Anesthesiology
62:688–690, 1985

Epidural Meperidine for Control of Autonomic Hyperreflexia in a Paraplegic Parturient

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Autonomic hyperreflexia (AH) is a syndrome of massive reflex sympathetic discharge that occurs in patients with chronic spinal cord lesions above the major sympathetic splanchnic outflow (T4–T6).[1] Eighty-five per cent of patients with cord transection above T6 will exhibit this reflex; it is unlikely to be associated with a transection below T10. The reflex sympathetic response can be initiated by cutaneous or visceral stimulation below the level of spinal cord transection. Distension of a hollow viscus such as the bladder or rectum is a common stimulus.[1–3] Also, AH can be triggered by uterine contractions during labor in parturients with cord transection.[4–7] This report shows that epidural meperidine may succeed in controlling AH during labor in the paraplegic parturient.

REPORT OF A CASE

The patient was a 25-year-old parturient who developed paraplegia three years earlier following a bullet injury of the spinal cord at the T6 level. Neurologic examination revealed complete loss of sensory and motor functions below the level of the lesion. On admission, arterial blood pressure was 110/70–130/80 mmHg and heart rate 70–80 bpm. ECG showed normal sinus rhythm, and chest roentgenogram was normal. An iv infusion of lactated Ringer's solution was started at a rate of 10 $ml \cdot kg^{-1} \cdot hr^{-1}$. The membranes ruptured spontaneously, and adequate and regular uterine contractions began. Although uterine contractions were painless, they were associated with paroxysms of signs suggestive of AH, such as headache, flushing of the face, hypertension to 180/100–200/110 mmHg and bradycardia to 50–60 bpm. After 30 min of having disturbing paroxysms of AH associated with uterine contractions, epidural meperidine was given. The technique was explained to the parturient, and an informed consent was obtained. In the lateral position, a 17-gauge Touhy needle was inserted in the epidural space at the second lumbar interspace. Preservative-free meperidine, 100 mg, diluted in 10 ml preservative-free normal saline was injected epidurally. The patient then was positioned in the left lateral decubitus position. Ten minutes following epidural meperidine, the patient became comfortable and sleepy. Also, uterine contractions were maintained without associated fluctuations of the blood pressure and heart rate, which remained near control levels throughout the progress of labor (fig. 1). After 2 h, the cervix was fully dilated, and a baby girl with a normal Apgar score was delivered by episiotomy and forceps extraction. No postpartum complications attributable to epidural meperidine were observed.

COMMENT

Most paraplegic women will resume prior menstrual patterns within about a year following spinal cord transection. Sexual relations usually are psychologically rewarding, despite sensory loss, and the fertility rate is relatively high.[4,5]

The uterus has the capacity to contract in labor, even when the nerve supply has been severed. When the cord is transected above the tenth thoracic segment, paraplegic women, although they do not feel labor pain, can have normal uterine contractions. Also, the progress of labor is often rapid.[4,5]

During the first stage of labor, pain is primarily due to uterine contractions and dilatation of the cervix. Impulses travel via visceral afferent fibers accompanying the sympathetic nerves and enter the spinal cord at T10–T12 and L1 spinal segments. In the late first stage and in the second stage, stretch of the perineum produces additional pain by impulses that travel via the pudendal nerves and enter the spinal cord at S2–S4 segments. In paraplegic parturients, labor is usually painless if the cord lesion is above the level of the tenth thoracic vertebra. When the cord lesion is above T4–T6, uterine contractions, although painless, can initiate autonomic hyperreflexia as a result of a spinal reflex sympathetic outflow that is uninhibited by the central nervous system.[4,5] Uterine contractions will be associated with paroxysms of hypertension and headache, which may be misdiagnosed as preeclampsia.[5] The hypertensive episodes stimulate the baroreceptors, resulting in bradycardia and vasodilation above the level of the spinal cord transection.[4–7]

Autonomic hyperreflexia traditionally has been attributed to massive unchecked reflex sympathetic discharge that occurs in patients with high spinal cord lesions. Mathias *et al.* presumed that this is due to lack of restraining supraspinal vasomotor reflexes.[8] However, plasma catecholamines (epinephrine or norepinephrine) are subnormal in patients with cord transection, and during AH the level of norepinephrine increases but still does not exceed the resting level of control normal patient.[3] These findings demonstrate that patients with chronic spinal cord transection have a subnormal sympathetic tone even during an attack of AH. Thus,

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Received from the Department of Anesthesiology, American University of Beirut, Beirut, Lebanon. Accepted for publication December 21, 1984.

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Key words: Anesthetic Techniques: epidural meperidine; sympathetic nervous system: autonomic hyperreflexia.

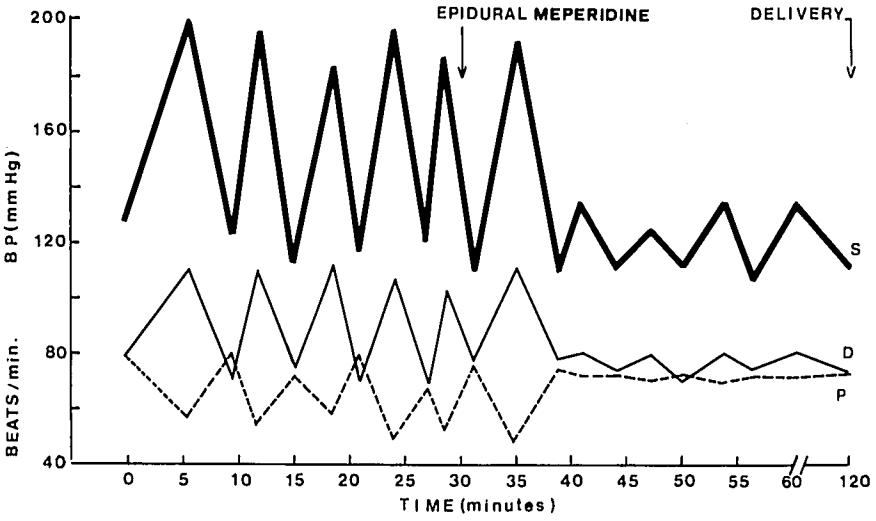

FIG. 1. A graphic recording of blood pressure and heart rate changes before and after administration of epidural meperidine. S = systolic blood pressure; D = diastolic blood pressure; P = heart rate.

