Site of action of 'intravenous regional anaesthesia'

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The technique of 'venous anaesthesia' described by Bier (1908) has recently been revived by Holmes (1963). On clinical grounds he concluded that the lignocaine used to produce the anaesthesia acted on the motor and sensory nerve endings. This suggestion has been examined by electro-physiological studies.

METHOD

Three normal subjects and four volunteer patients were examined. The latter were undergoing surgical operation for benign conditions and were receiving a general anaesthetic.

Motor nerve conduction was examined by stimulation of the ulnar nerve at wrist and elbow. A concentric coaxial needle electrode was inserted into the abductor digiti minimi muscle and the action potential produced by a supramaximal square wave stimulus (amplitude 250 volts; duration 40 μ sec.) applied to the ulnar nerve was displayed on the screen of a calibrated cathode ray oscilloscope. The sweep of the latter was triggered by the stimulus and so the latency of the action potential could be read off directly. Two results were thus obtained: the latency of the action potential following stimulation at the wrist, and the speed of conduction in the elbow-wrist segment of the ulnar nerve.

Sensory nerve excitability was tested by measuring the amplitude of the action potential recorded from the median nerve at the wrist following stimulation of the skin of the thumb by ring electrodes (Dawson, 1956).

In each case the limb was prepared by exsanguination using an Esmarch bandage and occlusion of the arterial supply by a sphygmomanometer cuff. Regional anaesthesia was then produced by injecting 40 ml. of 0.5% lignocaine through a Gordh needle. Control observations were obtained by carrying out the same tests in an ischaemic limb without lignocaine.

The response of the muscle to high frequency stimulation (10-50 c.p.s.) of the nerve was measured in (1) three conscious subjects both with and without lignocaine, and in the latter the observations were repeated following the injection of 1.0 mg. neostigmine; (2) four curarized anaesthetized patients.

RESULTS

EFFECT OF ISCHAEMIA AND REGIONAL ANAESTHESIA ON LATENCY OF MUSCLE ACTION POTENTIAL The results

are shown in Figure 1. In all cases the increase in latency was earlier and greater in the presence of lignocaine. In J.L.J., for example, at 25 min. ischaemia produced an increase of 2 msec. (66%) and ischaemia with lignocaine one of 4.7 msec. (180%).

RATE OF CONDUCTION IN THE ULNAR NERVE TRUNK Slowing of conduction was seen in all cases but that following the addition of lignocaine was not appreciably different from that produced by ischaemia alone (Fig. 2).

SENSORY ACTION POTENTIAL IN THE MEDIAN NERVE Ischaemia alone reduced the amplitude by 80-100% and increased the latency by 50-70% after

FIG. 1. The dotted line illustrates the latency in the distal portion of the ulnar nerve observed in the presence of lignocaine and ischaemia. The continuous line illustrates the effect of ischaemia only.

After 23 minutes the mean latency was 6.73 msec. (S.E. 0.175) for lignocaine and ischaemia and 4.37 msec. (S.E. 0.099) for ischaemia only. Student's *t* test gave a value of $p < 0.001$.

FIG. 2. The dotted line illustrates the ulnar nerve conduction rates in metres per second in the elbow-to-wrist segment in the presence of lignocaine and ischaemia. The continuous line illustrates the effect on conduction rate of ischaemia only.

FIG. 3. The dotted line illustrates the effect of lignocaine and ischaemia on the amplitude of the median nerve sensory potential recorded at the wrist. The continuous line illustrates the effect of ischaemia only.

minutes. The effect of ischaemia plus lignocaine was exactly similar (Figs. 3 and 4).

RESPONSE TO HIGH FREQUENCY STIMULATION In all subjects examined the response to high frequency stimulation (10-50 c.p.s.) and the recovery after it was normal in the ischaemic arm 15-20 min. after application of the sphygmomanometer cuff.

The combination of ischaemia and lignocaine produced an abnormal response (Fig. 5). This consisted of a rapid decay in the size of the action potential and a slow recovery of excitability following stimulation. Similar observations were made in the patients who had received curare alone (the dose of curare was not sufficient to produce complete neuromuscular block).

The injection of neostigmine did not modify in any way the effects of lignocaine. This is to be contrasted with the action of neostigmine on curare-induced neuromuscular block.

At the stage when the abductor digiti minimi failed to contract when the ulnar nerve was stimulated at the wrist by single or repeated square wave pulses, the electrodes were transferred to the muscle belly and action potentials were recorded and muscle contractions observed.

DISCUSSION

These results suggest that lignocaine acts on the peripheral parts of the neuron. The only effect

FIG. 4. The dotted line illustrates the effect of lignocaine and ischaemia on the latency of the median nerve sensory potential recorded at the wrist. The continuous line illustrates the effect of ischaemia only.

FIG. 5. The response to high frequency stimulation (30 c.p.s.) of the ulnar nerve after 15 minutes of (a) ischaemia only and (b) ischaemia with lignocaine.

observed consistently was an increase in the latency of the action potential in the hypothenar muscles following stimulation of the ulnar nerve at the wrist (Fig. 1).

At first sight the effect of lignocaine on neuromuscular conduction resembles that produced in the four patients by curare alone. However, the action of lignocaine is not reversed by neostigmine. The dose of neostigmine used was 1.0 mg. given in 10 ml. normal saline. This volume of vehicle was chosen in an attempt to carry the anticholinesterase throughout the vessels of the forearm. The fluid was massaged out of the superficial veins. The drug was confined to the forearm and the tissue level achieved must have been comparatively high. Harvey (1939) concluded that procaine acted at the preganglionic synapses to diminish acetylcholine production, but at the neuromuscular junction there was likely to be an additional effect, since the response of the muscle to injected acetylcholine was also blocked. If lignocaine acts similarly this would explain the lack of response to neostigmine in our experiments. Jaco and Wood (1944) produced experimental evidence that procaine acts by diminishing the production of acetylcholine at the neuromuscular junction. It may be therefore that local anaesthetics like lignocaine have a dual effect, inhibiting acetylcholine production and opposing its action.

The changes in the action potential in sensory nerves are apparently largely due to ischaemia. However, the anaesthesia was greater and the subjective discomfort (pain and paraesthesiae) less in the presence of lignocaine, even when it was still possible to record a sensory action potential. This suggests that the anaesthetic acts on sensory nerve endings

earlier than on the nerve trunks. A similar effect was shown experimentally in the cat (Diamond, Gray and Inman, 1958). It would seem therefore that stimulating the thumb with ring electrodes (Dawson, 1956) can evoke an action potential in the digital nerves directly rather than through sensory nerve endings.

SUMMARY

The changes in nerve conduction in the arm produced by ischaemia, and the combination of ischaemia and intravenous 0.5% lignocaine, have been studied by electromyography.

Lignocaine appears to produce a block at the neuromuscular junction which is not reversed by resting and acts on sensory nerve endings.

The isolation of the limb from the general circulation restricts the spread of the anaesthetic, producing rapid effects which are not due entirely to ischaemia.

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