Sub-total spinal analgesia

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Total spinal analgesia might be produced inadvertently whilst performing epidural, paravertebral or sacral blocks for diagnostic or operative procedures. Several such cases have been reported in the literature [2 14 12].

In practice these cases are of interest because immediate resuscitative measures will save life, while delayed or inneffective measures often give a fatal result.

Theoretically the effect on the cranial nerves and on consciousness of a local analgesic drug diffusing into the cranial subarachnoid space raises some points of neurophysiological interest.

In the cases previously described in the literature, there was a complete block of all the cranial and spinal nerves with loss of consciousness. This is an account of a case where the most anterior of the cranial nerves were spared and consciousness retained. Some practical and theoretical points are discussed.

DESCRIPTION OF CASE

The patient was a 40-year-old man in good general condition. Because of a vasospastic condition in the upper extremities the surgeon wanted to perform a paravertebral block of the sympathetic trunk to evaluate the relief the patient might have from a sympathectomy later on. The patient was in the lateral position on the operating table and a posterior approach to the thoracic sympathetic trunk was planned. The needle was inserted just above the second rib two inches from the midline in a medial direction towards the anterior aspect of the body of the vertebra. Without attempting to aspirate, 10ml of 10 per cent lignocaine with exadrin was injected. Shortly after the injection the patient experienced a general feeling of warmth and numbness and paralysis from below upwards. In a few moments he was unable to speak and respiration stopped. The surgeon grasped the situation immediately and started to perform manual artificial respiration. Two anæsthetists arrived after a few minutes and started positive pressure respiration with bag, mask and oxygen. In spite of a completely relaxed jaw it was easy to keep a free

airway and tracheal intubation was not performed. Systolic BP was found to be 80mm Hg. After 100mg ephedrine intramuscular the BP rose to 120mm Hg and did not drop later.

Shortly after the injection of the lignocaine the face of the patient became expressionless, and the lower jaw completely relaxed. He was, however, able to open his eyes and co-ordinate his eyeballs. Stimulating the skin on his forehead produced this reaction of opening the eyes and looking at the examiner. Shouting the patient's name produced no response, neither did stimulating the rest of his body.

After fifteen to twenty minutes swallowing movements were observed with some increase in the tone of the lower jaw. In about an hour attempts at spontaneous respiration were observed first with the accessory respiratory muscles in the neck, later with the diaphragm and, finally, full intercostal respiration was established. Later on the ability to speak was regained. He made a complete recovery, but complained of ringing in the ears for a day or two afterwards.

The patient said afterwards that he had been fully awake all the time, but had for a period completely lost all his sense of hearing. His vision, however, had been unaffected and he had seen all that had gone on. He had been without skin sensation up to the cheeks and ears, but had felt our pin-pricks on the forehead. On account of the respiratory paralysis and the loss of ability to speak he had been panic stricken for a few moments. For some weeks afterwards he was haunted by night-mares as he went through the episode in his dreams.

DISCUSSION OF TREATMENT

Block of cranial and spinal motor nerves as well as the sympathetic outflow results in immediate cessation of respiration and loss of vasomotor tone with fall of blood pressure. The first principle in the treatment of these patients is therefore as soon as possible to establish effective artificial respiration, paying special attention to a free airway, as the relaxed lower jaw will partly obstruct this. This is best done with intermittent positive pressure respiration with oxygen, bag and mask. If it is difficult to maintain a free airway an endotracheal tube should be inserted.

Secondly the loss of vasomotor tone with hypotension should be corrected as soon as possible by the use of a vasoconstrictor like ephedrine subcutaneously or intramuscularly or nor-adrenalin by slow intravenous drip. A slight head-down tilt will prevent brain anoxæmia and make it easier to elevate the blood-pressure. In the case described above these measures were taken at once, and at no time was the patient cyanosed or collapsed.

DISCUSSION OF EFFECT ON CRANIAL NERVES AND CONSCIOUSNESS

In the cases previously described in the literature of total spinal analgesia, a complete block of all cranial nerves as well as a period of

unconsciousness were found. In the case described above the four first cranial nerves as well as the anterior division of the fifth nerve was spared and consciousness retained. Such a partial subarachnoid block of the cranial nerves has attracted little attention in the literature. The complete loss of hearing with retained vision was a very striking experience of our patient, and should have prompted us to give him instructions in writing, if we had been aware of it. The tinnitus which persisted for a day afterwards also shows that the eighth nerve had been severely affected.

The loss of consciousness described by other authors is interesting and several explanations have been offered. If causes such as anoxia of the brain, prolonged hypotension or excess of analgesic reaching the brain via the blood stream can be excluded, only two explanations need be considered.

The first explanation is the possibility that the total sensory block has rendered the patients unaware of themselves and their surroundings so no memory is retained for this period. This explanation is given by several authors[8 7 2]. The experiments with the *encephalé isolé* contradict this explanation[6, 11]. After transection of the brain stem up to the pons but caudal to the chief trigeminal nucleus and interruption of all the afferent cranial nerve paths, the electro-encephalogram from this completely isolated brain showed the normal low voltage fast activity. Administration of nembutal to this isolated brain at once precipitated typical 'barbiturate spindles'.

The second explanation is the possibility of a direct action of the analgesic on certain parts of the brain via the cerebrospinal fluid[5 15]. The search for anatomical structures in the brain to which consciousness is linked is old and is still far from completed. Recent clinical and neurophysiological research has made it likely that the brain stem reticular system is the seat of consciousness[9]. These are the most superficial structures in the wall of the third ventricle. Penfield found by exposing the third ventricle in man under local analgesia and pressing on certain parts of the wall of this ventricle, that stupor and coma resulted[10]. This was relieved when the pressure ceased. Hypersomnolence may be produced by tumors involving the floor of the third ventricle[13]. Experimentally produced lesions in the posterior parts of hypothalamus in animals result in a condition of somnolence with an electro-encephalogram pattern of sleep type[4]. To reach these structures the local analgesic would have to diffuse into the ventricular system. The work on this problem of whether a local analgesic agent introduced into the spinal subarachnoid space can reach the ventricular spaces of the brain, is inconclusive. For instance Cotui and Standard showed in dogs that both procaine and methylene blue introduced into the cisterna magna diffused into the fourth ventricle[1].

Grodinsky and Baker, however, injected procaine into the subarachnoid space in dogs, cats and rabbits, and recognised the procaine post-mortem by a diazzo-colour reaction. It was found that the procaine mainly diffused to the ventral aspects of the brain. With increasing volume of injected drug some diffusion to the dorsal surface of the brain also occurred, but very large volumes of drug were needed for the ventricular fluid to be reached[3].

The fact that the cranial nerves were blocked indicates that the analgesic drug introduced into the spinal subarachnoid space has in fact diffused up into the cranial subarachnoid space. In the case described above this diffusion had only reached the posterior part of this cranial subarachnoid space, so only the posterior cranial nerves were affected. This did not affect consciousness. The fact that the fifth cranial nerve was only partly affected was perhaps a contributory reason for our patient remaining fully conscious all the time. It has been shown recently that sensory impulses from the trigeminal area are specially effective in activating the brain stem reticular system[11]. In any case it seems as if a diffusion of the analgesic to the anterior part of the cranial subarachnoid space as well is necessary to produce unconsciousness. Whether the drug then produces unconsciousness by a complete block of all sensory nerves or by a direct action on some parts of the brain stem, is still an open question.

Acknowledgement

My thanks are due to B. R. Kaada, Anatomical Institute, University of Oslo.

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