimposed polyneuropathy or ulnar mononeuropathy was excluded in all patients by the following electrophysiological tests: a. Motor conduction of the ulnar nerve using a bipolar saline-soaked felt pad electrode (6 mm-diameter cathode, 23 mm distal to the 6 mm diameter anode) for nerve stimulation at the wrist, below elbow and above elbow level and a surface bar electrode (2 × 10 mm stainless steel discs fixed in a plastic mount with a 30 mm centre-to-centre distance) placed over the motor point of the Abductor Digiti Minimi (ADM) muscle (active electrode set at the mid-point of a line drawn from the 5th metacarpophalangeal joint to the pisiform bone) for recording the Compound Muscle Action Potential (CMAP). Slowing of motor conduction across the elbow segment as compared to the distal segment by more than 10 m s^{-1} or a drop of CMAP amplitude higher than 25% across the elbow were criteria used to identify a focal ulnar neuropathy; b. Sensory conduction of the ulnar nerve (orthodromic technique, stimulation of the 5th finger with digital ring electrodes and recording with the surface bar electrode placed on the ulnar nerve at the wrist) and the sural nerve (antidromic technique, stimulation at the posterolateral aspect of the lower third of the leg and recording below and behind the lateral malleolus using the same electrodes as for the motor conduction).

2.2.3. MUNE

MUNE using the adapted multiple point stimulation technique was performed (Wang and Delwaide, 1995; Albrecht and Kuntzer, 2004). The baseline-to-negative peak area of the CMAP of the ADM muscle elicited by supramaximal stimulation of the ulnar nerve at the wrist was measured. Subsequently, the ulnar nerve was stimulated at very low intensity just sufficient to elicit a quantal response corresponding to a single Surface recorded motor unit potential (S-MUP). According to previous practice, S-MUPs smaller than 25 μV ms were not assessed (Daube, 2006). Starting from the distal end of the wrist, the stimulating electrode was slightly moved (in 1 cm distance proximally) along the nerve and if necessary, sites around the elbow were also considered in order to obtain enough quantal responses. In each nerve 4–6 stimulation points were used. At every stimulation point, the stimulus intensity gradually increased (approximately 1 mA increments) in order to obtain 1, 2 or maximum 3 steps, each differed from the previous one by $\geqslant$25μV ms in area and the baseline-to-negative peak area of the higher muscle response to that particular point, consisted of 1–3 single S-MUPs, was measured (Fig. 1). To ensure that a step was unique, it was repeated three times and the corresponding responses were identical when examined in the superimposition mode. Thus, by stimulating a nerve at 4–6 different points, measurements for a total of 10 morphologically distinct motor units per muscle were made in the following way: The summated area (μV ms) was calculated by adding the compound S-MUPs areas from all stimulation points. Only one value, the highest, from each stimulation point irrespective of the number of steps included in this response was used. The mean area of single S-MUPs was then estimated by dividing the summated area by 10 i.e. the number of the involved motor units. To yield the MUNE, the previously obtained maximal CMAP area was divided by the mean area of single S-MUP. In all patients, this procedure was completed for both the paretic and the non-paretic hand, which served as control. For all evoked responses, a constant current stimulator delivering