CASE REPORTS

Epidural Misplacement of Catheters and Mechanisms of Unilateral Blockade

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One of the most intriguing as well as disappointing mechanisms of failure of epidural anesthesia is the development of unilateral blockade. Since unexpected hemianalgesia is uncommon,[1,2] we wish to report several recent cases and to present a new explanation for its occurrence.

REPORT OF CASES AND STUDIES IN DOGS

Case 1. A 45-year-old man was admitted to the hospital with a diagnosis of gastric carcinoma. He had fever and anemia, and appeared to be in poor general condition. The removal of bilateral supraclavicular masses, believed to be metastatic ganglia, under cervical epidural anesthesia was scheduled. On the day of the surgical operation the patient was brought to the operating room without premedication. After aseptic preparation of the skin, the epidural space was entered at C7–8 using the hanging-drop technique, with the patient sitting up. A plastic catheter was introduced cephalad for about 5 cm, and a total of 10 ml of 2 per cent lidocaine with epinephrine, 1:160,000, was injected through the catheter. In approximately ten minutes the right arm became considerably heavier than the left and loss of pinprick sensation developed down to T2, on the right. No analgesia was found on the left. There was no change in arterial blood pressure. Pupils remained moderately dilated and equal throughout, and no signs of sympathetic blockade appeared on the upper extremities. Respiration, as judged by excursion of the thorax and abdomen, did not change. The operation on the right went on uneventfully. For the left side, a field block was performed by the surgeon. At the end of surgery, Hypaque, 10 ml, was injected through the epidural catheter and x-rays of the cervicothoracic spine were made. The anteroposterior film showed a unilateral distribution of the dye, which was confined to the right side of the epidural space. The patient was placed on his left side for about five minutes and a second x-ray taken (fig. 1). This film showed very little spread of Hypaque towards the left side. The patient died of pulmonary infection two weeks after the operation. Postmortem examination showed a normal epidural space, free of adhesions.

Case 2. A 25-year-old healthy primigravida with no history of previous spinal or epidural anesthesia was admitted to the Obstetric suite in active labor. An epidural puncture at L3–4 was performed with the patient in the sitting position, a vinyl catheter was introduced 6 cm cephalad, and a total of 11 ml of 1 per cent plain lidocaine (including 3 ml of test dose) was injected. The patient was turned supine and five minutes later she noticed tingling on the right side of the body, fingers included. Horner's syndrome on the right side was noted. Analgesia developed up to the right T10 dermatome. No anesthesia developed on the left side of the body. After 45 minutes Horner's eye signs and analgesia on the right began to disappear. Subsequent doses up to a total of 22 ml of 1 per cent lidocaine were injected with the patient in the left lateral decubitus position. This produced solid anesthesia on the right and spotty anesthesia on the left. Blood pressure remained stable at 125/80 mm Hg throughout the procedure. A healthy newborn baby with Apgar score of 9 at 1 minute and 10 at 5 minutes was delivered. The postpartum period was uneventful.

A year later, the same patient had an epidural block for delivery of a second baby. This time the initial 10-ml dose of 1 per cent plain lidocaine was injected through the epidural needle at L3–4, and subsequent doses of 15 ml were injected through an epidural catheter. This produced satisfactory bilateral anesthesia up to T10. Delivery was uneventful.

Animal Studies. Two instances of unilateral epidural block were observed in a series of 30 epidural anesthetics performed in loosely-restrained dogs. In both animals a catheter was placed epidurally by percutaneous puncture with a Touhy #17 needle at L6–7. Five ml of 1 per cent lidocaine produced anesthesia confined to the left front limb and left side of the trunk. Unilateral Horner's syndrome developed on the left. The animals were sacrificed with intravenous pentobarbital and necropsies performed. No adhesions or gross pathology were found in the entire epidural spaces. Both catheters, which remained *in situ*, were found to enter in the midline. Each deviated laterally, and after passing between two contiguous nerve roots, made a 130-degree-angle turn and went cephalad in front of the dural

