NERVE CONDUCTION STUDIES IN REGIONAL INTRAVENOUS ANALGESIA USING 1 PER CENT LIGNOCAINE

BY

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SUMMARY

The effects of regional intravenous analgesia in the upper limb were studied in eight subjects by means of clinical examination and serial nerve conduction techniques. The changes following 1 per cent lignocaine analgesia and ischaemia were compared with those following ischaemia alone; the latter produced analgesia and impairment of conduction at a considerably slower rate than it did when combined with 1 per cent lignocaine. It was found that 1 per cent lignocaine caused a block in conduction of the nerve trunks at the site of maximal concentration of drug in the limb. The proximal site of blockade may be of value in practice when the peripheral sensory nerve endings in the region of the operative site cannot be perfused adequately.

The reintroduction of intravenous regional analgesia by Holmes in 1963 has resulted in its widespread use. Holmes's (1963) suggestion that lignocaine acted on the motor and sensory endings received support from the studies of several workers (Miles et al., 1964; Fleming et al., 1966) and from Adriani (1968) in a recent review. On the other hand, Sorbie and Chacha (1965) concluded, on clinical and electrophysiological grounds, that the local anaesthetic acted mainly on nerve trunks. Most previous workers have employed 0.5 per cent lignocaine in their studies, but clinical experience has demonstrated that 1 per cent lignocaine is also a safe and effective agent in regional intravenous analgesia. The present study was undertaken to investigate the site of action of 1 per cent lignocaine, by means of electrophysiological techniques.

METHODS

Eight healthy male volunteers, aged 21–48 years and weighing 70–97 kg, were investigated. The subjects were divided into two groups. In Group I (5 subjects) a sphygmomanometer cuff was placed on the upper arm, and inflated to 200 mm Hg after the limb had been exsanguinated with a rubber bandage. Twenty ml of 1 per cent lignocaine was immediately injected intravenously through a Mitchell needle inserted into a dorsal hand vein. Electrophysiological studies were then performed on the median nerve in the forearm and its motor and sensory branches in the hand for 30 minutes after injection of lignocaine.

In Group II (3 subjects) the cuff was placed on the forearm 8–10 cm above the level of the wrist. In two subjects the hand and wrist were exsanguinated with a bandage and 10 ml of 1 per cent lignocaine was injected intravenously into a dorsal hand vein. In the third case exsanguination was not performed but the limb was elevated for 3 minutes, the cuff inflated, and lignocaine injected. Electrophysiological studies were performed on the motor and sensory branches of the median nerve in the hand.

In all eight subjects, control observations were made on the opposite limb with ischaemia alone.

Motor and sensory conduction were studied in the median nerve prior to exsanguination of the limb and at 5-minute intervals for 30 minutes after the onset of ischaemia. The muscle action potential was recorded from the abductor pollicis brevis muscle with needle electrodes following supramaximal electrical stimulation of the median nerve at wrist and elbow. The sensory action potential was recorded with surface electrodes following electrical stimulation of the index finger (Dawson, 1956). The electrical stimulation
was a square wave of duration 0.2 msec derived from a Disa Ministim. The recording electrodes were connected to a Tektronix FM122 pre-amplifier and displayed on the upper beam of a Tektronix 502A oscilloscope; a time-scale derived from a Digitimer (Devices Ltd) was displayed on the lower beam. Photographic records were made on 35-mm film.

The skin temperature on the back of the hand was measured with a thermistor and ranged from 31 to 33°C at the commencement of each study. The fall in temperature of the limb following 30 minutes of ischaemia did not exceed 2°C in any of the subjects.

Thirty minutes after injection of lignocaine, the sphygmomanometer cuff was deflated successively for periods of 5, 10 and 15 seconds with periods of reinflation of 30 seconds between each interval of deflation (Merrifield and Carter, 1965). In three subjects of Group I, venous blood was collected from the opposite arm at 30 seconds and 1, 2, 5, 10, 20 and 40 minutes after initial cuff deflation. Plasma lignocaine levels were measured by gas chromatography (Thomas et al., 1969).

