CENTRAL ENHANCEMENT OF EVOKED ELECTROMYOGRAPHIC MONITORING OF NEUROMUSCULAR FUNCTION†

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SUMMARY

Central neural influences on neuromuscular transmission may explain the frequent failure of evoked electromyographic (EEMG) responses to return to control values during offset of neuromuscular block. This study, performed in conscious subjects, did not demonstrate any change in EEMG response of either the first dorsal interosseous muscle during onset of ulnar nerve block or the flexor hallucis brevis during onset of subarachnoid block. It is concluded that central enhancement of EEMG response via a neural mechanism does not explain the observed failure of EEMG monitoring of neuromuscular block.

KEY WORDS


Evoked electromyographic (EEMG) monitoring of neuromuscular block during anaesthesia produces unreliable results in 5–42% of cases [1–3]. This is usually because of a failure of the first response of the train-of-four to return to the control value during offset of neuromuscular block, although the response occasionally returns to values greater than control. The reasons for this are unclear, but one possible explanation is the modification during anaesthesia of a central mechanism responsible for “muscle reactivity” [4]. This hypothesis has not been subject to experimental validation, in spite of its potential importance. Central enhancement of EEMG has been quoted as an explanation for the failure of EEMG monitoring [2, 3], although the neuroanatomical basis for the effect has not been defined.

This study was designed to test this hypothesis.

SUBJECTS AND METHODS

The study of Paloheimo and Rantala [4] was repeated in so far as the brief details in the report would permit. Electromyographic monitoring was performed using two Datex NMT100 Relaxographs; both machines gave identical responses on an electronic EEMG simulator and stimulus current monitor. The skin at the monitoring site was prepared by rubbing vigorously for 30 s with a gauze soaked in isopropyl alcohol. The Relaxographs were attached to the monitoring site via foam-based electrodes (Nikomed 4570); these electrodes have been found to be better than the electrodes supplied with the Relaxograph, producing stable EEMG records with minimal movement-related artefact [author’s unpublished observations]. The Relaxograph leads were taped with “Micropore” to the limb being monitored in order to reduce artefact from cable stresses. Calibration of the Relaxographs was performed in all cases with the subject awake, before any intervention. A supramaximal response was achieved in all subjects before the study began.

Volunteer study

Five adult males agreed to participate in this investigation. All were ASA I, devoid of neuromuscular disease and not receiving any medication. Electromyographic monitoring was performed on both arms; stimulation of the ulnar...
nerves at the wrist produced responses recorded over the first dorsal interosseous muscles. In order to reduce movement artefact, each hand was held in a fist, thumb on the outside, by a piece of 10-cm adhesive strapping. The elbows were supported on cushions with the forearms resting on the volunteer’s chest. This position allowed easy access to the ulnar nerves at the elbow, without disturbing the EEMG monitoring. Both Relaxographs were calibrated and recordings of the EEMG were made from both arms for 10 min. Subsequently, an ulnar nerve block was performed at one elbow using 6 ml of 2% lignocaine with adrenaline 1:200,000 (Xylocaine with adrenaline, Astra). This volume and concentration were chosen to ensure a dense sensory and motor block of the ulnar nerve. Recordings of EEMG from both arms were continued for 30 min after the nerve block.

**Patient study**

Twenty patients undergoing transurethral resection of the prostate under subarachnoid block were visited the night before surgery; supramaximal stimulation of the posterior tibial nerve was demonstrated and verbal consent for the study was obtained. Only 15 patients were enrolled into the study because five declined to participate after the demonstration of nerve stimulation. Premedication with temazepam 10–20 mg was administered 90–120 min before operation. Electromyographic monitoring was performed on the left leg: stimulation of the posterior tibial nerve at the medial malleolus produced a response recorded over the flexor hallucis brevis. The Relaxograph was calibrated and a control recording made for 5 min. The patient was turned to the left lateral position for induction of subarachnoid block, which was achieved with 0.5% bupivacaine in 8% glucose 3.5 ml (Marcain heavy, Astra) injected at the L2–3 or L3–4 interspace. Patients were returned immediately to the supine position and the recording was continued for a further 30 min, or until surgery was completed.

**RESULTS**

Clinically complete motor and sensory paralysis of the ulnar nerve was obtained in all five volunteer subjects; although the sensation of nerve stimulation did not disappear on the blocked side, its intensity was diminished mildly. In the patient group, a clinically complete, bilateral motor and sensory block was produced to at least T10 in every individual; the subjects ceased to feel the nerve stimuli 10–15 min after induction of the subarachnoid block.

Table I shows the EEMG responses measured during the patient study. There was no change in the EEMG response during onset of conduction block in each of the study groups, and no change in EEMG response in the control arms of the volunteers. Three patients (J.G., C.G.T. and F.W.) exhibited changes in EEMG response related to movement during positioning for lumbar puncture (fig. 1). These changes were abrupt, and temporally related to changes in position.

**DISCUSSION**

In contrast with the work of Paloheimo and Rantala [4], this investigation has not demonstrated any change in the EEMG response during onset of either subarachnoid block or ulnar nerve block. In their abstract, Paloheimo and Rantala suggested that complete motor block (defined as abolition of spontaneous EMG activity) was required to demonstrate the full effect of central enhancement. Assessment of the spontaneous
EMG was not available for the study reported here. However, the nerve conduction blocks were complete clinically, and this study has not demonstrated any central enhancement effect. The lack of agreement with the observations of Paloheimo and Rantala requires discussion.

The experimental details in the original abstract were brief, but there were small differences in methodology between the two studies [Paloheimo M., personal communication].

**Electrodes.** Paloheimo used the electrodes supplied by Datex Instrumentarium for use with the Relaxograph, and took no special precautions to avoid movement-related artefacts. As the only changes in EEMG response reported here occurred during patient movement, it may be significant that different electrodes were used in the two studies. In a series of 100 consecutive patients, the Datex electrodes were associated with a failure of the EEMG to return to within ±10% of the control value in 40.5% of cases, while the Nikomed electrodes exhibited only an 8% failure rate; in addition, the Datex electrodes were associated with a 16% failure to achieve satisfactory calibration of the Relaxograph at the first attempt [author’s unpublished observations].

**Nerve block.** Paloheimo studied six patients undergoing transurethral resection of prostate; subarachnoid block was obtained with isobaric 0.5% bupivacaine, and injection was made in the sitting position. The use of a different lumbar puncture technique, and hyperbaric instead of isobaric bupivacaine, is unlikely to have influenced the results, as a dense block adequate for surgery was obtained in both studies.

**EEMG monitoring.** Paloheimo used thenar or hypothenar EEMG in his volunteer group, and did not strap the hand. However, movement artefact occurs readily if the hand is not immobilized for EEMG monitoring (Datex application notes). Monitoring of flexor hallucis brevis EEMG was the same in both studies.

The lower motor neurone is the final common pathway for all neural traffic which converges on...
the motor end-plate [5]. Supramaximal stimulation of a peripheral nerve should excite all the lower motor neurones within that nerve, ensuring maximal response at the motor end-plate. This assumes that the nerve threshold for stimulation does not alter significantly after the stimulus current has been set. Any central motor mechanism which exerts its effect proximal to the site of nerve stimulation is, therefore, unlikely to modify the response of the motor end-plate to supramaximal external stimulation. Central modulation of plasma adrenaline concentration may modify neuromuscular performance [6] and nerve depolarization thresholds [7] under conditions of extreme stress, but these effects are small at physiological concentrations of adrenaline [6].

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REFERENCES