Are changes in the evoked electromyogram during anaesthesia without neuromuscular blocking agents caused by failure of supramaximal nerve stimulation?†

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Summary

The evoked electromyogram often decreases during anaesthesia in the absence of neuromuscular block. We have measured the electromyogram of the first dorsal interosseous muscle evoked by train-of-four stimulation of the ulnar nerve in 63 patients undergoing anaesthesia for minor surgery. We used Medicotest P-00-S electrodes, a Datex Relaxograph and a current sink in the stimulating leads in parallel with the current path through the patient. The current sink was used to shunt some of the maximum available output current from the Relaxograph while maintaining the supramaximal stimulus current passing through the patient. After 30 min of anaesthesia, when the muscle response to train-of-four was stable, the ulnar nerve stimulus current was increased by reducing the proportion shunted through the current sink. The electromyographic response did not change during the study in 13 patients. In the remaining 50 patients, the response decreased to 78.4% (SD 27.1%, range 7.5–95.0%) of baseline values over the first 20 min of anaesthesia. In 22 of these patients, the electromyographic response increased from 71.4 (SD 22.6)% to 92.3 (9.5)% of baseline responses when the stimulus current was increased by 12.3 (2.4) mA, while in the remaining 28 patients the response decreased to 83.7 (10.6)% and did not increase with increasing stimulus current. These results suggest that loss of supramaximal stimulation is partly responsible for the observed changes in the evoked electromyogram during anaesthesia. (Br. J. Anaesth. 1998; 81: 902–904).

Keywords: measurement techniques, electromyography; measurement techniques, neuromuscular block; neuromuscular block, measurement of response

The evoked electromyogram (EEMG) often fails to return to baseline values during offset of neuromuscular block. In patients who have not received a neuromuscular blocking drug, the EEMG response may also decrease to approximately 80% of control values over the first 20 min of anaesthesia, but the response does not decrease with time in awake subjects. Improper fixation of the recording electrodes and changes in forearm position may alter the EEMG response, possibly by alteration of the position of the stimulating electrodes relative to the ulnar nerve. If movement of the stimulating electrodes results in failure of supramaximal nerve stimulation, by simply increasing the stimulus current the EEMG response should increase towards the control value set at initial calibration.

To study the effect of stimulus current on the EEMG response during anaesthesia, we wished to be able to increase the output current from the nerve stimulator of a Relaxograph (Datex, Helsinki) after calibration, without changing the calibration values for the train-of-four responses in the memory of the Relaxograph. The Relaxograph stimulus current can be changed after calibration, but only in discrete steps of varying magnitude. Therefore, we decided to use a calibrated adjustable current sink in parallel with the stimulator output leads. This adjustable current sink was designed by Mr F. Clewlow along conventional lines, to have a sink capability of 40 mA at a maximum input voltage of 400 V and be capable of a square wave response to a current pulse of 100 μs duration. Its performance was independent of stimulus electrode impedance. Details of the device are available from the authors on request. The current sink shunts a variable proportion of the Relaxograph output current away from the patient during calibration, while the supramaximal stimulus current reaching the patient is maintained (fig. 1). The proportion of shunted current can later be reduced, increasing the current passing through the stimulating electrodes to the patient.

Patients and methods

After obtaining approval from Southampton Joint Ethics Committee, we studied 63 adult patients, ASA I or II, body mass index (weight/height²) <27, undergoing minor surgery which did not require a neuromuscular blocking drug during anaesthesia. Premedication was given at the discretion of the responsible anaesthetist.

We recorded the EEMG over the belly of the first dorsal interosseous muscle with the Relaxograph and Medicotest P-00-S electrodes. The indifferent electrode was on the proximal phalanx of the index finger. We stimulated the ulnar nerve at the wrist, with the current passing through the stimulating electrodes relative to the ulnar nerve. If movement of the stimulating electrodes results in failure of supramaximal nerve stimulation, by simply increasing the stimulus current the EEMG response should increase towards the control value set at initial calibration.

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the elbow flexed to 30°, and was loosely fixed in position with adhesive tape. The arm was left exposed on the armboard; theatre temperature was regulated to 22°C. Routine monitoring comprised pulse oximetry, non-invasive arterial pressure and ECG.

Anaesthesia was induced with propofol 2–2.5 mg kg⁻¹, and a laryngeal mask was inserted. Anaesthesia was maintained with 1–1.5% isoflurane and 66% nitrous oxide in oxygen, with the patient breathing spontaneously via a Mapleson A system. We started EEMG monitoring at the loss of the eyelash reflex during induction of anaesthesia. The Relaxograph was switched on and the automatic calibration routine determined the supramaximal stimulus current. The difference between the supramaximal stimulus current and the 70 mA maximum output from the Relaxograph was then dialled on the control knob of the current sink. We then recalibrated the Relaxograph to ensure that the output current from the Relaxograph was close to 70 mA while the original supramaximal stimulus current to the patient was maintained, with the maximum possible current passing through the current sink. We did not measure the actual stimulus current to the patient, but the Relaxograph and current sink had both been calibrated in the laboratory before the study began. The patient was then transferred carefully from the anaesthetic room to the operating theatre. The Relaxograph continued to measure the train-of-four response every 20 s for the duration of the study. After 30 min we adjusted the current sink to increase the stimulus current to the patient in an attempt to restore the T1 response to 100%. We repeated the Relaxograph calibration routine, without using the current sink, at the end of monitoring in 45 of the patients.

