TITLE: TACHYCARDIA INCREASES THE EXTENT OF RIGHT VENTRICULAR NECROSIS FOLLOWING RIGHT CORONARY ARTERY OCCLUSION AND REPERFUSION

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Increasing heart rate has been recommended as a therapeutic maneuver to improve cardiac output in patients with hemodynamic instability resulting from right ventricular (RV) infarction (1). However, it is not known whether tachycardia increases the extent of RV infarction. We tested the hypothesis that the extent of RV necrosis is less in dogs with heart rates of 80 bpm (n=6, Group 1) than 150 bpm (n=6, Group 2).

**METHODS:** Twelve anesthetized, closed-chest dogs were paced during right coronary occlusion (90 min) and subsequent reperfusion (120 min). Blood flow (radioactive microspheres) was determined in the RV area at risk (unisperse blue dye) and area of necrosis (triphenyltetrazolium chloride). Data were analyzed by multivariate analysis of variance.

**RESULTS:** Hemodynamic parameters including mean aortic pressure, pulmonary artery pressure, and RV end-diastolic pressure were similar in both groups. The size of the RV free wall (33.6±2.6 g, Group 1 vs. 31.5±1.9 g, Group 2) as well as the area at risk from ischemia (63.0±3.5% of RV, Group 1 vs 63.4±3.2% of RV, Group 2) were similar between groups. However, the extent of necrosis expressed as a percent of the area at risk (An/Ar) was 17.1±6.4% in Group 1 (range 3.0 - 24.7%) in contrast to 50.3±9.7% in Group 2 (range 19.8 - 73.3%) ($p<0.05$). Blood flow in the area of necrosis was similarly reduced during ischemia in Group 1 (3.1±1.0 ml/min/100g) and Group 2 (4.3±0.8 ml/min/100g; p=NS). As seen in Fig. 1, an inverse relationship was found between the area of necrosis and collateral blood flow in the RV free wall where An/Ar = 187.8 exp [0.085 collateral flow] ($r = 0.78$; $p<0.05$).

**DISCUSSION:** We found that tachycardia increases the extent of RV free wall necrosis in the ischemic RV. The animals with faster heart rates tended to be located on the steeper portion of the An/Ar-collateral flow relationship. Tachycardia may reduce salvage of the ischemic myocardium in patients with RV ischemia and should be avoided.

**REFERENCES:**

1. Roberts R et al: Ann Rev Med 34:377-1983.

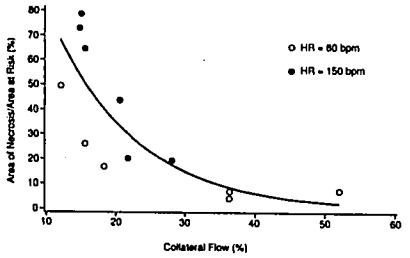


**Figure 1. An/Ar - collateral flow relationship.**

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TITLE: EFFECTS OF HEMATOCRIT ON O2 TISSUE EXTRACTION CAPABILITIES

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INTRODUCTION. The effects of hematocrit (Ht) on tissue O2 extraction capabilities were studied using a dog model of progressive hemorrhage (1).

METHODS. The study included 24 mongrel dogs (28.3 ± 3.3 kg) anesthetized with thiopental (20 mg/kg) and isoflurane 0.5 MAC (0.6% end-tidal). The dog was mechanically ventilated (FiO2: 0.21). O2 consumption (VO2) was calculated from measured FeO2 (paramagnetic method), FeCO2 (infrared method) and minute ventilation. O2 transport (DO2) was calculated from thermodilution cardiac output, hemoglobin and SaO2 determination. Thirty min after splenectomy, the dogs were randomized in 3 groups of 8, with a Ht of 40%, 30% or 20%, obtained following normovolemic hemodilution using HES 450/0.5. DO2 was reduced by progressive hemorrhage (100 ml aliquots) while Ht was maintained constant by retransfusion of packed red-cells. At each stage DO2, VO2 and lactate were measured after a 10 min equilibrium. In each animal critical DO2 (DO2crit) was determined from a plot of VO2 vs DO2, and a plot of serum lactate vs DO2, using the point of intersection of the 2 best-fit regression lines (1). Critical extraction ratio (ERcrit) was the ratio of VO2crit over DO2crit.

RESULTS. (Mean ± SD)

| | Ht 40% | Ht 30% | Ht 20% |
|---|---|---|---|
| DO2 crit (ml/min/kg) | 10.7±0.9 | 7.2±1.3** | 7.7±1.1** |
| VO2 crit (ml/min/kg) | 5.7±0.6 | 5.3±0.7 | 5.6±0.8 |
| ER crit (%) | 54.0±8.0 | 75.1±13.5** | 70.7±10.2** |
| DO2 lactate (ml/min/kg) | 10.2±1.2 | 7.9±1.1** | 8.3±0.8** |

** $p<0.01$ vs Ht 40%

DISCUSSION. Tissue O2 extraction capabilities were significantly higher at an Hct of 30% and 20% than at 40%. This could be related to an improved distribution of microvascular blood flow (2).

(1) J Appl Physiol 64(5):2074-2082, 1988
(2) Int J Microcirc Clin Exp 6:225-235, 1987