Title: MECHANICAL VENTILATION AND CARDIAC CONDUCTION

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Introduction. Hyperventilation with significant respiratory alkalosis and associated hypokalemia is a known cause of cardiac arrhythmias in anesthetized patients.[1] The effect of respiratory alkalosis on specialized cardiac conduction during anesthesia has not been determined. One report indicates that extremes of hyperventilation (P_aCO_2 < 10 torr associated with increases in CVP to 50 torr) prolonged total A-V conduction time, but ventilation to a P_aCO_2 of 14 torr (where CVP just began to increase) decreased A-V conduction time.[2] We have investigated the effect of hypocapnea on A-V conduction and refractory periods in dogs anesthetized with pentobarbital (PENT) or pentobarbital-halothane (PENT/HALO) as part of our ongoing effort to determine the effects of anesthetics on cardiac conduction.

Methods. Healthy mongrel dogs (N=17) were anesthetized with iv PENT (30 mg/kg body wt., loading dose) followed by a continuous infusion (2.3 mg/kg/hour) for the duration of the experiment (< 4 hours). Dogs were mechanically ventilated with O_2 to end-tidal CO_2 levels of 40 (normocapnea) or 25 (hypocapnea) torr. Normothermia (37.0-38.0°C, rectal) was maintained and decamethonium (0.25-0.5 mg/kg) used as necessary to prevent spontaneous movements. Measurements of arterial pH, pO_2, pCO_2, and serum K+ were made at each level of CO_2. Following testing with PENT alone, dogs were equilibrated with HALO (1.0 per cent end-tidal) during the infusion of PENT and testing at the 2 levels of CO_2 repeated. His bundle electrocardiography and high right atrial pacing or atrial extrastimulation were used for conduction and refractory period measurements.[3]

Results. There was no significant effect of hypocapnea (CO_{2ET} 25 vs. 40 torr) on spontaneous heart rate, mean arterial pressure (femoral) and His-Purkinje or ventricular conduction. Significant ($p < 0.05$) findings included: 1) AV nodal conduction time (AVN) prolonged; 2) an increase in the atrial paced cycle length that produced Wenkebach block (WB); 3) AV nodal minimum conduction time (MCT[3]) prolonged; 4) AV nodal functional refractory period (FRP) prolonged; and, 5) the atrial effective refractory period (ERP) shortened. Mean values (± SEM) for these parameters are given in milliseconds in the table (following discussion) along with the values for arterial pH and serum K+ (meq/l).

Discussion. Mechanical hyperventilation impaired AV nodal function and shortened the atrial effective refractory period in PENT and PENT/HALO anesthetized dogs. These effects may contribute to arrhythmias caused by hyperventilation. We consider it unlikely that depression of nodal function noted with moderate hypocapnea would be of physiologic significance in most patients (sufficient to cause advanced Type II 2nd or 3rd degree heart block). However, it is possible that the impairment of conduction would have been greater in this study had we corrected the mild metabolic acidosis noted during normocapnea thereby providing more marked degrees of alkalosis during hyperventilation.

Deliberate or inadvertent hyperventilation of patients during anesthesia is not uncommon. Indeed, common arrhythmias during anesthesia such as wandering atrial pacemaker, A-V dissociation and low junctional rhythms may be caused in part by the adverse effects of hyperventilation. We conclude that in addition to the well recognized arrhythmic potential of hypoventilation, anesthetists must be aware of potentially deleterious effects of moderate hyperventilation on cardiac conduction.

(TEST)	(CO_2-40)	(CO_2-25)
	PENT	
AVN	51 ± 3	58 ± 4
WB	190 ± 5	198 ± 7
MCT	48 ± 3	53 ± 3
FRP	214 ± 5	223 ± 6
pH	7.32 ± .01	7.46 ± .01
K+	3.9 ± 0.1	3.6 ± 0.1
	PENT/HALO	
AVN	61 ± 4	68 ± 5
WB	229 ± 8	251 ± 12
MCT	60 ± 6	67 ± 5
FRP	258 ± 10	269 ± 9
ERP	116 ± 6	108 ± 6
pH	7.30 ± .01	7.44 ± .01
K+	3.9 ± 0.1	3.6 ± 0.1

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References.

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