**RESULTS**

**Clinical observations.**

Approximately 5 minutes following the injection of lignocaine in Group I there was impairment of pain and tactile sensation along the dorsum of the forearm, proximal to the injection site. In 7–15 minutes, the limbs of all subjects were fully anaesthetic distal to a strip just below the tourniquet. Finger flexion movements were reduced in 7–20 minutes; total paralysis of all muscles in the forearm occurred in 10–25 minutes.

With ischaemia alone, dysaesthesia developed at about 5 minutes and anaesthesia in 15–30 minutes. A variable weakness developed but total paralysis occurred in only one subject.

Group II contained two subjects in whom ischaemia and lignocaine anaesthesia were followed in 10 minutes by total anaesthesia of the hand, whereas ischaemia for 30 minutes produced dysaesthesia only. In the case of the third subject, exsanguination was incomplete and analgesia occurred over the dorsum of the hand and proximal phalanges at 7 minutes. Anaesthesia spread very slowly ventrally and distally to produce total anaesthesia at 30 minutes. Ischaemia alone of the opposite hand led to dysaesthesia, but not complete anaesthesia.

**Nerve Conduction Studies.**

**Effect of ischaemia and anaesthesia on motor conduction velocity of the median nerve in the forearm.**

The changes in the motor conduction velocity in the median nerve in the forearm following ischaemia and intravenous lignocaine were compared with those following ischaemia alone of the opposite limb in the five subjects of Group I. It may be seen from figure 1 that there was a more rapid impairment of conduction when lignocaine was injected, and that in all cases total failure of motor conduction had occurred in 25 minutes. In one case (J.Th.) motor conduction had failed completely 5 minutes after injection of lignocaine, whereas with ischaemia alone conduction was still occurring at 30 minutes.

**Effect of ischaemia and anaesthesia on terminal latency of muscle action potential.**

The effect of lignocaine and ischaemia on the latency of the action potential recorded from the abductor pollicis brevis muscle following stimulation of the median nerve at the wrist, was compared with that of ischaemia alone in the five subjects of Group I. It may be seen from figure 2 that the injection of lignocaine made no significant difference to the rate of increase in latency when the whole of the forearm was exsanguinated and anaesthetized. By contrast, when only the wrist and hand were exsanguinated in the subjects included in Group II, the latency increased earlier and at a more rapid rate with injection of lignocaine (fig. 3A).

**Effect of lignocaine and anaesthesia on sensory conduction.**

When the whole forearm of the subjects of Group I was exsanguinated, the rate of increase in the latency of the sensory action potential and the rate of decrease in its amplitude was not significantly affected by the injection of lignocaine (figs. 4, 5). It was of particular interest that in the case of J.Th., in whom motor conduction in
Fig. 1
Effect of ischaemia alone (closed circles, solid lines) and 1 per cent lignocaine and ischaemia (open circles, interrupted lines) on motor conduction velocity in the median nerve in the forearm of subjects in Group I. Tourniquet applied above elbow.

Fig. 2
Effect of ischaemia alone (closed circles, solid lines) and 1 per cent lignocaine and ischaemia (open circles, interrupted lines) on distal motor latency in median nerve of subjects in Group I. Tourniquet applied above elbow.
the forearm segment of the median nerve failed within 5 minutes, conduction was not significantly affected in the distal motor and sensory branches (figs. 2, 4, 5).

When only the wrist and hand of the subjects in Group II was exsanguinated, lignocaine caused an earlier and more rapid impairment of sensory conduction (fig. 3b, c).

