Title : VENTRICULAR CONDUCTION DURING HALOTHANE ANESTHESIA

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Introduction. Changes in ventricular cardiac conduction associated with anesthesia in animal models is of interest and may be relevant to the possible mechanisms underlying arrhythmias during anesthesia. In Vivo Halothane (H) prolongs conduction times from the His Bundle (HB) to onset of QRS activity in a dose dependant manner.[1] In vitro H depresses conduction in Purkinje fibers and shortens their action potentials and refractory periods.[2] The present canine study was designed to examine changes in ventricular cardiac conduction and refractory periods of the Ventricular Conduction System (VCS) during H anesthesia.

Methods. HB recording and extrastimulus techniques were utilized in open chest animals to measure conduction intervals and refractory periods defined as follows. 1) Epicardial Activation Times (EAT)- the interval between onset of the HB deflection or HB pacing artifact and onset of its epicardial deflection at each of 3 bipolar ventricular recording sites (RS). 2) Functional Refractory Periods (FRP) of the VCS pathway to each RS- the minimum interval between epicardial deflections or responses at each RS to the last of a series of regular HB pacing stimuli and a following HB extrastimulus of any degree of prematurity. 3) Effective Refractory Period (ERP) of the VCS-the coupling interval of the earliest HB extrastimulus that propagated to the ventricles. 4) Conduction Delay (CD) to each RS of the earliest propagating HB extrastimulus- the derived interval measured as the difference in EAT between the earliest propagating extrastimulus and the preceeding regular drive stimulus. EAT was measured both during regular supraventricularly conducted and HB paced rhythms. The FRP, ERP, and CD were determined at HB pacing rates of 125, 150, and 175 bpm. Pacing and recording was performed by suturing a quadripolar plaque electrode over the HB during short periods of venous inflow occlusion.

Two groups of animals were studied. H in oxygen anesthesia was delivered from a Draeger Vaporizer into a circle system with Airsheilds ventilator at fixed settings and flow rates. In group I (8 animals) comparisons were made between measured parameters at 1.5 and 2.4% end-tidal H levels (range 1.4-1.6% and 2.3-2.5%, respectively). In Group II (6 animals) comparisons were made between Basal Thiopental Anesthesia (BTA) (total dose 116 mg/kg over 8-10 hr) and BTA with added H (1.5%, identical settings and flows). Within Group comparisons were made by an analysis of variance to assess the influence of variation in HB pacing rate, anesthetic treatment, and location of each RS. A probability of less than .05 was considered significant except where indicated, and the average of paired changes for anesthetic treatment is reported.

Results. EAT was significantly increased by change to the higher H level in Group I by an avg. of 2.4 msec during supraventricularly conducted and 2.9 msec during HB pacing. EAT increased but not significantly during addition of H to BTA in Group II. Parameters measured by the extrastimulus technique significantly decreased at increasing HB pacing rate in both Groups but were not dependant on the location of the RS. In Group I, change to the higher H level was associated with significant decrease in FRP (avg. -7.3 msec), nonsignificant decrease in the ERP of the VCS (avg. -2.7 msec), and decrease ($P<.07$) in CD if the earliest propagating extrastimulus (avg. -6.1 msec). In Group II the addition of H to BTA was associated with nonsignificant decrease in FRP of the VCS (avg. -1.4 msec), significant decrease in the ERP of the VSC (avg. -9.1 msec) and significant increase in CD of the earliest propagating extrastimulus (avg. +9.6 msec).

Conclusions. The results in Group I suggest that in vivo there is dose related depression of ventricular conduction and shortened FRP in the VCS associated with H anesthesia in this canine model. This finding is consistent with the actions of H in vitro on action potentials of Purkinje Fibers. The results in Group II suggest that different qualitative and quantitative changes may occur in ventricular conduction and the refractory periods of the VCS on addition of H to BTA. In theory, re-entrant ventricular arrhythmias might be expected to be facilitated by some of the observed changes in ventricular conduction and refractoriness in the VCS associated with Halothane anesthesia in this canine model. (Supported by Grant HL 16511 and the Med. Research Service of the VA).

References.

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