Isolated Neurologic Deficit Following Spinal Anesthesia

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Neurologic sequelae following spinal anesthesia have been the subject of considerable interest and controversy for many years. The classic studies of Dripps et al. have shown that major neurologic complications do not follow spinal anesthesia. There are instances where peculiar neurologic changes do, however, follow spinal anesthesia; and the case presented here may represent such a complication. It also presents a distinct diagnostic problem.

CASE REPORT

A 61 year old white man was admitted to the Urology Service with a four-year history of transitional cell carcinoma of the bladder. In the preceding three years he had undergone five transurethral resections of this lesion. For each of these procedures spinal anesthesia was administered using tetracaine 8-10 mg., and phenylephrine, 4 mg., in a hyperbaric dextrose solution. No sequelae were reported following any of these procedures.

Because of recurrence of this lesion, a sixth transurethral resection was performed during spinal anesthesia using tetracaine, 10 mg., and phenylephrine, 4 mg., in a hyperbaric dextrose solution. All ampules were heat sterilized. The drugs were administered in the left lateral decubitus position through a 22-gauge needle; the lumbar puncture performed through the midline of the L4-5 interspace. No mechanical difficulties were noted with the administration of anesthetic and a 2-hour operative procedure was completed without difficulty.

On the first postoperative day the patient's right knee “buckled” when he attempted to walk. Neurologic examination immediately after this incident demonstrated weakness involving only the right quadriceps femoris muscle group. There was no sensory deficit. Baseline electromyography was performed in order to document any future femoral nerve degeneration. Denervation activity was not detected at that examination. Muscle strength gradually improved with the aid of physical therapy; and at the time of discharge 10 days postoperatively, the patient was walking with the aid of only one crutch. Followup examination after two months revealed complete return of function.

COMMENT

Neurologic complications following spinal anesthesia may be divided into two categories: those which are coincident and not related to the anesthetic and those which causally related. Examples of the latter are: (1) exacerbation of pre-existing neurologic disease, (2) errors in the nature of/or concentration of the material injected, (3) infection resulting from faulty sterilization or improper technique, and (4) direct trauma to nervous tissue at the time of lumbar puncture.

The patient presented demonstrated loss of function of one muscle group following spinal anesthesia. Of the possible causes of this lesion, intrathecal trauma seems unlikely. Trauma to the cord itself or to the lumbar nerve roots involved would also be very unlikely because of the termination of the cord at the second lumbar interspace. Trauma to one of the three motor roots involved in the cauda equina would be conceivable, but the chance of damaging all three with a single puncture would be extremely remote. Chemosensory injury would also seem very unlikely because the mechanism responsible for involvement of only motor roots supplying one muscle unilaterally following the injection of a chemical into the subarachnoid space is very difficult to conceive. Arachnoiditis does not seem to be a plausible cause here as this entity de-

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