THE EFFECT OF TUBOCURARINE ON ULNAR NERVE CONDUCTION VELOCITY

BY

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SUMMARY

Nerve conduction velocity was studied in the ulnar nerve of human subjects during general anaesthesia. An increase in velocity of between 1.9 and 4.8 per cent occurred following the administration of paralytic doses of tubocurarine to subjects receiving nitrous oxide, oxygen and halothane anaesthesia in whom ventilation was controlled. This increase in velocity did not appear to be related to a change of subcutaneous temperature.

The technique of measuring both sensory and motor nerve conduction velocities is well known (Dawson, 1956), and has been applied to study the effects of both anaesthetic agents and muscle relaxants on peripheral nerves (Wyke, 1965; Thornton, Whelpton and Brown, 1968). It was reported by the latter workers that there was a statistically significant increase in nerve conduction velocity following the administration of gallamine triethiodide and this could not be easily explained. The purpose of the work about to be described was (a) to ascertain whether this finding was peculiar to gallamine, or whether there is a change in nerve conduction velocity when other neuromuscular blocking agents such as tubocurarine are used, and (b) to show whether or not changes in temperature were responsible for the change in nerve conduction velocity. It was suggested that the changes noted in the previous study might be related to temperature changes.

METHOD

The left ulnar nerve was chosen for study. Surface electrodes were used and the method of recording was similar to that used by Dawson (1956) and Thornton, Whelpton and Brown (1968). The technique consists of giving a short electrical stimulus to the nerve at the wrist and detecting the combined orthodromic sensory and antidromic motor impulses at the level of the elbow joint. To facilitate measurements of the nerve action potential the nerve was stimulated 10 times/sec for 10 sec and the 100 impulses averaged using an analogue averager of channel width 100 m.sec. This averaged response was then displayed on a storage oscilloscope and photographed using a polaroid camera. The signal latency was measured before and after the injection of tubocurarine, and the results expressed as a percentage change from the pre-injection measurements. Latency was taken as the time interval between electrical stimulus and first negative peak of the action potential. In order to avoid artefacts, blood pressure measurements and intravenous injections were made in the right arm and the left arm strapped to an arm board. The surgical procedures were carried out on the upper abdomen and hence were far enough away from the measuring site so as not to introduce movement artefacts.

Measurements were made on twenty patients, whose ages ranged from 19 to 74 years, and who had no known general or local neurological disorder. In all cases the pre-anaesthetic medication consisted of morphine 10 mg and atropine 0.6 mg given intramuscularly 1 hour pre-operatively. Induction of general anaesthesia was carried out using thiopentone and suxamethonium. Intubation of the larynx was then performed and anaesthesia was maintained using nitrous oxide, oxygen and halothane. Whilst the patient was being positioned on the operating table surface electrodes were attached to the patient; the subcutaneous temperature probe, comprising a sterile needle with a thermistor mounted in the tip, was inserted in the forearm 0.7 cm below the skin surface; a surface temperature probe comprising...
a thermistor bead was taped to the surface of the forearm. The patient was then completely covered with green drapes. After a baseline measurement of signal latency had been taken the patients received a dose of tubocurarine ranging from 30 to 45 mg depending on the physical status of the individual. After this injection, unless diathermy was being used during the measurement period, measurements of ulnar nerve action potential were made at 5-minute intervals. Throughout the operation surface temperature was measured in patients numbered 8–20, and subcutaneous temperature in patients numbered 16–20.

Accuracy of the technique.

The nerve conduction velocity changes reported in this communication are small and therefore it is useful to consider some of the problems and errors associated with latency and velocity measurements. The instrumentation must be checked and calibrated before use, taking particular care with regard to the time base. The time base of the display oscilloscope was calibrated just prior to the first measurement using a standard external signal generator of 0.1 per cent accuracy. As the size and shape of the action potential are functions of both stimulus size (for a submaximal stimulus) and electrode position, supramaximal stimulation was used in all cases. Care was therefore taken to position the monopolar electrode over the ulnar nerve as it passed between the medial epicondyle and the olecranon.

Conduction velocity of the combined orthodromic sensory and antidromic motor impulses of the ulnar nerve have been found to be between 50 and 60 m/sec (Thornton, Whelpton and Brown, 1968). Depending upon the length of nerve studied this conduction velocity will give a signal latency of 3–6 m.sec. In this study the mean latency was 5.0 m.sec. A major error in conduction velocity is due to measurement of the length of nerve studied. In order to avoid this we have not measured the length but expressed our results as a change in time only and have assumed that changes in conduction velocity are inversely proportional to this time.

