UNEXPLAINED SPREAD OF EPIDURAL ANAESTHESIA

BY

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The case to be described is unique, in that the high spread of local analgesia became obvious some 40 minutes after an apparently perfectly administered epidural block.

CASE REPORT

The patient was a male of average build, aged 60, a mild diabetic with resting blood pressure 180/80 mm Hg, pulse 76, and requiring treatment for occlusive vascular disease affecting the lower limbs. Ten weeks previously epidural block had been performed: (1) to test sympathetic release and (2) for the purpose of providing analgesia and sympathetic blockade during and immediately after a femoral arteriogram. It was decided to repeat the arteriogram.

11.15 a.m. Premedication of papaveretum, 20 mg, scopolamine 0.4 mg was given by subcutaneous injection.

12.15 p.m. On arrival in the theatre the patient was mildly sedated and co-operated well.

12.20 p.m. Epidural puncture was performed using a Macintosh needle, the patient being in the lateral position and the table horizontal. The epidural space at L3, 4 was located using the "Dogliotti" loss of resistance technique. Aspiration for cerebrospinal fluid (c.s.f.) was negative and no fluid efflux occurred throughout the procedure. 20 ml 1.5 per cent lignocaine without adrenaline was injected. Voluntary movement of legs was present and analgesia reached approximately T9. Some fall of blood pressure occurred. This was maintained with fractional intravenous doses of methoxamine (10 mg X 2) at a level of 130 mm Hg systolic. Otherwise the procedure was uneventful. There was no change of posture during the arteriogram, which was carried out in the supine position.

1.00 p.m. The arteriogram was now completed. The patient was very drowsy now but sensible and co-operative. Sensory block was found to extend to the level of the clavicles. Sensation in 5th cranial nerve involvement. Breathing was quite free and co-ordinate with a good respiratory excursion. Power gradually returned in the limbs, most rapidly in the first 15 minutes.

3.30 p.m. Complete recovery of sensory and motor function had now occurred and sensation in 5th cranial was now normal.

4.15 p.m. The patient was returned to ward. The blood pressure was now stabilized and the noradrenaline drip discontinued. Postoperatively there were no residual sequelae and the patient had no unpleasant memories. The total duration of amnesia was indefinite.

It was thought that total spinal block had occurred with limited cranial nerve involvement, loss of consciousness, and a most remarkable delay in onset.

COMMENTARY

Intrathecal spread of local anaesthetic solution is a well-recognized complication of the extradural technique, but usually the spread of the solution becomes obvious within a few minutes. Sykes (1958), however, described a case where the dura had been punctured, with a delayed onset of 15
Bonica (1957) reported two cases of total spinal in 3,637 cases: 17 others could have occurred had it not been for the use of the test dose. Puncture of the dura was not detected before injection. The majority of these occurred with the catheter technique. There were several cases in which an apparently normal epidural block developed within 15 minutes of injection and subsequently a much more extensive block followed. In none of these instances was dural puncture detected (Bonica, 1959).

From the University College Hospital figures quoted by Sykes (1958) the occurrence of total spinal would appear to be about eight times more likely when an epidural injection is given at the same vertebral level as a previous dural puncture, than when given at a different level when the incidence is just over 2 per cent. In the majority of cases where the dura has been definitely punctured high spinal block does not result.

In some, at least, of the above cases the dura had been punctured, thereby facilitating the spread of local anaesthetic. The delayed onset of the block in the case described suggests a slow transfer of analgesia into the cerebrospinal fluid and subsequent diffusion to the upper cord and brain stem, although one would normally expect the greater part of the epidural injection to have been absorbed by this time. One must consider now whether the diffusion occurred through the intact or damaged dura. It seems improbable that the dural permeability might have been altered as a result of the satisfactory epidural block carried out 10 weeks previously. It has been shown by Franksson and Gordh (1946) that following dural puncture in humans the wound may remain patent for as long as 14 days. Dural puncture had not been carried out at any time on this particular patient, nor had the patient received any corticosteroid therapy. As mentioned previously by Sykes (1958), following puncture of the dura, the pressure difference between the two compartments tends to prevent massive spread from the epidural to the subarachnoid space in a high percentage of cases. In such a case as this, the dura may or may not have been minimally damaged. The fact that this subject was a mild diabetic, with possibly an increased c.s.f. specific gravity which might favour diffusion, would seem of small importance.

Recently it has been pointed out by Saker and Shroder (1954) that in the presence of low c.s.f. pressures, a high concentration of extradurally deposited local analgesic could be obtained from the c.s.f. in the lumbar region, whereas none was obtained in the presence of a high c.s.f. pressure. If these results are substantiated and shown to be applicable to conditions occurring in clinical practice, this then raises the possibility that a lowered c.s.f. pressure of sufficient magnitude and duration might be the trigger mechanism on occasions when massive spread occurs following an epidural block. There is reason to believe that the normal healthy intact dura is permeable to local anaesthetics and some, if not all, the effects of epidural anaesthesia are attributed to the presence of an effective concentration in the c.s.f. (Frumin et al., 1953a, b). However, opinion is not unanimous on this subject, and Foldes (1954), while admitting that such penetration does occur, considers that the main site of action is outside the dura. The pros and cons of this argument have been admirably discussed by Geddes (1958).

