Title: EPIDURAL ANESTHESIA SIGNIFICANTLY REDUCES RESTING PLASMA CATECHOLAMINE LEVELS IN AWAKE DOGS

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Introduction: Many investigators have shown that epidural anesthesia blocks the "stress hormone response " to surgical pain. However, the effect of epidural block on circulating catecholamines in the resting state is much less well known. Investigation of this effect may elucidate control mechanisms involved in the maintenance of circulating catecholamine levels. Previous investigators have shown a reduction in circulating catecholamines in pentobarbital-anesthetized dogs during epidural blockade (1,2). Because of general anesthesia, the extent of neural blockade could not be evaluated definitively in these studies. Furthermore, baseline catecholamine levels were high, biasing results towards a reduction of plasma catecholamines with any additional anesthetic. To avoid the confounding influences introduced by the use of general anesthesia and experiment-related stress, we investigated the effect of epidural anesthesia from S5 to at least T1 on plasma norepinephrine(NE) and epinephrine(E) in trained, awake dogs, who were accustomed to the investigators and the laboratory environment.

Methods: We studied seven mongrel dogs (20-27 kg) , which were trained to lie unrestrained in the lateral decubitus position during the experiments. Under fluroscopic control, an epidural catheter was percutaneously inserted at L6/7 on the day before, under a brief thiopental (15 mg/kg) anesthetic. On the next day , cannulae were inserted into a peripheral vein and into a chronically exteriorized carotid artery for arterial blood sampling and continuous blood pressure (BP) measurement. Heart rate (HR) and respiratory rate (RR) were also recorded. The dogs were allowed to reach a calm state, judged by a decrease in RR to < 25/min, HR< 80/min, spontaneous eye closure, and appearance of respiratory arrhythmias. When the animals attained these criteria, 8-10 ml of 2% lidocaine were injected into the epidural catheter. Blood for determination of plasma NE and E (radio-enzymatic method) was drawn 10 min after cannulae placement when the animals were alert (A), after they reached the calm state(C), 20 (t20) and 45 (t45) minutes after epidural lidocaine injection. ABG's were drawn at C and t20. Blood for plasma lidocaine determination (fluorescence polarization assay) was drawn at t20. The data were analyzed using a repeated measures analysis of variance and, where necessary, the Newman-Keuls test for multiple comparisons. A $p<0.05$ was considered significant.

Results: All dogs developed a motor block extending to lower cervical segments. The membrana nictitans was also paralyzed in all dogs. Plasma lidocaine levels at t20 were 1.8 ±0.3ug/ml (mean ± S.D.) Plasma levels of NE and E did not differ between the "alert" and "calm" states despite significant changes in HR and RR (see fig). However, following epidural block, plasma NE and E fell significantly, to 14-35% of controls. BP, RR and ABG's did not change after epidural block , but HR rose(see table).

Discussion: Our findings indicate that sympathetic blockade can significantly reduce circulating catecholamines below those found in the unstressed awake state. This suggests that epidural anesthesia may inhibit systemic catecholamine release under tonic sympathetic control. Epidural block to mid-cervical levels in all dogs was confirmed by motor block of the hindlegs, partial motor block of the forelegs, and paralysis of the membrana nictitans, the latter criterium indicating a sympathetic block to at least T1(3). Therefore all dogs had complete block of spinal sympathetic outflow, including a block of the adrenal glands, which in dogs are innervated by sympathetic fibers originating from T4-L1(4).

Although the presence of high plasma lidocaine concentrations may be associated with reduced plasma NE and E levels , the range of plasma lidocaine levels measured in our dogs has not been shown to have a significant effect on plasma NE and E(5). Previous studies investigating the effect of epidural anesthesia on catecholamine levels in untrained, pentobarbital anesthestized dogs reported higher control HR, BP and plasma NE levels than those observed in our animals. This, together with the observed simularity of catecholamine levels in both the "alert" and the "calm" states, indicates that stress was attenuated in our controls owing to careful conditioning of the animals. The experimental conditions of this study eliminated the confounding influences of general anesthesia and reduced the likelihood that high baseline stress biased our results.

The observation that plasma NE and E was reduced significantly below concentrations measured in calm, unanesthetized animals confirms the findings of previous investigators. Furthermore, it indicates that epidural anesthesia lowers canine plasma NE and E to basal levels below those found in the resting state, presumably representing secretion of NE and E independent of spinal sympathetic innervation.

References:
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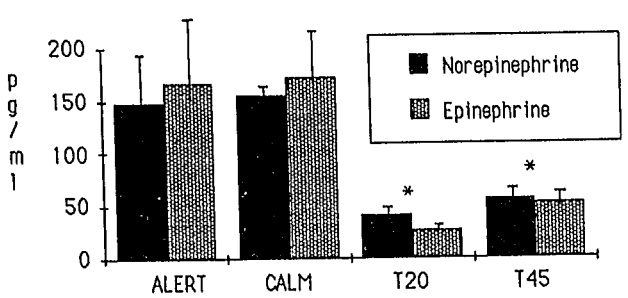
Fig: PLASMA CATECHOLAMINE LEVELS (±SEM) BEFORE AND AFTER EPIDURAL BLOCK TO T1



* p<0.05 vs. calm

Table: Cardiorespiratory parameters (Mean±SD)				
n=7	alert	calm	t20	t45
HR(bpm)	102±22	61±10	106±4*	99±7*
MAP(torr)	107±16	94±19	89±16	96±22
RR(r/min)	39±26	13±4	12±3	14±6
pH	-	7.40±0.02	7.37±0.03	-
PaCO2(torr)	-	36.0±1.9	38.8±5.1	-
PaO2(torr)	-	93.4±10.2	93.8±6.6	-

*p<0.01 vs. calm

This work was supported in part by USUHS grant #RO-8020.