To minimize errors of interpretation of the recorded display, G.E.W. carried out all the analyses. In order to assess the reproducibility of the technique three normal subjects, under stable laboratory conditions, had their ulnar nerve latencies measured in a similar way to that of the patients in this study. Measurements were made every 5 minutes for a period of 1 hour and the percentage change in latency revealed a standard deviation of 1.8 (36 measurements).

The accuracy of the temperature measurements was limited to the accuracy of the ability to read the scale of the recording thermometer (±0.1°C).

RESULTS

For each patient the latency, which had been derived from the averaged response, was expressed as the percentage change from the value obtained before injection of tubocurarine. As the percentage change is inversely proportional to nerve conduction velocity, the mean percentage change in conduction velocity may be considered. These are shown in figure 1 together with standard errors of the mean. Although twenty patients were studied the maximum number of readings shown in figure 1 is eighteen; this is because surgical diathermy was employed and this sometimes coincided with the time for a measurement.

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The mean of the results in figure 1 appears to peak at 20 minutes. This mean value is significantly different from zero at $P<0.05$. If the readings at 15, 20, and 25 minutes are taken as one collective group then the mean of this group of 1.9 per cent is highly statistically different from zero at $P=0.005$.

Subcutaneous and skin temperature are shown in figure 2 where it can be seen that both fall steadily by a mean maximum of approximately 0.9 deg. C over 1 hour after the first 5 minutes. The small rise in surface temperature over the first 5 minutes is most likely to be due to the placement of drapes; changes of similar magnitude are produced in the laboratory if the theatre procedures of application of electrodes and application of drapes is mimicked. The similar fall in both subcutaneous and surface temperature suggests that under stable conditions the surface temperature changes do reflect subcutaneous changes.

Consider the peak of the curve in figure 1, where the conduction velocity after 20 minutes had increased by a mean of 1.9 per cent, the corresponding subcutaneous temperature had fallen by a mean of about 0.3 deg. C. A fall in nerve temperature is well known to decrease velocity, hence we argue that at 20 minutes the increase in velocity of 1.9 per cent should be greater if the fall in temperature had not occurred.

Figure 3 shows the percentage change in conduction velocity assuming constant temperature conditions. In order to make this correction a temperature velocity coefficient of 4.5 per cent change for each deg. C was assumed. Having corrected the conduction velocity for changes in temperature, the results can be re-expressed. The increase in ulnar nerve conduction velocity after the injection of tubocurarine reaches a maximum of between 1.9 and 4.8 per cent some time between 25 and 35 minutes later.

**DISCUSSION**

The results show that there is an increase in nerve conduction velocity following the administration of the neuromuscular blocking agent tubocurarine. In this respect tubocurarine and gallamine triethiodide (Thornton, Whelpton and Brown, 1968) are similar. The results are different in that for tubocurarine the maximum increase is greater and occurs later. As the velocity/temperature coefficient of nerve conduction velocity used for normalization is open for discussion, these differences should be interpreted with caution. The value used to derive figure 3 was arrived at by taking the mean velocity/temperature coefficients reported by Henriksen (1956) and Johnson and Olsen (1960). Henriksen reported a change of 4.1 per cent per deg. C for the ulnar motor nerve over the temperature range 36–30° C, whilst Johnson and Olsen estimated 5 per cent per deg. C over the
same range. Other values which could have been taken were those of Carpendale (1956) who found a change of 4.9 per cent per deg. C for the ulnar motor nerve (although on three patients), or of Abramson and associates (1969). Abramson and co-workers reported a coefficient for the median nerve over the distance from wrist to thenar eminence and not wrist to elbow. Their coefficients were 2.8 per cent per deg. C for motor and 1 per cent per deg. C for orthodromic sensory impulses. Because of the problem of a reliable velocity/temperature coefficient for the ulnar nerve over the portion wrist to elbow we can only state that the increase lies between 1.9 per cent uncorrected for temperature and 4.9 per cent corrected.

The second question posed in the introduction concerned the effect of temperature on changes in nerve conduction velocity. For temperature to have produced an increase in nerve conduction velocity it would have had to increase. Apart from the artefact at the start, which has been explained in the results, the change in temperature has always been a decrease. Hence it is unlikely that the changes in surface and subcutaneous temperature are responsible for the increase in nerve conduction velocity.

Whilst the changes reported above are small, and in a single case are likely to be difficult to determine because of the size of the errors outlined in the method, they are significant as a group. The increase in ulnar nerve conduction velocity of 1.9 to 4.8 per cent for tubocurarine is similar to those reported for gallamine by Thornton, Whelpton and Brown (1968). The changes are, therefore, not specific to gallamine and the cause of them is still inexplicable.

REFERENCES