**DISCUSSION**

In both groups of subjects, clinical and electrophysiological impairment of motor and sensory function occurred more rapidly in the arm injected with 1 per cent lignocaine than in that affected by ischaemia alone. These observations confirm that the local anaesthetic agent plays a significant role in the production of analgesia. However, for rapid analgesia to be achieved, adequate exsanguination of the limb is required and the slow onset of analgesia in one subject in Group II resulted from incomplete exsanguination.

In all subjects, there was complete loss of painful sensation in the hand at a time when a sensory action potential could still be recorded. This finding may be explained by the fact that electrophysiological studies define the changes which occur in conduction in the motor and sensory fibres of large diameter only; local anaesthetic agents block conduction in unmyelinated and small diameter myelinated fibres before those of large diameter (Nathan and Sears, 1961; de Jong and Nace, 1968).

In all the subjects of Group I in whom the tourniquet was applied above the elbow, lignocaine resulted in a more rapid impairment of motor conduction in the forearm segment of the median nerve than occurred with ischaemia alone. By contrast, there was no significant difference between the impairment of conduction in the distal motor and sensory segments of the nerve which occurred with lignocaine, and that which occurred with ischaemia alone. Moreover, the results of repetitive stimulation of the nerve suggest that lignocaine had little effect at the level of the neuromuscular junctions. The more pronounced effect of lignocaine on the conduction in proximal segments of the nerve almost
FIG. 4
Effect of ischaemia alone (closed circle, solid lines) and 1 per cent lignocaine and ischaemia (open circles, interrupted lines) on latency of sensory action potential in median nerve of subjects of Group I. Arrows indicate time after which action potential became unrecordable.

FIG. 5
Effect of ischaemia alone (closed circles, solid lines) and 1 per cent lignocaine and ischaemia (open circles, interrupted lines) on amplitude of sensory action potential in median nerves of subjects in Group I.
certainly resulted from a greater concentration of anaesthetic occurring in the forearm than in the hand, since Fleming and associates (1966) and Sorbie and Chacha (1965) have shown radio-
logically that the local anaesthetic proceeds rapidly in a proximal direction when injected into the dorsum of the hand in an exsanguinated limb. The conclusion is supported by the experiments in the subjects of Group II, in whom the tourniquet was placed above the wrist. In these subjects, motor and sensory conduction failed rapidly in the distal segments of the nerve, indicating that the distal motor and sensory fibres were susceptible to lignocaine analgesia when the local anaesthetic was concentrated in their neigh-
bourhood.

It may be concluded from these experiments that the site of action of 1 per cent lignocaine in regional intravenous analgesia is on the nerve trunks, as well as on the peripheral nerve endings. The results of our experiments are similar to those of Sorbie and Chacha (1965) who employed 0.5 per cent lignocaine, but in marked contrast to those of Miles and associates (1964) who con-
cluded that the local anaesthetic acted on sensory nerve endings. These conflicting observations probably result from different concentrations of anaesthetic being achieved in the limbs of the experimental subjects. Atkinson (1969) states that the pharmacological action of local anaesthetic agents in the tissues depends upon the concentra-
tion and volume of the drug administered, and on the efficiency of exsanguination; with 0.5 per cent lignocaine, the major effects of the drug are peripheral, whereas with higher concentrations nerve block may be superimposed.

In none of the subjects was more than 200 mg of lignocaine injected. The plasma lignocaine con-
centrations measured in our subjects suggest that there is no additional danger from high systemic levels in clinical situations when 0.5 per cent ligno-
caine is replaced by the same dose of 1 per cent lignocaine. Since 1 per cent lignocaine appears to act at least partly on the nerve trunk, this mode of action may be preferable to more peripheral effects of 0.5 per cent of lignocaine in circum-
stances, such as those caused by tense fracture haematomata, which prevent adequate perfusion of the operative site.

