Journal of Vascular and Interventional Neurology
Official journal of Zeenat Qureshi Stroke Research Center

Journal of Vascular and Interventional Neurology

Abnormal spontaneous muscle activity in plegic limb appears to initiate distal to the upper motor neuron: a case report in a stroke patient

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Abstract

Objective: To study the effect of the cutaneous silent period (CSP) on spontaneous muscle activity occurring after an upper motor injury from stroke, with a goal of developing an insight into the origin of the pathological activity.

Methods: A patient with an acute right centrum semiovale ischemic stroke had left hemiparesis. Fibrillation potentials and positive sharp waves were recorded in several left arm muscles. CSP silent period studies were performed in both arms.

Results: The CSP inhibited the volitional activity in the unaffected right arm. In the plegic left arm, fibrillation potentials and positive sharp waves persisted during the time period during which the CSP would have been expected, based upon the right-sided studies.

Conclusions: Spontaneous activity after a cerebrovascular accident was resistant to inhibition from CSP. These findings suggest that the localization of the origin of the spontaneous activity is distal to the upper motor neuron. A confirmatory study with more patients and in a variety of stroke subtypes would strengthen this conclusion.

Keywords

stroke; cutaneous silent period; fibrillation potential; positive sharp wave; upper motor neuron

Introduction

Fibrillation potentials and positive sharp waves may occur after an upper motor neuron injury such as in hemiplegia of vascular origin and in paraplegia [1–4]. The site of generation of this pathological spontaneous activity has not been established. Theories that have proposed are (1) muscle membrane hyperexcitability originates from the upper motor neuron lesion itself [5] or (2) the generator is at a distal localization such as the nerve terminal, neuromuscular junction, or muscle [3,6–10].

The cutaneous silent period (CSP) is a transient suppression of electromyography (EMG) activity during an ongoing sustained voluntary muscle contraction following strong stimulation of a sensory or mixed cutaneous nerve [11]. Thinly myelinated fibers are considered to be the afferent pathway carrying the impulses of the CSP [11]. These cutaneous afferents relay to an unknown spinal cord circuit capable of producing a strong and secure post-synaptic inhibition of motor neurons [11]. In this report, we studied the effect of the CSP on spontaneous muscle activity occurring after an upper motor injury from stroke, with a goal of developing an insight into the origin of the pathological activity.

Methods

A TECA Synergy (Viasys Healthcare, Madison WI) EMG system was used in this study. Sweep duration was 20 ms/division, sensitivity was 500 μV/division, and filter settings were 20 Hz–5 kHz. Stimulation was performed by ring electrodes at the index finger for the median sensory CSP, at the fifth finger for ulnar sensory CSP, and by a bar electrode over the median and ulnar nerves at the wrist for mixed median and ulnar CSP,

Published February, 2013.

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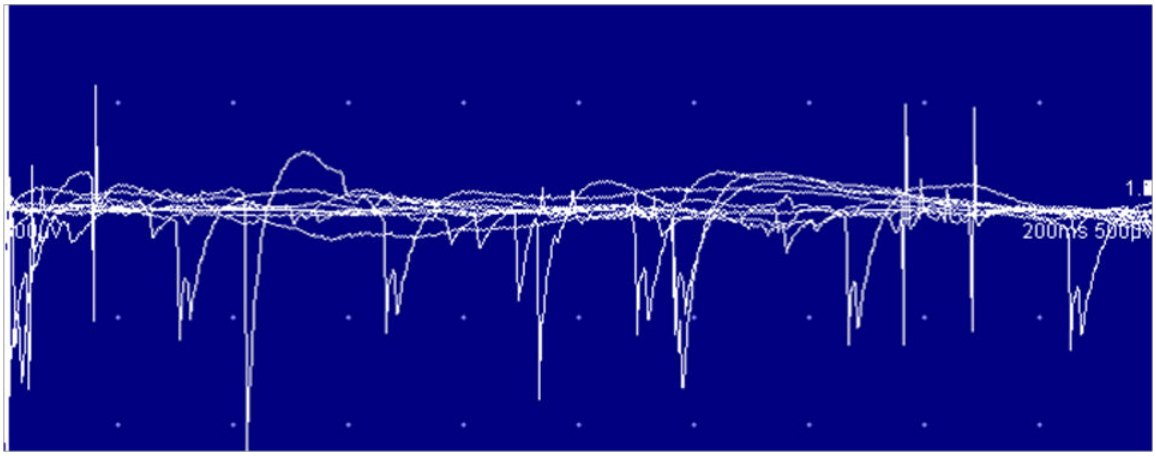

Figure 1. Left ulnar sensory cutaneous silent period recording from the abductor digiti minimi: fibrillation potentials and positive sharp waves persist.

respectively. Recording was performed using a concentric needle electrode in the abductor pollicis brevis muscle (for the median sensory and mixed CSP) and the abductor digiti minimi muscle (for the ulnar sensory and mixed CSP) in the paralytic limb. A similar procedure was performed in the unaffected side using surface disc electrodes instead of a needle electrode. A single electrical stimulation was applied with an intensity of 15 times the sensory threshold and with a duration of 0.5 ms. Five consecutive silent period responses were superimposed. A maximum sustained isometric contraction was requested from the patient in the normal upper extremity.