**CONSIDERATION OF SPINAL BLOCK**

The high spread of lignocaine produced a progressive ascending paralysis, resulting in what appeared to be a total spinal block. Before consciousness was lost the motor innervation of the larynx was depressed as evidenced by the husky whispering speech. It will be recalled that the motor nerve fibres to the larynx, which travel in the recurrent laryngeal nerve, are derived from the medullary part of the accessory nerve and extend along the surface of the medulla to join the vagus.

The absence of sympathetic activity can result in an extreme degree of miosis, clinically occurring as a result of block at any point of the sympathetic pathway—in the brain stem, cervical or upper thoracic cord, thoracic inlet, or along the carotid sympathetic plexus. High spinal blockade results in a complete cessation of sympathetic outflow activity. However, miotic pupils may be seen from time to time during the conduct of standard anaesthetic procedures. The fact that the pupils dilated somewhat on arousal, when sympathetic activity was still in abeyance, would suggest an alteration in oculomotor activity...
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occurring in conjunction with the change in the level of consciousness.

The isolated involvement of the trigeminal nerve, the sensory nucleus of which is in the pons, would suggest that the long sensory root which descends into the upper cervical region of the cord, had been blocked at this level in its relatively superficial course.

As resting vagal tone is wholly reflex in origin and depends on afferent impulses along the sinus and aortic nerves, the occurrence of extreme bradycardia relieved by atropine suggests the persistence of some degree of autonomic vagal activity in association with ablated sympathetic and adrenal medullary function.

Von Euler (1955) has pointed out that the positive chronotrophic action of noradrenaline in the living organism is often masked by a reflex bradycardia. The above observations are consistent with this view.

The loss of consciousness is of particular interest here, occurring as it did with restricted cranial nerve involvement in the presence of an apparently adequate cerebral circulation.

Disturbance of consciousness as a result of the passage of local analgesics into the cranial c.s.f. has been commented on from time to time and more recently in this country (Woolmer, 1948; Jones, 1953; Stovner, 1957; Sykes, 1958).

Sykes (1958) quotes several authorities supporting the view that low concentrations of local analgesics will affect the cranial nerves or their nuclei and greater concentrations will also have an effect on the vasomotor and respiratory centres.

SOME ASPECTS OF MECHANISM OF UNCONSCIOUSNESS

In recent years anatomical and physiological studies have added much to the better understanding of brain mechanism and consciousness and have demonstrated the mutual interdependence of the cortical neuronal systems and the multisynaptic systems of the reticular formation of the brain stem.

Direct electrical excitation of the reticular formation of the brain stem induced changes in the electro-encephalogram (e.g.) seemingly identical with those observed in waking from sleep. The reticular formation is well orientated up the brain stem to thalamic level, not as a straightforward tract system but as a diffuse neuronal network, linked equally to the cortices of both hemispheres (Delafresnaye, 1954).

It is known that isolated cortical lesions do not disturb consciousness, whereas upper brain stem lesions do. Bremar (1935) showed that transection of the midbrain in animals produced sleep, whereas after transection at C1 (Bremar's Encephalé isolé) the head end remained awake. E.g. studies exhibited the characteristic activities. Bremar thus demonstrated that an ascending influence from the brain stem was responsible for wakefulness, which would appear not, as was thought, to be due to the blocking of the sensory paths in this instance but to injury to the upper end of the reticular activating system, where these influences are elaborated for cortical propagation (Magoun, 1950).

It has been shown by French et al. (1953) that impulses from peripheral sensory receptors ascend to the cerebral cortex not only by the classical long tract systems but relay in this multisynaptic reticular formation, which then projects to all parts of the cerebral cortex by various paths. It appears that barbiturates alter the threshold of arousal by preventing the stimulation of the reticular formation which arrives via the collaterals from the classical somatic sensory pathway. King (1956) supports this view as a result of intensive studies.

Discussion on the specific cause of loss of consciousness in this case tends to be rather speculative. Circulatory and respiratory insufficiency of a degree sufficient to cause loss of consciousness did not occur, and one must consider the possibility of systemic absorption possibly occasioned by the injection of the radio opaque media. There was, however, nothing to suggest any type of generalized reaction to iodide. The relatively long period of unconsciousness favours a subarachnoid effect.

If the mechanism was due to a direct action on the reticular formation, then involvement of the upper brain stem region would be required if one accepts a direct analogy with the experimental transection studies (Bremar, 1935). The spread of local analgesics in the subarachnoid space demonstrates that when high levels are reached diffusion takes place mainly on the ventral aspect of the brain. Very large volumes must be
given before the ventricular fluid will be reached (Grodinsky and Baker, 1933).

Direct action at upper brainstem level would seem unlikely in the absence of extensive cranial nerve involvement, unless some low critical concentration of lignocaine can be effective in causing loss of consciousness, acting very selectively on the reticular system.

Stovner (1957) has described a case of "subtotal spinal analgesia" which occurred when performing paravertebral block, using lignocaine. In this case the upper four cranial nerves as well as part of the fifth cranial nerve were spared and consciousness retained.

Sensory impulses from the trigeminal area have been shown to be especially effective in activating the brain stem reticular system (Rossi and Zirondoli, 1955). Deafferentation would seem to be of special significance in this case. An interesting comparison can be made with the parallel effect on the phrenic reflex as suggested by Bromage (1958, 1959).

**SUMMARY**

A case of delayed spread of extradural anaesthesia with loss of consciousness is described. The possible mechanisms of this phenomenon are discussed.

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