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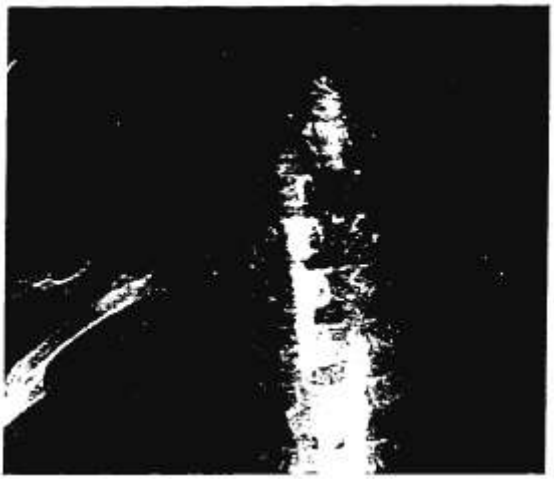

FIG. 1. Case 1. The catheter has entered the epidural space between the sixth and seventh cervical vertebrae and then passed down to the right side of the epidural space. Contrast medium has spread preferentially to the right along the cervical and thoracic epidural space.

sheaths. In each dog, the tip of the catheter was in the angle formed by the ventral aspect of the dura and the anterior wall of the spinal canal.

COMMENTS

Four causes for unilateral epidural analgesia have been suggested previously: 1) With the patient lying on his side, the slow injection of small volumes of local anesthetic solutions permits gravity to deposit all the solution along the lowermost part of the canal [3] and favors escape towards the dependent paravertebral area. This mechanism is mainly operative in young individuals [4] and allows planned or unexpected unilateral analgesia to develop. 2) Congenital [5] or 3) acquired [1] midline adhesions between the dura mater and the lamina may act as diffusion barriers, regardless of position. 4) Passage of the catheter tip into the paravertebral area and subsequent unilateral anesthesia may occur when excessive length of the catheter is introduced epidurally.[6] Although these mechanisms may explain many instances of unilateral spread (perhaps case 2) they are not responsible for all. As shown by case 1 and the two dogs studied, unilateral anesthesia may occur even when *the catheter is in the epidural space* and *there are no epidural adhesions*. Therefore, there must be some other explanation. Because of our findings in the animal experiments, we hypothesize that the ventrolateral position of the catheters, in front of the dura, may also result in unilateral anesthesia.

The epidural space, although a continuum on the longitudinal axis, is fragmented in cross section. The dural sac and its root prolongations divide the epidural space into a *large posterior* and a *small anterior* area, both of which communicate between each adjacent pair of nerve roots. In the normal individual there are no adhesions between the dura and the lamina, but there are trabeculations between the ventral aspect of the dura and the posterior longitudinal ligament which covers the body of the vertebra. Thus, while the posterior epidural space is single and unobstructed, there are two anterior epidural areas which do not communicate directly with each other.

Anesthetic solutions are usually injected into the posterior epidural area. From there, they follow three routes of dispersion [7] along the pathways offering minimal tissue resistance (fig. 2): 1) longitudinally, along the craniocaudal axis; 2) laterally, escaping through each pair of intervertebral foramina; and 3) circumferentially around the dura. Pathways 1 and 2 are important for determining the extension and quality of epidural blockade;

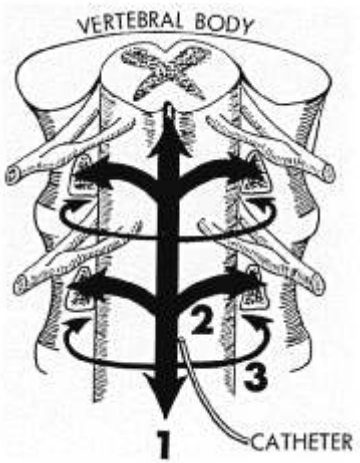

FIG. 2. When the catheter (or needle) tip is in the posterior epidural area, the anesthetic solution spreads along the longitudinal (craniocaudal) axis (*1*). Transverse (paravertebral) spread (*2*) is approximately bilateral and reinforces the block. Anterior (circumferential) spread (*3*) is negligible.

circumferential spread is irrelevant. If, however, the tip of the catheter is in the anterior epidural area (fig. 3), circumferential spread may become important. Longitudinal spread will still determine the extent of the blockade, but because of anterior midline trabeculations lateral escape is mostly confined to the side of the injection, with possible unilateral blockade. In this situation contralateral anesthesia will depend entirely on backflow of the solution around the circumference of the dura. If this does not occur at each segmental level, incomplete anesthesia or even lack of anesthesia may result on the contralateral side. Indeed, the x-ray film of patient 1 reported by Shanks [2] suggests this mechanism. Contralateral anesthesia can be favored by increasing the anesthetic volume (more overflow), slowing down the speed of injection (less longitudinal spread), positioning the patient on the nonanesthetized side (gravity) or withdrawing the catheter a few centimeters until its tip is into the posterior epidural area. Since circumferential spread is inversely proportional to transforaminal escape, and this is a function of age, it could be predicted that unilateral blockade due to the ventral position of the epidural catheter will be less likely to occur in elderly people. Conversely, unilateral blockade due to epidural adhesions could be favored by advancing age.