One patient was evaluated. He was a 67-year-old right handed man with no significant past-medical history who was admitted through the emergency room with sudden onset of slurred speech and left side weakness. On admission, neurological examination demonstrated left hemiparesis including left facial weakness. Using the Medical Research Council scale, his muscle strength was 4/5 in left upper extremity and progressed to 0/5 1 week after his admission. In the left lower extremity, his muscle strength was 4/5.

MRI of the head with and without contrast demonstrated an acute right centrum semi-ovale ischemic stroke. Clopidogrel therapy and daily physical therapy were initiated without substantial improvement. He remained hospitalized for approximately 14 weeks related to placement issues. Approximately 10 weeks after his admission, the patient reported increasing left shoulder pain. MRI of the cervical spine did not reveal nerve root or spinal cord impingement. An electrodiagnostic study was requested to assess for left upper extremity nerve dysfunction approximately 12 weeks after admission. At that time, the patient's neurological examination demonstrated left spastic hemiparesis with muscle strength of 0/5 in left upper extremity and 4/5 in the left lower extremity.

Results

Nerve conduction studies and needle EMG were performed in both upper extremities. Nerve conduction studies demonstrated normal motor, sensory, and F-wave responses in bilateral ulnar and median nerves.

Needle EMG demonstrated diffuse fibrillations and positive sharp waves in the left biceps, left abductor pollicis brevis, left abductor digiti minimi, left flexor carpi ulnaris, and flexor carpi radialis muscles. Pathological spontaneous activity was more intense in distal than in proximal muscles. No voluntary motor unit activity was recorded as the patient was totally monoplegic in his left arm.

In the right arm, the CSPs were present. In the left arm, the sensory and mixed CSPs were unobtainable as there was no voluntary motor unit activity elicited. Fibrillation potentials and positive sharp waves persisted during the time period during which the CSP would have been expected, based upon the right sided studies (see Figure 1). The patient was treated with focal injection of botulinium toxin type A with some improvement of muscle spasticity.

Discussion

Fibrillation potentials and positive sharp waves may occur in hemiplegic muscles within 2–3 weeks after a cerebrovascular accident and may last up to 1 year [3,5].

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This activity is more prominent in the arms than the legs and is more prominent in distal muscles than proximal muscles [3,5]. The abnormal spontaneous activity usually progressively decreases until totally disappearing paralleling motor function recovery or the development of spasticity [5]. In our patient, fibrillation potentials and positive sharp waves were more intense in distal left upper extremities muscles (abductor pollicis brevis and abductor digiti minimi) than in more proximal muscles (biceps, flexor carpi ulnaris, and flexor carpi radialis). Abnormal spontaneous activity was not recorded in the left triceps, left deltoid, and left extensor digitorum communis muscles, in which spasticity was notable.

In our patient, several lines of reasoning suggest that the abnormal spontaneous activity was related to the cerebrovascular accident, as opposed to an independent peripheral nerve lesion: (1) the distribution of the spontaneous activity did not follow a radicular or peripheral nerve pattern. The abnormal spontaneous activity did predominate in distal muscles, where paresis usually predominates in upper motor neuron dysfunction. (2) The normal nerve conduction studies in the left upper extremity exclude left brachial plexopathy, compressive neuropathies, or other neuropathies of the left upper extremity. (3) A cervical MRI study did not reveal any imaging suggestion of radiculopathy or myelopathy.

The salient finding in our study is that the fibrillation potentials and positive sharp waves were resistant to the robust motor inhibition induced by CSP. This suggests that the origin of the muscle membrane hyperexcitability has a localization that is distal to the upper motor neuron. Our study does not address whether the localization is at the level of the peripheral nerve, neuromuscular junction, or in the paralyzed muscle.

A potential limitation of this study is that the pathway that mediates the CSP is not clearly known. It is conceivable that this pathway could be altered by the cerebrovascular accident itself [12]. A study involving more patients with different areas of cerebral involvement would aid in excluding this potential confounding variable.

To the best of our knowledge, this is the first report of the use of the CSP technique to study pathological spontaneous muscle activity after a stroke. Several reports have advanced the theory of trans-synaptic degeneration of the lower motor neurons to explain the occurrence of fibrillation potentials and positive sharp waves after a stroke [3,5,9,13]. The loss of synaptic input from upper motor neurons may cause disturbance of axonal transport in the inactive anterior horn cells which may lead disturb neuromuscular transmission and axonal degeneration [13]. This is supported by morphological muscle rearrangement and abnormal jitter observed after stroke [7,9].

Conclusion

Cutaneous silent period has been shown to inhibit the voluntary upper motor neuron activity. In this study of one patient with CVA and EMG spontaneous activity, we demonstrated that the CSP inhibited upper motor neuron activity in the unaffected arm, but did not inhibit the spontaneous activity present in the affected arm. This suggests that the origin of the spontaneous activity is distal to the upper motor neuron.

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