IMMEDIATE REACTIONS TO SPINAL ANESTHESIA

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IMMEDIATE reactions following the injection of spinal anesthetics occasionally occur which are best explained as being due to the action of the anesthetic agent on the cranial centers but which may be difficult to interpret as being caused by the diffusion of the anesthetic agent through the cerebrospinal fluid to the brain. It has been noted that these reactions become evident in a matter of seconds, following which the level of effective anesthesia becomes established at the desired abdominal or thoracic segments in a matter of minutes. The promptness of the appearance of symptoms suggests that the anesthetic is transported to the brain by the vascular route.

A rich anastomosis exists between cranial and vertebral plexuses of veins. Experimental work of Batson (1) demonstrated that a reversal of current readily occurs in the vertebral plexus of veins under physiologic conditions so that blood in the vertebral plexus may flow into the cranial veins and reach the brain. At each spinal segment the vertebral veins connect with veins of the thoracico-abdominal cavity. In this low pressure and essentially valveless system of veins he proved that an increase in the thoracic or abdominal pressures could reach a height so that blood would reverse from the usual flow through the caval system into the vertebral plexus and cranial veins. The circulation by way of the vertebral veins thus gave a good explanation for the so-called paradoxical metastases from tumors in organs such as the prostate or breast to the spine or brain without necessitating the route involving the pulmonary circuit.

The usual method of administration of spinal anesthetic agents fulfills the requirements that would tend to increase the thoracico-abdominal pressure to cause a reversal of flow of blood from the vertebral plexus of veins into the cranial system. With the patient in lateral position, the spine is hyperflexed and the hips are flexed to place the thighs against the abdomen. Frequently, the patient is tense and may tighten the chest, abdominal muscles and the pelvic diaphragm as well as hold his breath and close his larynx. All of these maneuvers tend to increase the intrathoracico-abdominal pressure. In Batson's experimental work a reversal of flow in the vertebral veins occurred in monkeys anesthetized with sodium amytal by tying a towel around the abdomen. By way of comparison, it seems

probable that many of the patients during the administration of a spinal anesthetic would duplicate or surpass the abdominal hypertension produced by the experimental constricting towel in the monkey experiments, and thus cause the blood in the vertebral plexus to flow into the cranial veins.

If, during the administration of an anesthetic, the spinal needle is inserted into the anterior spinal plexus of veins, the anesthetic agent might be injected directly into the venous circulation, and thus be carried to the cranial vessels and brain in a few seconds. It would be unlikely for this situation to occur unless the spinal needle were mistakenly advanced deeper during the process of injection. The venous plexus, however, could be entered or traumatized by the needle so that when the anesthetic agent was injected in close proximity to the injured vessel wall a significant amount of the agent would be absorbed into the venous circulation. In such a situation, with the patient hyperflexed and perhaps straining so that the venous flow would course from the vertebral to the cranial veins, the absorbed anesthetic agent could be carried directly to the brain. This would result in much less dilution than if the absorbed agent passed from the vertebral venous plexus into the inferior vena cava, the heart, the pulmonic circulation, then back to the heart and eventually to the brain. Thus, a relatively small amount of drug carried directly to the brain would have more pronounced pharmacologic effect than if placed directly into the systemic circulation.

The following case report is given as an example of an immediate reaction to the injection of a spinal anesthetic which is explained by the above mechanism.

REPORT OF CASE

A 24-year-old white woman was admitted to Touro Infirmary, New Orleans, on December 7, 1948. The patient was primiparous, with full term pregnancy in labor on admission to the hospital. She had had rheumatic fever one and one-half years previously. Her pregnancy had been uneventful. The onset of labor was at one a.m. on the day of admission.

The temperature was 98°, pulse 102 beats per minute and respirations were 20 per minute. Physical examination revealed that the patient was nine months pregnant and in active labor. The only physical abnormality noted was a slightly enlarged thyroid gland. Blood pressure was systolic 120 mm. and diastolic 70 mm. of mercury.

Because of cephalopelvic disproportion, the fetal head was still floating after a trial labor of thirteen hours, and the patient was then scheduled for cesarean section. At 5:10 p.m., with the patient in the right lateral position, a number 20 gauge spinal needle was inserted into the subarachnoid space between the third and fourth lumbar vertebrae. The patient was poorly relaxed and showed considerable anxiety concerning the procedure. The spinal fluid was at first bloody but promptly cleared. Ten milligrams of pontocaine (1 cc. of a 0.1 per cent pontocaine solution) dissolved in 2 cc. of 10 per cent dextrose solution was injected at the termination of a uterine contraction. She was promptly placed

in the supine position on the horizontal table. She immediately complained of a sensation of generalized tingling and said that she felt as if she were having trouble breathing. The patient was requested to breathe deeply and she showed full respiratory excursions. Four minutes after the administration of the anesthetic the blood pressure reading showed a systolic pressure of 75 mm. and diastolic pressure of 45 mm. of mercury. The level of sensory anesthesia reached 2 inches above the umbilicus and stabilized at that level. The tingling and suffocating sensations gradually diminished and disappeared in about ten minutes and the blood pressure rose to 110 mm. systolic and 70 mm. diastolic, with little fluctuation thereafter. The operation and postoperative course were uneventful.

COMMENT

Pontocaine solution was administered into the subarchnoid space to a poorly relaxed patient after the spinal puncture needle had traumatized a vein of the vertebral plexus. The pontocaine solution was injected after clear cerebrospinal fluid flowed through the needle. Within a few seconds the patient complained of symptoms suggesting that some of the pontocaine solution had reached the cranial centers. The transient symptoms gradually decreased and disappeared in about ten minutes. The desired level of clinical anesthesia was obtained and the operation was uneventful.

Similar immediate reactions to spinal anesthesia may occur in any patient if some of the anesthetic agent is absorbed into the vertebral plexus of veins and the thoracico-abdominal pressure is increased to produce a flow of blood from the vertebral plexus into the cranial system of veins. The symptoms are transient because a relatively small amount of the drug is absorbed into the venous circulation through the injured vessel wall and because the drug which reaches the brain is diluted and washed out by the circulating blood. If the anesthetic agent reached the cranial centers by diffusion through the cerebrospinal fluid one might expect a more prolonged effect on the cranial centers than is found in these brief and transient reactions.

SUMMARY

Reactions to the administration of spinal anesthetic agents in which the patient presents symptoms indicting that small amounts of the drug reach the brain within a few seconds after the injection of the anesthetic are discussed. Evidence is presented to support the theory that the anesthetic agent reaches the brain by the vertebral plexus of veins rather than by diffusion through the cerebrospinal fluid to explain these immediate and transient reactions. One case is presented.

REFERENCES

1. Batson, O. V.: The Function of the Vertebral Veins and Their Role in the Spread of Metastases, Ann. Surg. 112: 138–149 (July) 1940.
2. Cullen, S. C.: Anesthesia in General Practice, Chicago, The Year Book Publishers, Inc., 1946, p. 132.
3. Lundy, J. S.: Clinical Anesthesia, Philadelphia, W. B. Saunders Company, 1942, p. 225.