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A551

TITLE: DIFFERENCES IN THE ATTENUATION OF α1 AND α2 ADRENOCEPTOR-MEDIATED PRESSOR RESPONSES BY HALOTHANE AND ISOFLURANE IN DOGS

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There is controversy as to whether halothane and isoflurane attenuate α1 adrenoceptor-mediated pressor responses in vivo and in vitro (1,2). In the present study, we investigated the effects of both anesthetics on α adrenoceptor-mediated pressor responses induced by phenylephrine (P), a pure α1 agonist, and clonidine (C), an α2 dominant agonist.

METHOD: Twenty-four mongrel dogs were anesthetized with thiamylal, urethane and α-chloralose, iv. The left carotid artery was cannulated to measure mean arterial pressure (mBP). After left thoracotomy, a catheter was introduced into the right atrium to measure right atrial pressure (RAP). An electromagnetic flow probe was placed on the ascending aorta to monitor ascending aortic blood flow (AoF). After autonomic nervous system blockade with propranolol, atropine and hexamethonium, dose response curves to P (n=12) and C (n=12), 0.3, 1, 3, 5 μg/kg, were obtained both before (-Halothane, -Isoflurane) and during (+Halothen, +Isoflurane) the administration of 0.5 MAC endtidal halothane or isoflurane.

RESULTS: P and C increased mBP and systemic vascular resistance [SVR: (mBP-RAP)/AoF] in a dose dependent manner before the administration of the inhalation anesthetics. P and C also increased mBP during the administration of halothane or isoflurane, but the response was significantly reduced by the anesthetics. Both anesthetics reduced the maximal increase in SVR (expressed as % increase in SVR) by C, whereas they did not reduce the P-induced increase in SVR (Fig).

DISCUSSION: Halothane and isoflurane reduced the C-induced increase in mBP and SVR, as well as the P-induced increase in mBP but not the P-induced increase in SVR. Our results correspond very well with previous work where both anesthetics reduced pressor responses of α1 and α2 agonists in vivo under autonomic nervous system blockade (2), whereas halothane did not attenuate α1-mediated vasoconstriction in vitro (1). The attenuation of α1 adrenoceptor-mediated pressor responses by halothane and isoflurane should not result from the suppression of an increase in SVR.

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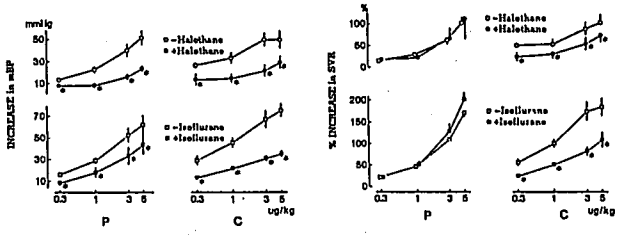

Fig. Effects of halothane and isoflurane on the increase in mBP (left) and % increase in SVR (right). Data are means ± SE. *P<0.05 compared with the corresponding dose.

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TITLE: RENAL BLOOD BLOW AND FUNCTION DURING CONTROLLED HYPOTENSION WITH ADENOSINE OR SODIUM NITROPRUSSIDE IN DOGS UNDER HALOTHANE ANESTHESIA

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Adenosine (ADO) is used experimentally to induce controlled hypotension during anesthesia and surgery.[1] Hypotension is rapidly achieved and easily controlled with adenosine which lacks toxic metabolic side effects. However, adenosine has been reported to cause at least transient decreases in renal blood flow (RBF) and sustained reduction of glomerular filtration rate (GFR).[2,3] Very little information exists comparing the effects of adenosine with sodium nitroprusside (SNP) on renal function during anesthesia.[4] The present study was therefore designed to evaluate and compare adenosine's and SNP's effect on global and region RBF, renal function and renin release during graded controlled hypotension.

Material and Methods: Eight female mongrel dogs weighing 15-23 kg were studied. Under halothane anesthesia an electromagnetic flow probe was positioned around the left renal artery. Cortical renal blood flow was measured with a laser doppler flow probe positioned under the renal capsule. Catheters were inserted into a femoral artery, the left renal vein and the urinary bladder. The animals were allowed to rest for 60 min after completion of surgery. Anesthesia was maintained at 1 MAC with halothane in 0_2 as assessed by mass spectrometry. RBF (global and regional), GFR (inulin-clearance), sodium, osmolar and free water clearances were measured for two 20 min control periods. Mid-period arterial blood samples for ABG's, inulin, sodium and osmolarity and arterial and renal vein samples for renin analysis (RIA) were obtained. Hypotension to MAP levels of 70 and 50 mmHg was then induced and maintained by titrated infusion of adenosine or SNP in a random fashion. Renal function as above was evaluated during steady state hypotension and finally during a post-hypotensive control period. Statistical analysis was performed with repeated-measures analysis of variance. $p < 0.05$ was considered as significant.

Results: Hypotension was easily achieved and controlled with adenosine. It was difficult to achieve a MAP of 50 mmHg with SNP and there was marked tachyphylaxis necessitating frequent adjustments of the infusion rate.

	Control	ADO-70	ADO-50	SNP-70	SNP-50	Post
MAP	90	70	51	71	57	99
RBF	165	158	136	161	169	164
Cortical RBF	100	112	73a	117	118	91
GFR	85	42a	23b	79	63a	89
Urine flow	1.04	0.38b	0.19c	0.82	0.52a	1.55a
Renin (art)	3.72	5.70	4.53	8.67a	10.03b	4.64
Renin (vein)	5.52	11.60a	9.03a	16.72b	19.54b	5.21

a: p <0.05 b: p <0.01 c: p <0.001 from control

Conclusion: Global RBF was maintained with both agents during hypotension under halothane anesthesia. Adenosine caused a reduction in cortical RBF. Despite the maintained blood flow, adenosine caused marked reductions in GFR and urine flow rate. These data suggest that adenosine, if used, should be limited to short periods of hypotension.

References
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