We analysed the data using a computer spreadsheet (Excel v4.0, Microsoft). Results are presented as mean (sd). We regarded a 5% change in either direction from baseline EEMG responses as a clinically significant change.

Results

Patients were aged 22–68 yr, with a 50:50 male:female ratio. The T1 response remained stable within ±5% of the control response in 13 of the 63 patients. In these patients the T1 response did not increase when additional stimulus current was passed through the electrodes, and the supramaximal stimulus current did not change between initial calibration and recalibration at the end of the study.

In 50 patients the T1 response decreased to 78.4 (27.1)% of baseline responses over the first 20 min of anaesthesia. These 50 patients were divided into two groups (A and B) according to their response to increased stimulus current:

Group A (22 of 50 patients). Mean T1 response decreased to 71.4 (22.6)% (range 7.5–95.0%) of control over the first 20 min of anaesthesia and remained stable thereafter. Increasing the amount of current passing through the stimulus electrodes by 12.3 (2.4) mA increased the T1 response to 92.3 (9.5)% of control (fig. 2). In four of these patients the new supramaximal stimulus current was greater than the 70 mA maximum output from the Relaxograph and could not be determined, while in the other 18 patients the mean supramaximal stimulus current increased from 52.5 mA at initial calibration to 64.8 mA at recalibration.

Group B (28 of 50 patients). Mean T1 response decreased to 83.7 (10.6)% (range 47.5–95.0%) of control over the first 20 min of anaesthesia and remained stable thereafter. Increasing the stimulus current by 12.3 (2.4) mA did not increase the T1 response (fig. 2). The mean supramaximal stimulus current at initial calibration was 47.1 mA, and after recalibration, 49.7 mA.

There was no difference in age, body mass index or sex distribution between the subgroups which could account for these differences in T1 response.

Discussion

The decrease in EEMG response from the calibration baseline was similar to that reported previously. This has largely been ignored in the past, although some workers reject those recordings in which the EEMG response does not return to within 10–15% of baseline values and others allow the EEMG to stabilize for 20 min before recalibrating the response.

![Figure 1](image1.png)

**Figure 1** Operating principle of the current sink. During calibration of the Relaxograph, the sink shunts a variable proportion of the stimulus current to ground, bypassing the patient. The amount of shunted current may later be reduced, increasing the stimulus current to the patient.

![Figure 2](image2.png)

**Figure 2** Mean changes in the evoked electromyographic response during anaesthesia in groups A and B. Stimulus current was increased at 30 min. Error bars represent 95% confidence interval for the mean value.
to 100%. We found that the EEMG response was not often restored to the 100% baseline value with additional stimulus current, although the variability of the response about the mean value was reduced. In those patients in whom the T1 response did not change during the study, we cannot assume that there was no change in the maximal nerve stimulation current, as any change may simply be hidden within the 20% margin of the supramaximal stimulus current.

The decrease in the EEMG response during anaesthesia is unrelated to changes in the electrical impedance of the stimulus or recording electrodes, but is associated with a reduction in the area and amplitude, but not duration, of the evoked compound action potential. This implies either that the decrease in the EEMG response results from a decrease in the number of active motor units, or that the end-plate depolarization current is reduced during anaesthesia.

Increasing the distance between the stimulus electrode and nerve reduces the stimulating current density reaching the nerve by the fourth power of the distance, as the current flows in three dimensions through the tissues, which may allow the effective stimulus current to become submaximal. Movement of the stimulating electrodes relative to the ulnar nerve may result from pronation or supination of the forearm during the procedure, possibly during repositioning of the patient during surgery. Alternatively, the volume of the soft tissues of the forearm may increase as a result of vasodilatation during anaesthesia. Obese patients have greater distances between the stimulating electrodes and nerve than slimmer patients, and therefore obese patients may be more susceptible than thin patients to decreases in the EEMG control response during anaesthesia. We excluded obese patients from our study.

Volatile anaesthetic agents may modify neuromuscular transmission by reducing acetylcholine output from the motor nerve terminal, although routine concentrations of volatile anaesthetics do not modify either the motor nerve or muscle membrane thresholds for depolarization. Isoflurane at concentrations greater than 1.25 MAC and enflurane at concentrations greater than 3.5% induce tetanic fade and depress the mechanical single twitch response by 10% in humans.

The mechanism for this is unclear. More standard concentrations of halothane and isoflurane increase the mechanical twitch response but decrease the EEMG response in humans, but the mechanical response recovered on cessation of anaesthesia while the EEMG response did not.

In summary, the decrease in the control response of the EEMG during anaesthesia was caused, at least in part, by failure of supramaximal stimulation of the motor nerve. However, we cannot explain why increasing the stimulus current increased the T1 response in only 44% of those patients in whom it decreased, and further work is required to provide a complete solution to this problem.

References