Transient Decreases in Respiratory Rate Following Epidural Injections

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Pressure-volume relationships in the spinal space and the interactions between the intracranial and spinal compartments rarely have been studied. While performing epidural anesthesia for routine orthopedic procedures, we were recording epidural pressure. In one patient we noted a decrease in respiratory rate following epidural injection of local anesthetic. We describe our observations on three patients and discuss possible mechanisms for this respiratory response to an epidural injection.

REPORT OF A CASE

Three patients, ASA physical status III, with severe rheumatoid arthritis, were given epidural anesthesia for elective lower-extremity total joint replacement. All were older than 60 years of age and had multiple joint disease. Oral diazepam and im morphine sulfate were given as premedication. In the operating suite, a central venous line was inserted through the right internal jugular vein and diazepam and morphine sulfate given iv until each patient was comfortably sedated. The patients then were placed in the lateral position and a lumbar epidural catheter inserted using a 17-gauge Weiss needle and the hanging drop technique. The catheter was advanced 2 cm beyond the tip of the needle. Etidocaine 1.5% with 1/200,000 epinephrine was then given through the catheter in divided doses. All patients received 3 ml at a time for a total of 12 ml as an induction dose. The patients were positioned and surgery begun. Ninety minutes after the initial dose and at 60-min intervals thereafter, each patient received two additional 3-ml doses of 0.5% bupivacaine. Each 3-ml dose was injected over 9 s and repeated when epidural catheter pressure returned to within 30% of preinjection pressure. This happened within 1.5 min.

The anesthetics were uneventful. All patients had adequate levels of anesthesia and were awake and breathing spontaneously throughout the procedure. After a short stay in the recovery room, each patient was returned to the ward.

Epidural and central venous catheter pressures were measured continuously using Gould® transducers (P29) and a recorder. The epidural catheter was connected to a 10-ml syringe and transducer via a four-way stopcock (fig. 1). The epidural catheter was connected to the transducer; during injection the stopcock was opened to include the syringe in the system. We analyzed changes in epidural and central venous pressure using the recorded traces. Respiratory rate was noted as periodic decrements in the central venous pressure of about 3 cmH₂O.

RESULTS

Patient I showed no measurable decrement in respiratory rate with the first additional 3-ml dose. However, with the second 3-ml dose, respiratory rate changed from 16.5 breaths/min to 10.25 breaths/min, a decrease...
of 38%. Patient 2 became apneic for 23 seconds with the first supplemental 3-ml dose (fig. 2). During this period the patient was awake with spontaneous eye opening. During the second 3-ml injection he again became apneic but took a deep breath on command. Patient 3 showed no change in respiratory rate with epidural injection.

In two of three patients, a decrease in respiratory rate occurred abruptly within 5 s of the start of injection. Respiratory rate returned to the preinjection level within 3.5 minutes postinjection. There was no apparent change in mental or cardiovascular status for any patient during or postinjection. The level of anesthetic blockade was well maintained throughout surgery.

DISCUSSION

Two of the three patients studied had brief periods of slowed or absent breathing at, or shortly after, the time of injection of a 3-ml dose of anesthetic into the epidural space. None of these episodes was considered dangerous or required treatment. There are several possible mechanisms that could lead to the changes in the breathing pattern. First, the decrease in respiratory rate could be the result of administered respiratory depressants. The patients received diazepam and morphine. However, when we observed what appeared to be epidural injection-related changes in respiratory rate, respiratory rate had been stable for at least 15 min and no morphine or diazepam had been given within 30 min. Second, this could be a response to the cool anesthetic solution passing through the catheter taped to the patient’s back. However, the patients did not move, speak, or appear to be aroused by the injection. Third, the change in respiratory rate could be a response to stimulation at or near the site of injection in the epidural space. We know of no relevant evidence for this.

The decreased respiratory rate could have been a response to a brief rise in cerebrospinal fluid pressure (P<sub>CSF</sub>). Shah<sup>1</sup> found that when fluid is injected into the epidural space, the dura is displaced and pressures on either side tend to equalize. During an epidural blood patch, Coombs and Hooper<sup>2</sup> found a threefold rise in subarachnoid pressure after injecting 7.5–10 ml. Thus, increased epidural space pressure, P<sub>epi</sub>, caused a transient rise in P<sub>CSF</sub>, which can cause a Cushing response, which includes shallow respiration plus periods of apnea. These

![Diagram of measurement system](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931410/)

**Fig. 1.** System used for measuring and recording central venous and epidural pressures.

![Graph of central venous and epidural pressures](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931410/)

**Fig. 2.** Central venous and epidural pressure following injection of 3 ml local anesthetic solution into the epidural space. Epidural trace enhanced with ink overlay.
are thought to result from increased intracranial pressure. There is also some evidence that pressure on certain areas of the brain stem and spinal cord, apart from increases in intracranial pressure, can produce Cushing’s response.

We wonder whether the magnitude and duration of the increases in $P_{epi}$ were large enough to account for changes in $P_{CSF}$ sufficient to cause this respiratory response. The increase and decrease of pressure in the epidural space and the associated changes in cerebrospinal fluid (CSF) pressure, are a complex time-dependent function of system mechanics, the system embracing the epidural, subarachnoid, and vascular compartments. We know little about the mechanics of the epidural–subarachnoid–vascular system, but there are a few pertinent reports. In 1950, in an effort to treat sciatica, Evans studied epidural compliance by injecting large volumes of solution into the caudal canal. With 5–15 ml of fluid, pressure increased steeply, then decreased rapidly. Successive injections, up to 140 ml of solution, caused pressures that rose to over 30 cm H$_2$O. His patients complained of headache and pressure in the back and often became anxious and disoriented. It is not clear whether these symptoms were the result of local anesthetic overdose or the result of a transmitted rise in CSF pressure.

Some animals appear to have a very low spinal compliance. When sheep are given 5–10 ml epidural injections they develop transient syncope lasting 60–90 s. In dogs, Gissen et al. found marked and prolonged pressure increases in the epidural space that changed respiratory rate and caused Cushing’s response, hind limb paralysis, cauda equina syndrome, and, when accompanied by systemic hypotension, death.

Usubiaga et al. investigated the rise and fall of epidural pressure after a standard 10-ml injection of local anesthetic solution. Peak pressures varied between 5 and 60 cmH$_2$O. He detected no changes in epidural pressure at a distance of 10–12 vertebrae from the site of injection. Compliance of the epidural space decreases with increasing age. In young subjects $P_{epi}$ increased rapidly upon injection but also fell off rapidly afterward. In older patients the rate of rise was much faster; peak pressure was higher; and the decrease in pressure was more gradual, taking much longer to return to baseline.

Our three patients were at least 60 years old. Thus, we would expect them to have low epidural space compliance, with a rapid rise and relatively fast fall in epidural pressure after epidural injection. We suggest that even small injections of fluid into the epidural space can cause a transient increase in cerebral spinal fluid pressure ($P_{CSF}$) large enough to cause a reflex decrease in respiratory rate. We do not know what the actual changes in $P_{CSF}$ might be nor do we know the range of individual variation in the response. Spinal compliance is unknown. All of these factors might contribute to the respiratory rate changes described above.

The authors thank Maud Naroll, Ph.D., for editorial assistance.

REFERENCES

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