POSTER SESSION XI

A573

TITLE: REPERFUSION-INDUCED DIASTOLIC DYSFUNCTION CORRELATES WITH EXTENT OF RIGHT VENTRICULAR INFARCTION

AUTHORS: WE Johnston MD, J Vinten-Johansen PhD, CF Toombs BS, R Anderson PhD, E Tommasi BA

AFFILIATION: Depts Anesthesia, Cardiothoracic Surgery, Physiology/Pharmacology, and Public Health Sciences, Wake Forest Univ Med Ctr, Winston-Salem, NC 27157-1009

Introduction: The acute effects of myocardial ischemia and reperfusion on diastolic stiffness of the right ventricular (RV) free wall have not been quantitated. We postulate that the alteration in diastolic stiffness parallels the development of RV necrosis.

Methods: Seventeen anesthetized, closed-chest dogs underwent right coronary occlusion (90 min) followed by reperfusion (120 min). RV end-diastolic pressure (Ped) was measured by a micromanometer-tipped catheter. Through small pericardial slits, 2 pairs of sonomicrometer crystals were implanted in the RV free wall in horizontal and vertical directions. RV free wall area at end diastole (Aed) was calculated on-line by multiplying horizontal and vertical segment lengths. RV free wall stiffness was determined by curve fitting a range of Ped and Aed points generated during caval occlusion to the relation $Ped = \alpha(e^{\beta Aed})$, where α is the Ped intercept and ß is the modulus of free wall stiffness. We used the Aed at Ped = 4 mmHg to describe the position of the diastolic curve. Measurements were taken at baseline, 5 min ischemia, and 5 and 105 min reperfusion. The area of necrosis (An) in the area at risk from ischemia (Ar) was determined by differential stains.

Results: RV ischemia acutely decreased ß from 0.040±.008 to 0.022±.003 ($p<0.05$), with a rightward shift, indicating diastolic "creep." Five min reperfusion shifted the curve position back to the left as ß increased to 0.078±.021 ($p<0.05$), indicating increased stiffness with the onset of reperfusion. After 105 min reperfusion, ß remained elevated (0.086±.20). The change in ß (from baseline) correlated closely with An/Ar ($r = -0.77$; $p<0.05$; Figure 1) in that larger infarcts were stiffer.

Conclusion: These results clearly show significant time-related variability in diastolic stiffness of the ischemic-reperfused RV free wall, making the determination of optimal RV preload difficult. The increase in diastolic stiffness correlated significantly with the extent of necrosis and occurred during reperfusion and not ischemia.

Supported in part by NIH R29-40395.

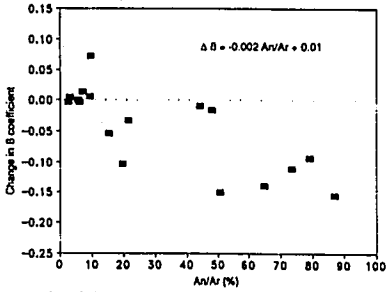


Figure 1. Significant correlation between change in ß coefficient (baseline - 105 min reperfusion) and An/Ar.

A574

EFFECT OF HALOTHANE ON DEFIBRILLATION THRESHOLD

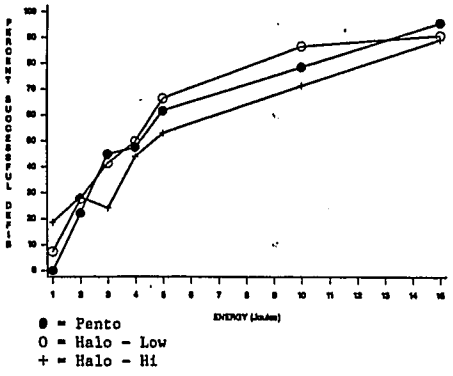
JS Guo BS, DJ Jones PhD, J Gelineau BS, D Solomon MD, CB Hantler MD, Dept Anes, Univ TX Hlth Sci Ctr, San Antonio, TX 78284

Introduction: Since 1985 the Automatic Interval Cardioverter-Defibrillator (AICD) has gained wide acceptance for treatment of intractable cardiac dysrhythmias. Defibrillation thresholds (dft's) determined intra-operatively can predict the success rate of post-operative defibrillation. Anesthetic agents may alter the dft compared to the unanesthetized state.[1] The present study is the first report of the effects of halothane on dft.

Methods: After approval by our Institutional Animal Care Committee, 19 mongrel dogs were randomly divided into two groups. Group 1 animals were anesthetized with pentobarbital 30mg/kg IV and 2mg/kg/hour. Group 2 animals were induced and maintained with halothane at expired concentrations of 1.3 and 1.8 MAC. Through a left thoracotomy a 10 x 10 cm. defibrillating patch electrode was placed on the free wall of the left ventricle (LV). A second 10 x 10 cm. patch was sewn to the right ventricle. A pacing frequency of 99 Hz at 20mamp was initiated and continued for 5 sec. If ventricular fibrillation did not occur, burst pacing was reinstituted. After onset of fibrillation dft was determined as follows: randomly determined energy levels (1,2,3,4,5,10,15,20 joules) were delivered (cathodal through the large patch) and success or failure of defibrillation was recorded. This sequence was repeated at least 6 times to determine the success rate at each energy level.[2] Comparison between pentobarbital and halothane anesthetized animals at all energy levels were compared using 2-way repeated measures ANOVA.

Results: There were no differences in success rates at any energy level between pentobarbital- and halothane- anesthetized animals (fig.1). All animals were successfully defibrillated at 20 joules, thus all animals demonstrated at least a 10 joule margin of safety. There was no difference between groups in number of episodes of burst pacing required to induce VF ($p > .9$, Mann-Whitney).

Discussion: Halothane anesthetized animals have dft curves similar to those receiving pentobarbital anesthesia. Compared to the awake state the effects of halothane or pentobarbital on dft's are unknown.



References:
1. J Cardiothorac Anes, pp 236-44.
2. Am Heart J, pp 77-84.