UNINTENTIONAL SPREAD OF EPIDURAL ANALGESIA

BY

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At a meeting recently the topic for discussion was: "Is epidural analgesia worth the bother?" One might as well ask if the choice of ether or cyclopropane is ever justified, because, like epidural analgesia, an irrefutable incidence of spectacular complications accompanies their employment—explosions with ether and cyclopropane and total spinal blocks with epidural analgesia. It is nevertheless a fact that epidural analgesia is altogether eschewed for that reason in many reputable centres the world over.

If epidural analgesia is ever to attain its rightful place in anaesthesia, then all the possible mechanisms for its complications must be clearly recognized.

It is today not enough merely to avoid inadvertent subarachnoid or intravenous injections, because those account only for immediate reactions, whereas the delayed complications are becoming increasingly recognized. They may occur unexpectedly some 15 to 40 minutes after the injection is completed (Morrow, 1959; Stovner, 1957; Sykes, 1958) or they may only become apparent postoperatively as persistent neurological defects (Wolfson and Ingram, 1957; Davies et al., 1958).

The mechanism of all these delayed sequelae is still shrouded in mystery.

This paper attempts to relate the thorough work of Moore et al. (1954) on the intraneural spread of fluids in monkeys to the occurrence of delayed complications of epidural analgesia.

METHODS

On many occasions a large nerve of an extremity was dissected out immediately after amputation in the operating theatre. It was gratifying to be able to demonstrate how easily intraneural spread occurred and often how little pressure was required to accomplish this. Moore et al. (1954) state that it is, on occasion, as easy as intravenous injection. Thus, cross sections of the nerve 2 to 3 inches from the site of an injection of 2 per cent lignocaine containing 1 per cent methylene blue displayed clearly the nerve fasciculi macroscopically visible in a sea of blue (methylene blue occupying the perineural spaces).

A brief apologia is required before describing another experiment. Moore et al. (1954) demonstrated in monkeys that solutions coloured with methylene blue and injected 3 to 4 cm lateral to the intervertebral foramina, under direct vision, reached the parenchyma of the spinal cord in 2 to 5 minutes. The spinal fluid did not immediately become tinged with the dye, but it required 10 to 15 minutes for the solution to pass through the pia mater and epineurium to stain the spinal fluid lightly, and it did not become heavily stained for 35 to 40 minutes. Morrow (1959) has probably reported the longest delay before the onset of total spinal block after intended epidural injection, namely 40 minutes. Unfortunately these workers used Efocaine, a known neurogenic poison (Scott, 1958) which caused many mishaps after paravertebral block during the first few years of the last decade. Transverse myelitis, Brown-Séquard syndrome, neuritis, inflammatory reactions and lasting absence of nerve function were all attributed to the action of Efocaine (Nowill et al., 1953a; Nowill et al., 1953b; Moore, 1954; Moore et al., 1954; Parsonage et al., 1955). A severe destructive effect of the major solvent vehicle, propylene glycol, was demonstrated on rabbit tissues in 0.5 ml doses, even in a dilution as great as 1:8 (Margolis et al., 1953). These experiments of Moore et al. (1954) nevertheless demonstrated that a substance can be introduced mechanically into the cerebrospinal fluid and spinal cord by direct injection into the nerves distal to the intervertebral foramina, quite apart...
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from any injection into an outward prolongation of the subarachnoid space. There is no reason or evidence to suggest that Efocaine is unique in this respect, especially in view of the relatively large size of the dorso-lumbar nerve roots in the human being, and their transfixion in the intervertebral foramina—as opposed to the cervical roots of the brachial plexus.

In order to clarify this point and to exclude the confusing neurotoxic properties of Efocaine, a large dog was operated on under halothane anaesthesia. A lumbar nerve root was mobilized and using a No. 18 hypodermic needle, carefully injected directly at a site 1 inch (2.5 cm) from the spinal cord. About 5 ml of 2 per cent lignocaine, containing 1 in 200,000 adrenaline and 1 per cent methylene blue were injected at a rate of 1 ml every 30 seconds.

Within 15 seconds blue streaks appeared on the exposed part of the spinal cord, and they gradually became thicker and more numerous until, after 60 to 75 seconds, the whole of the exposed portion of the dura mater suddenly turned blue (figs. 1 and 2).

Up to this time the depth of anaesthesia was maintained at a plane which allowed movements of the limbs to occur almost without interruption. Although the general anaesthetic was now completely withdrawn, the dog only managed to move his head. The paraplegic state was allowed to persist for half-an-hour, when the dog was sacrificed with an overdose of halothane and the whole spinal cord dissected out; it was found to be stained blue along its whole length, more intensely in the lower dorso-lumbar area, as was to be expected.

**Fig. 1**
A lumbar nerve in a dog being directly injected with a lignocaine-methylene blue solution; showing the cephalad spread into the spinal cord. See also figure 2.

