TITLE: IMMEDIATE VENTILATORY SUPPORT AFTER BUPIVACAINE-INDUCED APNEA PREVENTS CV COLLAPSE IN ANESTHETIZED RATS.

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Introduction: Apnea (A) is one of the major signs of Bupivacaine (B) toxicity that often if not always preceeds CV collapse. We investigated in anesthetized rats if ventilatory support (Vent) provided immediately after A altered subsequent hemodynamics and outcome.

METHODS: Sixteen Sprague-Dawley rats, weighing 432 to 520 g, received 50 mg/kg intraperitoneal pentobarbital, had tracheotomy, catheterization of an IJV for fluid/drug administration and retrograde catheterization of the left carotid artery for placement of a 2 mm Millar transducer-catheter in the aorta (Ao) for the measurement of SBP, DBP and peak Ao dP/dt. Heart rate (HR) was obtained from the ECG. All rats received 5-min spaced cumulative IV doses of 1 (B_1), 2 (B_2), and 4 (B_4) mg/kg of B and were randomly assigned to receive Vent (Harvard Rodent ventilator, 90 rpm) about 5 sec following A (Group V, n=8) or not to receive Vent (Group S, n=8). High speed recordings (Gould) were made before B_1 (Co), at A and 4 min following the B dose that had caused A ($B^{A,4}$). Data analysis included ANOVA, unpaired t test and Fisher's Exact Test (FET).

RESULTS: Mean values and SEM are listed in the table. A occurred after B_2 in 2/8 rats in both groups and at B_4 in 5/8 rats of group S and 6/8 rats of group V. After A, HR, SBP and DBP were lower than at Co in both groups. The rat of group S that did not have A did not have hypotension and survived. All other rats (7/8) of group S died. All rats in group V survived (P<0.01 by FET). At $B^{A,4}$, their HR was still lower than at Co but their SBP, DBP and peak Ao dP/dt were no longer statistically different from Co.

DISCUSSION: CV collapse appears to be intimately dependent on B-induced A. Preliminary arterial blood gas data obtained in the post-A period indicate that acid-base abnormalities may be strong determinants of subsequent CV collapse.

		Co	A	$B^{A,4}$
HR (bpm)	S	442±7.4	226±32.2^{C}	75±31.1^{C,A}
	V	425±17	220±30^{C}	319±25^{C,A,S}
SBP (mm Hg)	S	151±7.8	73± 9.8^{C}	16.2±12.1^{C,A}
	V	141±11.3	101±10.2^{C}	130±11^{A,S}
DBP (mm Hg)	S	120±5.9	38±8.2^{C}	12±10.4^{C,A}
	V	111±10.3	70.4±11.4^{C}	9.7±11^{A,S}
peak A_o dP/dt (mmHg/sec)	S	1250±124	1250±184	225±149^{C,A}
	V	1550± 82	1437± 94	1362±103^{S}

C: p < 0.01 compared to Co, a and A, p < 0.05 and p < 0.01 compared to apnea by Newman-Keuls test.

S: p < 0.01 compared to group S by unpaired t-tests.

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TITLE: SPINAL ANESTHESIA AND MIDAZOLAM EXERT ANTAGONISTIC EFFECTS ON CO2 RESPONSE

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Spinal anesthesia (Sa) has been associated with cardiac arrest in healthy patients.[1] Deafferentation of the chest wall by Sa[2] combined with sedative medication may interact to alter the ventilatory response to CO2.

With institution approval and informed consent, 19 ASA I subjects (8F, 12M) aged 19-38 completed CO2 rebreathing experiments (Read's technique[3]) under 4 conditions: Baseline (B), Midazolam (M), Spinal Anesthesia (SA) and Spinal Anesthesia plus Midazolam (SA+M). Ventilation was also monitored noninvasively during rest using respiratory inductance plethysmography (RIP)..

Ten subjects received 0.075 mg/kg and 9, 0.050mg/kg midazolam in M and SA+M. Mean anesthesia level in SA and SA+M was T5 (T3-T8). Response to CO2 decreased in both M and SA (p<.05). The combination (SA+M) resulted in an antagonistic effect and a smaller decrease than either individually. The relationship between minute ventilation and ETCO2 was more variable after sedation with midazolam and was most variable in SA+M.The use of midazolam in either M or SA+M led to a decrease in tidal volume and increase in respiratory rate (p<.02). As a result, minute ventilation was reduced in SA+M (p<.05). SA reulted in an increase in VT, decrease in rate, but a subsequent increase in MV. Mean inspiratory flow rate, an index of respiratory drive, was decreased in M and SA+M but increased in SA (p<.02).

	CONDITION			
	B	M	SA	SA+M
SLOPE(L/M/mmCO2)	2.76	2.36*	2.37*	2.53
R SQUARED SLOPE	0.79	0.65**	0.74	0.60**
VT (L)	0.56	0.40**	0.74**	0.40**
RATE	14.8	17.0**	12.7**	15.1**
MV (L/M)	6.93	6.36	7.81*	5.63*
MIFR (L/S)	0.32	0.25**	0.40**	0.24**
RIB%	47.9	61.4**	43.1	65.8**

* p < 0.05
** p < 0.02

There was a significant interaction between midazolam and Sa on CO2 ventilatory response. However, when combined in SA+M there was an antagonistic rather than synergistic effect on CO2 response.

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