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Title : HEMODYNAMIC STABILITY WITH EPIDURAL ANESTHESIA?

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Introduction. Epidural anesthesia is reported to cause only minimal change in cardiovascular hemodynamics unless excessive blockade of sympathetic outflow occurs. The basis for this concept is the observation of a stable heart rate and arterial blood pressure during epidural anesthesia.[1] The purpose of the present study was to determine the completeness of hemodynamic stability during epidural anesthesia by measuring cardiac output and related hemodynamic parameters using a Swan-Ganz thermal dilution catheter. The sympatho-adrenal response to epidural anesthesia was additionally measured and correlated with the hemodynamic responses.

Methods. Eight adult male beagle dogs were prepared with a venous and femoral artery catheter, a thermal dilution Swan-Ganz catheter, and a femoral vein catheter positioned cephalad to the adrenal gland outflow. An epidural catheter was introduced in L_3-L_4 interspace using the loss of resistance technique and advanced 2 inches cephalad. Using the paw-withdrawal response to toe clamping, it was determined in each dog that the sensory level achieved following 7.0 cc of 1½% lidocaine was T_8-T_{10}. To eliminate erratic hemodynamic values resulting from body movement we found it necessary to maintain the dogs on enflurane anesthesia during the experiment. A complete hemodynamic profile was determined during a control period, after injection of saline and 15, 30, 60, 90 and 120 minutes after the injection of lidocaine, and in non-lidocaine treated "sham" dogs.

Results. Epidural anesthesia caused no change in heart rate, arterial blood pressure, central venous and wedge pressure or stroke work index, however, a significant drop in cardiac output and stroke volume occurred 15 minutes after the injection of licocaine, the time of maximum analgesia (Table). Systemic vascular resistance was found to decrease over the duration of the block, peaking between 60 and 90 minutes after the lidocaine. 120 minutes after lidocaine the hemodynamic parameters returned to the baseline level. There was no evidence of sympathetic-adrenal response during the epidural block. Epinephrine and norepinephrine levels remained unchanged over the 120-minute experimental period.

Discussion. This study has demonstrated significant hemodynamic change during epidural anesthesia which is not evident by the usual heart rate and blood pressure monitoring. The lack of sympatho-adrenal response to epidural anesthesia would seem to account for the stability in heart rate and arterial blood pressure, since the opposite has been found by us to occur with spinal anesthesia. The finding of diminished cardiac output during epidural anesthesia raises a question as to whether epidural anesthesia is superior for surgical procedures of the lower extremity in patients with cardiovascular and respiratory diseases. The need for carefully controlled hemodynamic studies in man during epidural anesthesia is indicated from these results.

TABLE

EPIDURAL

	Saline	Inj ↓ 15	30	60	90	120	min post Mean Values
CI	4.5	3.6*	3.8*	3.9*	3.7*		4.2
SV	15.3	12.4*	13.3*	12.9*	12.6*		14.8
HR	144	142	140	145	145		144
Wedge	8.2	10.2	10.3	10.2	10.3		9.2
BP	77.8	75.8	81.7	85.3	84.2		79.1
CVP	2.6	2.3	2.4	3.1	2.3		2.4
TVR	1358.6	1681.5	1735.1	1817.7*	1825.3*		1420.4
SWI	30.8	23.5	26.7	27.8	27.5		28.9
NE+	1.02	1.14	1.24	1.10	1.18		1.11
EPI+	1.78	2.43	2.37	2.25	2.04		1.82

*Statistically different from saline control
$p < .05$ (Paired t)
+ = ng/ml plasma
Lidocaine dose = 8.88 mg/kg

1. Bromage, P.R.: Epidural Analgesia. W.B. Saunders Co., Philadelphia 1978