**Fig. 2**
Same dissection as in figure 1, but a minute later at the exact moment of rupture of epineurium and pia with instantaneous discoloration of the whole spinal cord.
DISCUSSION

The proof of a spread centrally via the perineural spaces of lignocaine in figures 1 and 2, is perhaps clearer, more striking and convincing than the beautiful illustration of the same avenue of spread of Efocaine on page 52 of Moore's (1955) book on the complications of regional blocks. This is exactly what a consideration of the anatomy will demand, because a nerve root consists of fasciculi, each surrounded by perineurium and perineural spaces, the whole root being surrounded by the epineurium. After intraneural injection, spread therefore occurs initially along the perineural spaces only and may continue thus directly into the spinal cord (fig. 1). With mounting pressure the relatively fragile epineurium, and especially its continuation the pia mater, are stretched, the latter thus rendered more permeable to solutions (Moore et al., 1954) until the pia mater may suddenly rupture with wide subarachnoid dissemination of the injected solutions as a result (fig. 2).

Clinically a patient has been noted in whom a neurological lesion, consisting of lower limb motor defects coupled with preservation of more superficially situated sensory tract function, persisted after spinal analgesia was induced during thiopentone anaesthesia (Mostert, 1958). Surely intraneural or even direct injection into the spinal cord must be invoked as a possible, or even probable cause. Although in 94 per cent of humans the spinal cord terminates in the region of the first lumbar vertebra, in some it may end as low as the third. Dripps and Vandam (1951) encountered paraesthesia in 13 per cent of a series of lumbar punctures.

While intraneural injection must be very rare during the induction of epidural analgesia, there has been a failure to recognize it as a prelude to disaster by all the British authors referred to in this article and this has led to a lack of faith in the utility of the test dose and a lack of appreciation of the need for good rapport with the patient during the epidural injection in order to avoid pain and paraesthesia, and so intraneural injection. This is particularly unfortunate, because the fact that no spinal fluid is obtained, even on aspiration, is no assurance that the needle is not in the intrathecal space (Adrian et al., 1952). Two cases of total spinal block were reported by Bonica et al. (1957) despite the presence of this sign, but a further 17 total blocks were inevitable in their series of 3,637 cases had it not been for their routine use of the test dose.* In any case, it is faulty reasoning (similar to stating the conclusions of an experiment before it has taken place) to say that a test dose followed by a 5 minutes waiting period is without value because widespread subarachnoid extension may be delayed up to 40 minutes. Finally it is essential, when investigating these complications, to know whether pain, paraesthesia, and weakness were present after the test dose, as well as knowing whether spinal fluid or blood was encountered prior to the block.

SUMMARY AND CONCLUSION

Previous work on the intraneural spread of Efocaine in monkeys is reconsidered in the light of a similar sequence demonstrated in a dog injected with lignocaine, and in human nerves obtained from amputated limbs. The conclusion, shown to be entirely feasible, is that a spread via the perineural spaces must be considered in any attempt to account for the occurrence of unexpectedly delayed extension of epidural analgesia. To avoid these complications good rapport with the patient during epidural injection in order to avoid all pain and paraesthesia and the routine use of a test dose are absolute requisites.

* During the past year the author personally administered or supervised 300 administrations of epidural analgesia between thoracic 8 and lumbar 5 vertebrae, at the King Edward VIII Hospital in Durban. The conscious patients were invariably sitting up; their ages varied between 7 and 90. An ordinary 10 cm 18 S.W.G. autoclaved needle provided as a routine for drawing up of drugs was used, and the analgesic solution consisted of 8 to 22 ml 2 per cent lignocaine with adrenaline. A test dose was not used, but all other precautions were observed. Three total spinal blocks, complete with respiratory arrest during the hypotensive period, irregular pupils for the duration of unconsciousness, and tolerance of an endotracheal airway in the presence of good pharyngeal tone unexpectedly ensued after two to twenty-two minutes the earliest total block progressing to cardiac arrest. A fourth patient had convulsions remedied with suxamethonium, and a fifth brief but profound hypotension after her legs were lowered from the lithotomy position, both prior to Caesarean section. There were nine failures, no neurological sequelae, and all recovered completely (100 Caesarean sections were in the series), but today all the participating colleagues agree on the grave necessity of the test dose.
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REFERENCES

BOOK REVIEW


This is likely to be a "Bible" for research workers in this field for some time to come. A great deal of experimental work in anaesthesia has been based upon pH estimations which by modern standards would be quite unacceptable. Professor Woolmer did a great service by arranging the Symposium, the proceedings of which are here reported, and by persuading undoubtedly international authorities to contribute. We can only endorse the remarks of Professor Woolmer in his Foreword: "that this book brings together a wealth of information about a subject so diffuse that no textbook deals with it as an entity; and though it does not set out to cover every aspect fully, there are not many which escape mention in the discussion or in the references, even if they are not dealt with in one of the ten papers". The Symposium was held in three sessions. Each paper was followed by a discussion and there was a general discussion on the whole subject of each session. All of this is here reported and as is usual in this type of work the discussions are most instructive material.

Among the most valuable subjects covered are the interpolation methods of estimating blood Pco₂; the accuracy of which is fully evaluated; the problem of whether "true" and "separated" plasma are identical for the purpose of equilibration techniques; the value of the end-tidal sample as an estimation of arterial Pco₂; and particularly useful are the sections during the first and third sessions devoted to a consideration of the various electrodes for pH measurement, the direct estimation of blood O₂ and CO₂ and the electrochemical aspects of blood pH measurement. This book has already become a standard reference. Cecil Gray