TITLE: RELATIONSHIP BETWEEN TISSUE OXYGENATION AND PLASMA ADENOSINE [ADO] DURING SEPSIS

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INTRODUCTION: UNDER PHYSIOLOGIC CONDITIONS TOTAL BODY OXYGEN CONSUMPTION [VO2] IS KEPT CONSTANT OVER A WIDE RANGE OF OXYGEN DELIVERY [DO2] BECAUSE OF VARIATIONS IN OXYGEN EXTRACTION. HOWEVER, IN SEPTIC PATIENTS OXYGEN EXTRACTION IS SIGNIFICANTLY DISTURBED AND TISSUE OXYGENATION IS CRITICALLY DEPENDENT ON DO2.
BECAUSE ADO IS A SENSITIVE INDICATOR OF ADEQUATE TISSUE OXYGENATION, THIS STUDY INVESTIGATES THE INFLUENCE OF INCREASED DO2 FOLLOWING BLOOD TRANSFUSION [BT] ON PLASMA ADO, HYPOXANTHINE [HYPO] AND LACTATE [LAC] IN PATIENTS WITH SEVERE SEPSIS.

METHODS: IN 12 PATIENTS (AGE 46 +/- 20) WITH HCT < 30% MEAN ARTERIAL BLOOD PRESSURE [MAP], HEART RATE [HR], CARDIAC INDEX [CI], AND PULMONARY CAPILLARY WEDGE PRESSURE [PCWP] WERE DETERMINED (WITH INFORMED CONSENT AND AFTER INSTITUTIONAL APPROVAL) BEFORE [C], 10 AND 60 MIN, AND 24 HOURS AFTER BT. ALSO DO2 AND VO2 (CALORIMETR.) WERE DETERMINED AND PLASMA ADO, HYPO, PH, AND LAC WERE ANALYZED.

RESULTS:
TABLE 1: (MEAN VALUES; N=12; * P < 0.05)

		TIME AFTER BT		
	C	10'	60'	24 H
MAP (MM HG)	77	99*	93*	81
HR (1/MIN)	92	88	90	88
CI (L/MIN/M2)	4.7	4.9	5.0	5.1
PCWP (MM HG)	10	14*	11	12
HCT (%)	27	39*	38*	37*
DO2 (ML/MIN/KG)	13	18*	19*	19*
VO2 (ML/MIN/KG)	3.3	3.5	3.6*	3.5
ADO (NMOL/L)	508	299*	262*	236*
HYPO (NMOL/L)	706	424*	461*	536
LAC (MMOL/L)	1.1	.90	.82*	.98
PH	7.42	7.40	7.41	7.40

CONCLUSIONS: 1.) BT ELEVATES MAP, PCWP, AND DO2. 2.) THE ENHANCED VO2 (VO2 = 2.3 + 0.1 X DO2, R = 0.54, P < 0.005) IS PARALLELED BY A DECREASE IN ADO, HYPO, AND LAC. 3.) DURING SEPSIS ADO IS 4-5 TIMES HIGHER THAN IN NON-SEPTIC PATIENTS. ADO IS INVERSELY RELATED TO THE RATIO OF [DO2]/[VO2] (ADO = 1203 - 176 X [DO2/VO2], R = 0.98, P < 0.01) AND BEST REFLECTS THE IMPROVEMENT IN TISSUE OXYGENATION.

TITLE: RIGHT HEART FAILURE AND PROSTAGLANDIN E1 (PGE1) AFTER HEART TRANSPLANTATION

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Acute pulmonary hypertension (PH) after heart transplantation (Tx) and increase in right ventricular (RV) afterload with RV failure is often the limiting factor of biventricular function. Because of its metabolism, prostaglandin E1 (PGE1) is known as a relatively selective pulmonary vasodilator with reduction of pulmonary vascular resistance as well as poor systemic hypotensive effects. In fact 80% to 95% of the drug is cleared during a single pass through the lungs so that its systemic effects are minimized. We report our experience with 22 pts receiving PGE1 for intraoperative and postoperative control of PH after Tx.

From January 1989 to February 1990 40 pts underwent Tx for dilated or ischemic cardiomyopathy at the Department of Cardiovascular Surgery of Padova University. With institutional approval and informed consent, PGE1 was used in 22 pts. who showed PH perioperatively. Routine hemodynamic monitoring included indwelling arterial and flow-directed pulmonary artery catheters, and electrocardiogram. Anesthesia consisted of high fentanyl-muscle relaxant-oxygen/air technique. The indications for PGE1 infusion were mean pulmonary arterial pressure (mPAP)>25 mmHg and/or pulmonary vascular resistance (PVR)>240 dyn sec cm^{-5} and/or RV dysfunction, with right arterial pressure (RAP) above pulmonary capillary wedge pressure (PCWP) and cardiac index>2.5 l/min/m^2. Infusion rates ranged from 10 to 50 ng/Kg/min for up to 8 days, with a variable concurrent therapy of isoproterenol, dopamine, epinephrine and norepinephrine.

Three pts. died during postoperative course. After about one week treatment, there was a significative decrease in mPAP (from 31+/-7 to 25+/-4) and in PVR (from 317+/-88 to 215+/-22) with an increase in cardiac output (CO) (from 4.22+/-1.7 to 4.93+/-1.6). There was no significative change in RAP, PCWP, and systemic vascular resistance.

The normal donor right ventricle is often subjected after Tx to the recipient chronic pulmonary hypertension. Acute increase in RV afterload leads to an increase in both end-diastolic volume (RVEDV) and end-diastolic pressure (RVEDP). Because of ventricular interdependance, this event results in a shift of the interventricular septum into the left ventricular cavity, an increased PCWP, and a decreased CO. Moreover, the increase in PVR with increased RV volume results in increased RV wall stress and subsequently RV dysfunction secondary to ischemia.

Our results indicate that PGE1 can significantly and selectively reduce mPAP and improve RV function after Tx by decreasing PVR and RV afterload. The selectivity of PGE1 for pulmonary vasculature, associated with norepinephrine (10 pts.) to maintain a normal mean arterial pressure, is essential to improve coronary perfusion pressure and to prevent RV ischemic dysfunction.

References
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