A517

Title: **Critical level of hemodilution in myocardium with compromised coronary blood flow: A study on regional function and retransfusion**

Authors: DR Spahn M.D., RL Smith Ph.D., AJ Menius Ph.D RL McRae, CD Veronee MD, BJ Leone M.D.
Affiliation: Department of Anesthesiology, Duke University, Durham, NC, 27713

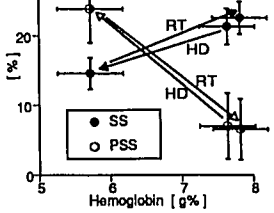
Introduction: Acute normovolemic hemodilution (HD) to a hematocrit (HCT) of 25% is well tolerated in areas of the left ventricular myocardium supplied by a critically constricted coronary artery, at a HCT of 15%; however, severe regional dysfunction occurs [1]. The aims of the present study were to identify the lowest level of hemoglobin (Hb) *not* associated with systolic dysfunction and to investigate the effects of retransfusion (RT) following HD induced regional dysfunction.

Methods: Anesthesia was introduced in 19 dogs with thiopental and maintained with halothane in an air-oxygen mixture to achieve an arterial Hb saturation of 95-98%. The animals were instrumented so as to determine central aortic and LV pressure, aortic root and left anterior descending coronary artery (LAD) flow as well as regional myocardial function (sonomicrometry). Regional myocardial function (systolic shortening, SS, post-systolic shortening, PSS) was assessed in the LAD territory, in the basal part of the anterior LV wall supplied by the left circumflex coronary artery (LCA), and in the mid LV posterior wall (LCP). A critical coronary constriction (CC) was imposed on the LAD (blunting the physiological hyperemic response to a 10 s total LAD occlusion) and HD was performed by simultaneously exchanging blood with Dextran 70 to achieve LVEDPs of 5-8 mmHg. The animals were hemodiluted to target Hbs of 9.0, 7.5, 6.0 and 4.5 g% until systolic dysfunction was induced in the LAD territory, i.e. SS decreasing below 85% of SSLAD at CC. In 9 animals retransfusion (RT) was then started and measurements were made in steps of 1.5 g% until SSLAD was restored (>95% of SSLAD at CC). Analysis of variance and Least Square Means tests with $p<0.05$ were used to compare data at CC, at the highest level of Hb *not* yet associated with regional dysfunction (HD1) and after induction of regional dysfunction (HD2).

Results: The lowest median level of Hb *not* associated with systolic dysfunction was 6.1 g% with lower and higher quartiles of 4.6 and 8.9 g%. Heart rate, mean arterial and coronary perfusion pressures as well as LVEDP were unchanged during HD, cardiac output (CO) and LAD flow increased. The decrease of SSLAD at HD2 was partially offset by increases in SS in nonischemic areas.

	CC	HD1	HD2
Hb [g%]	12.2±0.3	6.6±0.4★	6.2±0.5★
SSLAD [%]	23.4±1.6	23.2±1.9	16.0±1.9★✚
PSSLAD [%]	3.9±2.2	6.8±3.0	20.0±4.6★✚
SSLCA [%]	16.0±2.0	16.4±2.5	19.7±3.0
PSSLCA [%]	3.5±2.6	3.7±3.0	0.6±0.3
SSLCP [%]	13.0±1.0	14.9±0.9	16.2±1.4★
PSSLCP [%]	5.3±2.6	1.3±1.1	2.2±2.0
CO [l/min]	2.4±0.3	3.3±0.4★	3.4±0.5★
LAD flow [ml/s]	0.43±0.05	0.66±0.07★	0.52±0.06★✚

★ = sign. different from CC, ✚ = sign. different from HD1



Increasing Hb by only 2.1±0.3 g% fully restored LAD function in every dog; SSLAD recovered and the HD induced PSSLAD disappeared during RT (fig.).

Discussion: Increasing transstenotic LAD flows during HD may explain in part why relatively low levels of Hb are well tolerated in compromised myocardium. The fact that RT fully restored regional function indicates that the HD induced dysfunction was purely functional and no structural damage or stunning occurred.

References: [1] Leone et al.,Anesthesiology 73:A596, 1990

Supported in part by the Swiss National Science Foundation

A518

TITLE: EFFECT OF VOLUME LOADING ON THE ISCHEMIC HEART

AUTHORS: André Coetzee PhD, P. Fourie PhD
AFFILIATION: Department of Anesthesiology and Physiology University of Stellenbosch Medical School Tygerberg 7505, South Africa

This study examined the effect of acute volume loading on regional myocardial function in the presence of a critical stenosis on a coronary artery.

7 Pigs were anesthetized with thiopentone, a tracheotomy was performed and the animals ventilated with oxygen and nitrogen. Anesthesia was maintained with 0.5% halothane. Aortic, pulmonary and a femoral artery cannula were inserted. A thoracotomy and pericardectomy were performed and left ventricular pressure was directly measured. Two pairs of ultrasonic crystals were inserted in the subendocardium: One pair in the region supplied by the LAD coronary artery and the other pair in the region supplied by the LX coronary artery. Regional length changes were combined with LV pressure changes on an oscilloscope to obtain pressure-length loops for both regions. A micrometer controlled snare was applied to the LAD artery and a small cannula was inserted in a vein draining the LAD area. After control measurements (A), a critical stenosis was applied to the LAD artery (B). Thereafter Haemocel was infused until such time that the myocardial lactate dynamics in the LAD region changed significantly (C). Results were analyzed with ANOVA (Tukey) and the multiple range test.

Application of the constriction did not change global LV function. The venous hemoglobin saturation from the LAD area decreased from 31.36±3.76 to 20.43±0.97% ($p<0.05$) and lactate extraction changed from 15.88±2.38 to -9.56±5.32% ($p<0.05$). Volume loading increased the LVEDP from 9.71±1.77 to 15.93±2.07 mmHg ($p<0.05$) and improved the stroke volume from 22.90±0.80 to 26.10±1.00 ($p<0.05$).

Lactate production increased from 9.56±5.32 to 67.63±19.19% ($p<0.05$). The venous oxygen tension in the LAD area decreased from 38.74±9.37 to 17.34±1.34 mmHg ($p<0.05$) and the venous pH decreased from 7.34±0.02 to 7.28±0.01 ($p<0.05$).

Volume loading improved global left ventricular function according to the Starling principle. However, data indicate that regional myocardial ischemia occurred during volume loading in the area supplied by a coronary artery which is critically constricted. The lactate production did not correlate with the coronary perfusion pressure. As regional stroke work remained constant, data suggests that volume loading decreases oxygen supply resulting in regional myocardial ischemia.