It would have been desirable to make x-ray contrast studies before necropsies were performed in our dogs, but this was not done. Such a procedure might have provided a different type of evidence. But whatever the x-rays might have shown, it is quite unlikely that this would in any way have affected the major finding of the study, that unilateral anesthesia may develop in the absence of epidural adhesions and without the catheter leaving the epidural space.

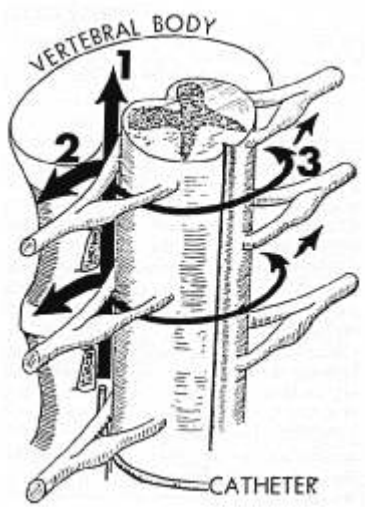

FIG. 3. When the tip of the catheter is into the anterior epidural area, longitudinal (*1*) and ipsilateral transverse (*2*) spread predominate. The magnitude of circumferential spread around the dura (*3*) determines the quality of contralateral blockade.

This presentation, incidentally, illustrates a possible disadvantage of catheter epidural anesthesia, in that failures are more likely to occur with it than when single-injection epidural anesthesia is administered via a needle. When an injection is made through a needle, patient position and anatomic abnormalities may produce unilateral blockade, whereas when the injection is made through a catheter there are *at least five* possibilities for failure. This is not intended to discredit continuous techniques. The benefits of injecting through a catheter are so important that they far outweigh the possibility of an occasional failure.

Finally, it is apparent from these and other [1, 2, 4] observations that bilateral blockade may not develop when the anesthetic solution spreads unilaterally into the epidural space. Since considerable concentrations of the local anesthetic drugs appear in the cerebrospinal fluid in this situation,[8] it would appear that cerebrospinal fluid levels of the local anesthetics are unrelated to the extension and quality of the blockade. Furthermore, this would tend to support the contention [4] that transdural passage of the local anesthetic drugs is not the only blocking mechanism in epidural anesthesia.

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Massive Hyperkalemia after Administration of Succinylcholine

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Hyperkalemia of sufficient magnitude to cause serious arrythmias, even to the point of ventricular fibrillation, is an often-recorded effect of administration of succinylcholine to the burned or injured patient.[1, 2] Recently we have encountered three neurosurgical patients, all with upper-motor neuron lesions and hemiplegia or paraplegia, who had electrocardiographic evidence of massive hyperkalemia after receiving succinylcholine. This is a report of the third patient.

Case Report

A 54-year-old man was admitted for evaluation of increasing disorientation and memory lapses. During his hospitalization a pneumoencephalogram was made with the patient under nitrous oxide and halothane anesthesia. Succinylcholine, 100 mg, had been injected intravenously for tracheal intubation. No electrocardiographic abnormalities were seen during anesthesia. Ventricular dilatation was noted on the pneumoencephalogram and the patient was scheduled for a right ventriculo-jugular shunt at a later date. Anesthesia again was nitrous oxide–halothane with succinylcholine, 100 mg intravenously, for intubation. Again, no electrocardiographic abnormalities occurred. Difficulty was encountered in placing the ventricular catheter and the procedure was abandoned. The next day the patient developed left hemiparesis.

Three weeks later the patient was scheduled for a left ventriculo-jugular shunt. He was premedicated with atropine, 0.5 mg intramuscularly, and anesthesia was induced with thiopental, 200 mg intravenously, followed by methoxyflurane and oxygen. An arterial catheter was placed and baseline samples for electrolytes drawn. The patient was then given succinylcholine, 60 mg intra-

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