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Title : EPIDURAL MORPHINE IN DEXTROSE VS. BUPIVACAINE IN LABOR

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Introduction: The recent report of Behar et al (1) describing successful results with low dose epidural morphine in the management of acute and chronic pain, stimulated us to study the use of epidural morphine in labor. Several opiate receptor sites exist in the spinal cord and brain and endogenous ligand believed acting at these sites has been identified. Snyder (2) found that affinity of opiate receptors for exogenous drugs is related to receptor "sodium state." When receptors are in sodium binding conformation, binding of opiate agonists is reduced and of narcotic antagonists enhanced. Pure opiate agonists become 12-60 times weaker in the presence of sodium. Most investigators have diluted epidural morphine in saline.

To avoid any negative effects of sodium we diluted morphine in dextrose for lumbar epidural analgesia (LEA) in labor, and compared it with bupivacaine.

Methods: Sixteen parturients with a singleton fetus in cephalic presentation, requesting LEA, were studied. Subjects weighed > 45 kg or < 100 kg and were healthy in all respects.

The Clinical Research Practices Committee approved the protocol and each subject gave informed consent. We diluted morphine sulphate (15 mg) with 5 percent dextrose to provide six 10 ml syringes each containing morphine 0.25 mg/ml (.025%). Bupivacaine 0.25% was prepared in six 10 ml syringes. Random selection determined the choice of drug. Neither investigator nor subject was aware of drug identity. We instituted LEA in the active phase of labor, injecting 8 ml of study drug initially.

Pulse and blood pressure (Riva Rocci method) were recorded before establishing LEA and 3, 5, and 10 mins after injection. We scored pain on a scale from 1 to 5 at the same intervals and measured thigh skin temperature with a surface probe. Fetal heart rate and uterine activity were continuously recorded. 2 ml increments of solution were permitted at 10 min intervals to secure pain relief. If pain relief failed after 3 increments (or earlier at investigator's discretion) 2% 2-chloroprocaine was substituted for the test solution. When test solution gave relief we plotted and recorded dermatome levels at 30 and 60 mins. Through labor we repeated 8 ml doses as required and made serial observations. Subjects received 15 ml of the study drug, while sitting, for perineal analgesia at delivery. We recorded any need for additional analgesia.

Neonatal status was assessed by cord blood gases, Apgar scores at 1 and 5 mins, and neurobehavioral assessment at 2-6 and 20-26 hours after delivery.

Results: Bupivacaine provided pain relief more consistently than morphine (See Table). Two morphine subjects appeared to receive adequate pain relief for labor. All other morphine subjects received 2-chloroprocaine for failed analgesia. Mean duration of chloroprocaine was longer than anticipated (83 ± 5.5 min).

Blood pressure decreased significantly 5 mins after bupivacaine administration, gradually increasing with time. Morphine produced no significant change in blood pressure. After bupivacaine skin temperature increased within 3 mins and remained significantly elevated for 60 mins. Morphine caused no temperature change (See Table). Numbness to pinprick occurred in morphine subjects with improved pain scores and in all parturients receiving bupivacaine.

A significantly greater incidence of variable decelerations occurred during first stage labor in bupivacaine subjects.

Apgar scores and cord blood gases were similar in both groups.

The first neurobehavioral examination revealed significant differences, in the examiner's general assessment, "borderline" status predominating in the morphine group.

Conclusions: In the doses employed bupivacaine is superior to morphine in dextrose for LEA. Like Behar we failed to demonstrate sympathetic blockade with morphine which might explain poor results with 1st stage labor pain.

Epidural morphine has been shown to provide analgesia after 2nd trimester induced abortion (3). The distension of epidural veins at term, because of IVC compression (with venous uptake, restribution and early hepatic metabolism of morphine diminishing CSF levels) might also explain our findings.

The longer duration of analgesia of 2-chloroprocaine after morphine perhaps suggest synergism.

References:

1. Behar M, et al: Epidural Morphine in Treatment of Pain. Lancet (1979) 1:527.
2. Snyder SH, et al: Opiate receptors and internal opiates. Sci Am (1977) 236:44.
3. Magora F, Olshwang D, Eimerl J, et al: Observations on Extradural Morphine Analgesia in Various Pain Conditions. Br J Anaesth (1980) 52:247.

	Morphine n=8	Bupivacaine n=8
Pain Relief (Study Drug)	2	7*
Syst. (Control	116 ± 5.8	122 ± 6.1
BP (5 Min	112 ± 4.9	107 ± 4.7 *
(10 Min	113 ± 5.5	108 ± 5.1
Diast. (Control	47 ± 11.0	75 ± 5.3
BP (5 Min	28 ± 10.7	48 ± 11.6 *
(10 Min	47 ± 10.2	55 ± 8.9
Skin (Control	32.9 ± 1.5	33.3 ± 0.3
Temp. (3 Min	33.2 ± 0.5	33.7 ± 0.2 *
(60 Min		34.2 ± 0.5 *
1st Stage Variables	0	5 *
Neonatal assessment	Borderline 6	0 *
	Normal 2	8

* P < 0.05 Values Mean ± SEM