LIDOCAINE AND EPINEPHRINE IN DOGS

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Introduction. It is generally assumed that epinephrine by causing vasoconstriction, minimizes the absorption of the local anesthetic from the subarachnoid space, and thereby prolongs the duration of the subarachnoid anesthesia. Axelsson and Widamen measured blood lidocaine levels in two sets of patients following spinal anesthesia with lidocaine and lidocaine with epinephrine 1. However, when two groups of patients are used variations in cerebrospinal fluid (CSF) kinetics, neural sensitivity, vascular absorption, drug metabolism, distribution and elimination cannot be controlled and therefore the observed variations in the blood levels or duration of neural blockade are not necessarily valid. In view of this consideration we undertook this study to determine the effect of addition of epinephrine on the absorption of lidocaine from the subarachnoid space as well as on the duration of anesthesia, utilizing the same set of dogs, at two different times.

Methods. Ten mongrel dogs (17 to 24 kg) were sedated with intravenous ketamine (10 mg/kg). Six dogs then received a lumbar subarachnoid injection containing 30 to 40 mg of lidocaine. One week later, the same dogs were again sedated and given a subarachnoid injection containing the original dose of lidocaine plus 0.3 mg of epinephrine. In four dogs, the lidocaine plus epinephrine was injected first followed l week later by lidocaine alone. The regres-sion of motor block produced by the subarachnoid injection of lidocaine with or without epinephrine was measured by noting the time taken for the dogs to stand and walk unassisted. Arterial blood for measurement of lidocaine concentrations was obtained 5, 10, 15 and 30 minutes following the subarachnoid injection.

Results. The addition of epinephrine to lidocaine that was injected into the subarachnoid space prolonged the duration of motor block by an average of 43 per cent (range 11 to 63 per cent) (P<0.001) as compared with the duration of motor block produced by the same dose of lidocaine without epinephrine (Fig 1). Nevertheless, the maximum arterial concentration of lidocaine was not altered by epinephrine. For example, the arterial concentration of lidocaine was 0.9 ug/ml when epinephrine was added to the local anesthetic and 0.8 ug/ml in the absence of epinephrine (Fig 2).

Discussion. Axelsson et al observed that the addition of epinephrine to lidocaine that was injected into the lumbar subarachnoid space of patients decreased the mean venous concentration of lidocaine but did not prolong the motor block. In contrast, using dogs as their own controls, we observed that the addition of epinephrine to lidocaine that was injected into the subarachnoid space did not alter the absorption of lidocaine from the subarachnoid space but in an inexplicable way prolonged the duration of motor block.

## Reference:

Axelsson K, Hidman B: Blood concentration of lidocaine after spinal anesthesia using lidocaine and lidocaine with adrenaline Acta Anesth Scand 25: 240-245, 1981.

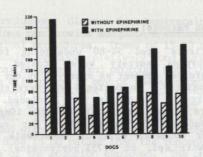


Fig 1 Duration of motor block

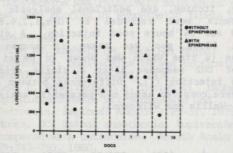


Fig 2 Arterial blood levels at 15 minutes following the subarachnoid injection

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