patients with chronic spinal cord transection have both somatic and autonomic denervation, resulting in a supersensitivity response to acetylcholine and succinylcholine at the neuromuscular junction[9,10] and to catecholamines at the adrenergic receptors. During AH, exaggerated vasoconstriction will occur below the level of cord transection, which is mediated via reflex release of norepinephrine with predominantly alpha-adrenergic smooth muscle receptor activity. This will result in a marked increase in arterial blood pressure, which is not buffered effectively by the baroreceptors via the limited intact sympathetic outflow above the level of cord transection.

Epidural analgesia has been used to prevent AH in parturients with chronic spinal cord transection.[6,7] However, the level of anesthesia is difficult to control in such patients. Also, epidural and spinal anesthesia result in a nonselective motor, sensory, and sympathetic blockade. Extensive sympathetic block in such patients can be complicated with serious hypotension, bradycardia, and even asystole.[2]

Epidural or spinal narcotics, in contrast with epidural or spinal anesthesia, can produce "selective" blockade of the opiate receptors in the substantia gelatinosa of the dorsal horn of the spinal cord.[11,12] Nociceptive impulses evoked by painful stimuli enter the dorsal horn of the spinal cord at the dorsal root entry zone and terminate largely within the outermost laminae of the spinal gray matter, the marginal layer, and the substantia gelatinosa, respectively. The spinal effect of opiates selectively would antagonize the discharge evoked by high-intensity peripheral stimulation of the spinal dorsal horn neurons and inhibit both the rostral transmission and the spinal reflexes evoked by such noxious stimuli.[11] All current data indicate that the efferent sympathetic fibers within the blockade area are unaffected.[13-15]

Experimental work in spinal cord-transected cats pointed out that the opiates can act on the spinal cord of transected cats, independent of supraspinal control, to modify the transmission of information concerning noxious stimulation of peripheral receptors.[16,17] In our patient having spinal cord transection, epidural meperidine could control AH during labor without inducing any changes of the arterial blood pressure or heart rate. This effect suggests that epidural narcotics in humans, similar to experimental animals, can block the opiate receptors of the spinal cord below the level of transection and hence can inhibit the autonomic reflexes initiated by noxious stimuli.

Epidural meperidine was preferred to morphine for control of AH in our paraplegic parturient. The rate of absorption of meperidine from the epidural space is similar to that of lidocaine and, like lidocaine, has a rapid onset of action after epidural use that coincides with early meperidine concentration of cerebrospinal fluid (CSF).[15] In contrast, morphine has a lower lipid solubility than meperidine. It has a slower onset of action after epidural use, which coincides with delayed peak concentrations of morphine in CSF, and its relative hydrophilicity results in slower efflux from CSF and greater rostral spread to the brain.[18]

Epidural and intrathecal narcotics previously have been recommended in normal patients to relieve chronic pain,[19] postoperative pain,[14,15] and labor pain.[20,21] The present case report suggests that epidural meperidine also can be used during labor to control AH in parturients with chronic spinal cord transection, while sparing the resting sympathetic tone, which already is compromised by the cord transection.

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Anesthesiology
62:690–692, 1985

Pneumocephalus with Headache during Spinal Anesthesia

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Pneumocephalus is not an uncommon occurrence following air pneumoencephalography,[1] pneumomyelography,[2] or brain surgery, particularly when nitrous oxide is used.[3] While air usually is reabsorbed after 2 days, in some patients it may take up to 7 days.[4] Also, dural tap for spinal anesthesia has been suggested as a possible cause of pneumocephalus,[5] but it has not been documented. This case provides evidence that pneumocephalus can result from spinal anesthesia and a small volume of air.

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Received from the Department of Anesthesiology, RN 10, University of Washington School of Medicine, Seattle, Washington 98195. Accepted for publication December 21, 1984.

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Key words: Anesthetic techniques: spinal. Complications: headache, pneumocephalus.

Report of a Case

A 61-year-old man, weight 63 kg, height 185 cm, was hospitalized for more than 2 months, receiving treatment for a chronic right trochanteric bursal infection. He was classified as ASA physical status III on the basis of malnutrition, anemia (Hct 25%), stable angina, a previous myocardial infarction, chronic obstructive pulmonary disease, and albumin 2.9 (normal range 3.5–5.2 g/dl) with normal serum electrolytes. Because of low back pain, several myelograms had been performed. Medication included morphine, hydroxyzine, and nitroglycerin. A trochanteric bursectomy with debridement and a gluteal thigh flap had been done with hypobaric spinal anesthesia. He was scheduled for a flap debridement and closure.

Another hypobaric spinal anesthesia was planned, using 7 ml sterile distilled water containing 7 mg tetracaine and 0.2 mg epinephrine. Because of the relatively large volume of local anesthetic solution, the technique to be used requires the removal of approximately an equal amount of cerebrospinal fluid (CSF) before its injection.[6]

After iv fluids had been started and one unit of whole blood infused, the patient was placed in the left lateral position. A 26-g single-use spinal needle was inserted into the subarachnoid space via L3–4 vertebral interspace. CSF dripped slowly from the needle, but only 0.5 ml of it could be aspirated. In an effort to obtain a free