Immediate Respiratory Arrest after Caudal Epidural Sufentanil

To the Editor,—Life-threatening early respiratory depression requiring treatment has been reported after the epidural administration of sufentanil, but only after repeated injections containing 50–75 μg.1,2 A case is presented in which respiratory arrest occurred within 3 min after single caudal epidural administration of 50 μg of sufentanil.

A 34-yr-old female, ASA physical status 1, 56 kg, was scheduled for elective surgical repair of a perineal fistula in outpatient surgery under caudal epidural anesthesia. The solution for caudal epidural administration was prepared by adding 1 ml containing 50 μg of sufentanil to 20 ml bupivacaine 0.25% with epinephrine 1:200,000. Caudal epidural block was performed with the patient in the prone position. ECG, pulse rate, blood pressure, and oxygen saturation were monitored by automatic devices throughout the procedure.

Entry of the sacral canal was easily established using a 23-G short-bevelled caudal needle. No blood or cerebrospinal fluid could be aspirated and the solution of bupivacaine, epinephrine, and sufentanil was slowly injected over a 60-s period. After every 5 ml an attempt for aspiration was made, which proved negative at all times. Heart rate remained constant between 70–75 beats per min throughout the procedure. Immediately after injection the patient was turned into the supine horizontal position. At that time she complained of a light-headed feeling and became drowsy. It was noted that she had pinpoint pupils and during the next minute her respiratory rate decreased to a complete respiratory arrest.

During the next minute the patient remained apneic and did not respond to verbal commands to take a deep breath; peripheral cyanosis became apparent, oxygen saturation decreased from 97 to 85% and ventilation via face mask was instituted with a mixture of 35% oxygen and 65% nitrous oxide, leading to an immediate increase in oxygen saturation to 99%. Blood pressure decreased slightly from 120/80 to 110/75 mmHg, heart rate remaining constant at all times. The operation was commenced with the patient in the lithotomy position. Fifteen minutes after completion of the caudal epidural injection, two increments of 0.1 mg of naloxone were given intravenously, spontaneous respiration returning after the second dose. Nitrous oxide was discontinued after which the patient regained full consciousness. She experienced no pain and the operation was completed with the patient awake and breathing room air.

Upon arrival at the recovery, the level of sensory blockade as measured by temperature discrimination was at the twelfth thoracic dermatome. Motor blockade of the lower limbs for both legs was grade 1 on the 0–3 scale as described by Bromage.3 Two hours after the caudal epidural injection the patient was able to raise the extended leg on both sides. Four hours after the caudal epidural injection sensation in both legs was restored, a tingling feeling in the perineum the only remaining sign of a receding caudal block.

The patient was observed until 8 h after the caudal epidural administration of the sufentanil/bupivacaine solution. Although she experienced no pain, all signs of the caudal block had worn off and she was discharged from the hospital.

Although the rapid onset of respiratory arrest in combination with the observation of pinpoint pupils after caudal epidural injection of a solution containing 50 μg of sufentanil suggests iv injection, this is not likely for the following reasons. First, before, during, and after caudal epidural injection aspiration was negative for blood. Second, when using a solution containing epinephrine 1:200,000, iv injection of as much as 3 ml should result in an increase in heart rate,4 and the heart rate in our patient remained constant. Moreover, no central nervous system or cardiac symptoms related to iv injection of bupivacaine were observed. Third, the patient developed a symmetrical block with a sensory level of blockade reaching the twelfth thoracic dermatome, and bilateral partial motor blockade of the lower limbs, as would be expected after the caudal epidural administration of 21 ml of a solution containing 50 mg of bupivacaine. The only possible explanation remaining therefore must be an immediate, massive, selective vascular absorption of sufentanil from the site of injection, despite the fact that the solution employed by us did contain epinephrine.

In a study comparing arterial plasma concentrations of meptivacaine 1 or 2% with or without epinephrine 1:200,000 after intercostal, brachial plexus, sciatic femoral, lumbar epidural, and caudal epidural block,
it was demonstrated that systemic uptake of mepivacaine 2\% was higher after caudal epidural when compared to lumbar epidural administration, the difference being more pronounced for the epinephrine-containing solutions. This observation may provide an explanation for the early and complete respiratory arrest seen in our patient.

RUDOLF STEINSTRA, M.D.
FRANS VAN POORTEN, M.D.

Department of Anesthesiology
Reinier De Graaf Gasthuis
Reinier de Graafweg 11
2625 AD DELFT
The Netherlands

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REFERENCES

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The Measurement of Expired Oxygen as Disconnection Alarm

To the Editor:—The method reported by Doctors Knaack-Steinberger and Thomson was long ago described as an unreliable method to detect disconnections, except under ideal conditions. At other times, especially when the disconnection site is covered with drapes, the anesthetic mixture collects there and is drawn back into the inspiratory circuit by a falling bellows ventilator, where little loss of oxygen may be detected. With a rising bellows ventilator, flow terminates in the expiratory limb of the circuit with similar results.

Modern ventilation monitors, such as carbon dioxide monitors and exhaled volume monitors, specified for anesthesia systems by the ASTM F 29 standard,* are readily available and have proven their reliability as disconnection alarms. Users should not depend on a less-reliable device for this critical function.

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In Reply.—We do agree with Dr. Spooner that users should not depend on less-reliable devices to detect disconnections within the anesthetic circuit. However, Spooner did not prove our method to be unreliable but only stated that not all disconnections occur under ideal circumstances and that in the case of a tracheal tube disconnection under drapes, detection may not be made with an oxygen analyzer. We have data to show that our method is reliable, even when a disconnection occurs under drapes (table 1).

The data shown in table 1, columns I and III, were the figures that we published. The data shown in columns I* and III* were collected at the same time that we did the original work. In columns I* and III*, we show the data that we received when we disconnected the circle under drapes with an additional covering of a pillow to make the area even more airtight. These circumstances are somewhat difficult to standardize; therefore, we did not intend to publish these data. Nevertheless, the detection of a disconnection is reliable even under these difficult versus ideal conditions.

The data show that the reliability of the method changes considerably when the alarm settings are not close to the expected expiratory oxygen concentration as stated in the Discussion of our paper.

We use the oxygen measurements in the expiratory limb to monitor the amount of oxygen that our patients received, not only what the machine delivers. In the same way, we use the spirometer in the expiratory limb to monitor the amount of volume that the patients re-