ACKNOWLEDGEMENTS

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BOOK REVIEWS

Physiological and Practical Aspects of Shock. By J. Freeman. Published by International Anesthesia Clinics. Pp. 1066; indexed. 8/-. The first problem which faces anyone who sets about writing a book on shock is that of definition. In this most informative and helpful book shock is defined as "a state of circulation in which tissues in widespread areas of the body are being damaged by nutritive insufficiency resulting from inadequate cardiac output". Starting from this point various contributors to this volume discuss shock from the practical point of view, as indeed the title of the volume indicates. Thus there are sections on cardiac output, lung function, renal failure and hepatic changes associated with shock. Therapy receives appropriate attention under the headings of blood and transfusion fluids, respiratory therapy, and hyperbaric oxygen. In each instance the subject is dealt with at a relatively simple level but in a clear manner which will be of value to every practising anaesthetist. There is a section on the acidosis produced by low blood flow and one on myocardial infarction. A specially interesting discussion relates to the use of emergency extracorporeal circulation in states of cardiac failure, a procedure which may well offer the prospect of survival to many who die of myocardial infarction or similar disturbances. The authors go on to take the reader through the increasingly complex subjects of the disturbances of rate and rhythm, the deviations of the electrical axis, the diagnosis of hypertrophy and infarction, and a number of other miscellaneous effects as well. At the end of the book there is a series of little sheets for cutting out and putting into one's diary for memorizing, and for reference to, the main points in e.c.g. diagnosis. Finally, a series of e.c.g. tracings are included for practice.

This book is very very good, in that it strikes the right level for anaesthetists (and perhaps even for the cardiologists). It uses an easy illustrated learning method in which just one main fact is presented, with a diagram, on each page. The author refers to this as a "programmed course" though this is not strictly speaking so. The information is conveyed by a series of simple questions, in the answers to which the reader fills in a missing word or two. By working through this book anaesthetists cannot fail to learn, in a sound manner, the elements of electrocardiography. The probability is, that the major uncertainties and mysteries of electrocardiographic interpretation will be dispelled for the rest of his life. However, this is by no means a book that stops at an elementary level. It goes on to take the reader through the increasingly complex subjects of the disturbances of rate and rhythm, the deviations of the electrical axis, the diagnosis of hypertrophy and infarction, and a number of other miscellaneous effects as well. At the end of the book there is a series of little sheets for cutting out and putting into one's diary for memorizing, and for reference to, the main points in e.c.g. diagnosis. Finally, a series of e.c.g. tracings are included for practice.

This book is heartily recommended to all anaesthetists. They will be delighted with it. Many more along the same lines are badly needed, and prospective authors should have a glance at it before they start on their own.

A. R. Hunter


Every anaesthetist must know how to interpret an electrocardiogram sufficiently accurately for the purpose of detecting and correcting serious abnormalities during anaesthesia, and sufficiently quickly to come to such an opinion at the bedside, or even more importantly, in the operating theatre, without undue loss of time. Most books dealing with this subject are either very detailed, being intended for aspiring cardiologists, or too simple and didactic, aiming at undergraduate students. This book is very very good, in that it strikes the right level for anaesthetists (and perhaps even for the cardiologists). It uses an easy illustrated learning method in which just one main fact is presented, with a diagram, on each page. The author refers to this as a "programmed course" though this is not strictly speaking so. The information is conveyed by a series of simple questions, in the answers to which the reader fills in a missing word or two. By working through this book anaesthetists cannot fail to learn, in a sound manner, the elements of electrocardiography. The probability is, that the major uncertainties and mysteries of electrocardiographic interpretation will be dispelled for the rest of his life. However, this is by no means a book that stops at an elementary level. It goes on to take the reader through the increasingly complex subjects of the disturbances of rate and rhythm, the deviations of the electrical axis, the diagnosis of hypertrophy and infarction, and a number of other miscellaneous effects as well. At the end of the book there is a series of little sheets for cutting out and putting into one's diary for memorizing, and for reference to, the main points in e.c.g. diagnosis. Finally, a series of e.c.g. tracings are included for practice.

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