TITLE: EPINEPHRINE AND CAUDAL EPIDURAL ANESTHESIA IN INFANTS: ONSET, DURATION, AND HEMODYNAMIC EFFECTS

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Epinephrine increases the duration of caudal analgesia in older infants and children.[1] We attempted to determine if 1:200,000 epinephrine would prolong the duration of caudal anesthesia in conscious infants.

Materials and Methods: Following IRB approval and written parental consent, infants were randomized to receive 1 ml/kg of 0.375% bupivacaine with (E) or without (P) 1:200,000 epinephrine. Our technique of caudal anesthesia in conscious infants has been described previously.[2] Onset and duration of anesthesia were defined as loss and return of withdrawal to toe-pinch. Vital signs were recorded immediately before and 5 minutes after caudal block. Demographic data and vital signs are presented as mean ± SD. Results are presented as mean ± SEM. Data were analyzed using analysis of variance and paired or unpaired Student's T tests. Results were considered significant for $P<0.05$ with appropriate Bonferroni correction for multiple comparisons.

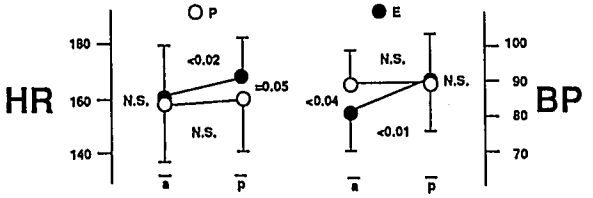
Results: Twenty-nine subjects were enrolled, 19 in E and 10 in P. There were 2 block failures; both occurred in P. Data were also available on 33 other infants who had received caudal anesthesia (22 P and 11 E). While allocation of these infants to P or E was neither randomized nor blinded, duration of anesthesia was determined by PACU nurses who were not otherwise involved with the study. Demographics are shown in the Table. Onset of anesthesia occurred in 8 ± 1 min in P and 10 ± 1 min in E (N.S.) Median anesthetic level was T_4 in both groups. Our overall experience with caudal anesthesia in infants did reveal a significant difference (Table); however, repeated pairwise Student's T tests with Bonferroni correction failed to reveal any pairs which differed significantly. Analysis of the pooled data did reveal a significant increase in duration with E. Vital signs before and after caudal block are shown in the Figure. Heart rate and systolic blood pressure increased after caudal block with E.

Conclusions: Our study population made recruitment of large numbers of subjects difficult; nonetheless, our data shows an increase in duration from 120 min with P to 150 min with E. This is consistent with reports of prolongation of caudal analgesia in children receiving E.[1] We have also shown small, but significant, increases in heart rate and blood pressure after caudal block in infants receiving E.

References:

1. Warner et al. **Anesth Analg** 1987; 66:995
2. Gunter et al. **J Ped Surg** [in press]

	Plain		Epinephrine		
	Blind	Unblind	Blind	Unblind	P
EGA (wk)	44 ± 7	46 ± 11	45 ± 9	42 ± 3	N.S.
Wt (kg)	3.4 ± 0.9	3.6 ± 1.4	3.7 ± 1.2	3.4 ± 1.0	N.S.
Dur (min)	122 ± 8	118 ± 8	147 ± 11	154 ± 11	<0.05
Pooled	119 ± 6		149 ± 8		<0.005



TITLE: EFFECT OF CONCOMITANT ANALGESIC THERAPY ON THE AWAKENING CONCENTRATION OF HALOTHANE IN CHILDREN

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The alveolar concentration of an anesthetic when a patient first opens his eyes is termed the MAC-awake. This study was designed to determine the MAC-awake in children during halothane anesthesia and to determine if (1) analgesic doses of morphine, or (2) regional blockade significantly affects MAC-awake.

After obtaining IRB approval and parental consent, 70 healthy ASA I or II children scheduled for elective surgery under general endotracheal anesthesia were studied. No premedication was administered. Following a mask induction with nitrous oxide (N_2O) and halothane, all patients underwent tracheal intubation facilitated with atracurium 0.5 mg/kg. Anesthesia was maintained with halothane in an air-O_2 mixture. For supplementation, children received either morphine, 0.1 mg/kg IV, or a caudal block with 0.25% bupivacaine, 0.6 ml/kg, with epinephrine 1:200,000. The control group received no analgesic supplements. At the end of the study neuromuscular block was reversed with atropine and edrophonium, halothane was discontinued and the circuit flushed with high flow oxygen. A premeasured catheter was passed to the distal end of the tracheal tube and connected to a Perkin Elmer MGA 1100 mass spectrometer. The end-tidal concentration of halothane when a patient first opened his/her eyes was recorded as the awakening concentration (Table 1).

Table 1

Age Range (yr)	Awakening Concentration		
	Control	Morphine	Bupivacaine
0.5-2.5	0.15±0.02%	0.13±0.02%	0.11±0.03%
2.5-7	0.15±0.01%	0.16±0.02%	0.13±0.01%
>7	0.16±0.01%	0.17±0.01%	0.15±0.01%

We determined the awakening concentration when the inspired concentration was zero rather than at a constant alveolar concentration. (1) This method may underestimate MAC-awake, but provides more clinically relevant data. (2) The lack of effect of morphine and regional anesthesia on the awakening concentration of halothane may indicate that analgesia and loss of consciousness occur by different mechanisms during halothane anesthesia.

In conclusion, this study would suggest that the awakening concentration of halothane in children is not affected by an analgesic dose of morphine or by regional anesthesia.

References

1. Stoelting RK et al: Anesthesiology 33:5, 1970
2. Gross JB et al: Anesth Analg 67:27, 1988