Title : HEMODYNAMIC CHANGES DUE TO NORMOVOLEMIC ANEMIA IN CORONARY DISEASL PATIENTS

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Introduction. Autologous fresh, whole blood is the ideal solution for the restoration of the fibrinogen, platelets and other labile clotting factors. The use of autologous blood alleviates the inherent dangers of homologous blood, namely viral hepatitis and other infectious diseases, allergic reactions to cellular antigens and proteins, hyperkalemia, mismatched transfusion and febrile reactions related to leukocyte and platelet antigen. Withdrawal of patients' own blood immediately prior to cardiopulmonary bypass and reinfusion of the autologous blood following bypass to save bank blood is becoming a common practice in open heart surgery. During the withdrawal of patients' blood, twice the volume of blood removed is replaced by infusion of Ringer's lactate, and this produces an acute moderate normovolemic anemia. Though the hemodynamic effects of normovolemic anemia have been studied in animals with normal coronary circulation or experimentally induced coronary insufficiency, its effects in patients with coronary artery disease have not been reported. In this investigation, we report the hemodynamic and electrocardiographic (ECG) changes secondary to acute moderate normovolemic anemia in patients with coronary artery disease.

Methods. Forty-eight patients scheduled for coronary artery bypass surgery were included in the study. Patients were divided into two groups depending on their left ventricular and diastolic pressure (LVEDP) measured during cardiac catheterization. Group I (n=21) had a LVEDP of less than 15 torr, and Group 2 (n=27) had a LVEDP of greater than 16 torr. In all other respects including height, weight, body surface area, drug intake and anesthetic management, both groups were comparable. The following parameters were measured or derived: heart rate (HR), mean arterial pressure (MAP), mean pulmonary artery pressure (MPAP), right atrial pressure (RAP), left atrial pressure (LAP), cardiac index (CI), stroke volume index (SVI), systemic vascular resistance index (SVRI), pulmonary vascular resistance index (PVRI), hemoglobin (HG), endocardial viability ratio (EVR), and the product of diastolic pressure time index times arterial oxygen content over systolic pressure time index (DPTI x CaO_2/SPTI). ECG was continuously recorded throughout the period of observation. The parameters were measured prior to withdrawal of blood and after 5 minutes of stabilization following withdrawal of 10 ml/kg of blood and simultaneous infusion of 20 ml/kg Ringer's lactate. Data was analyzed using Student's paired t-test for within group comparisons and Student's t-test for unpaired values for comparison in between the group (Table). P value of <0.05 was considered significant.

Results.

Table

Indices	Group I (n=21) LVEDP < 15 torr		Group 2 (n=27) LVEDP > 16 torr	
	Before	After	Before	After
HR	66	72*	68	83***
MAP	87	86	89	84*
MPAP	14.9	15.0	17	20***
RAP	9.8	10.1	10	12
PCWP	11.1	10.8	13	17**
CI	2.19	2.61**	2.02	1.57***
SI	33.5	37.3	29.8	18.99
SVRI	2901	2451**	3152	3761**
PVRI	139	117	163	162
HG	14.3	9.8**	14.1	10**
EVR	1.08	1.29	1.12	0.78**
DPTI x CaO_2 / SPTI	21.66	19.02	21.42	14.94**
ST CHANGES	-	0	-	11***

P Values: * < 0.05
** < 0.01
*** < 0.005

Discussion. The data indicate that patients in Group I, with LVEDP < 15 torr responded to normovolemic anemia by increasing HR and CI, and decreasing SVRI. But patients in Group 2 with left ventricular dysfunction as evidenced by a LVEDP >16 torr responded differently. Though HR increased, CI decreased significantly. This may be due to further depression of left ventricular function secondary to decreased myocardial oxygen supply as evidenced by a decrease in EVR and DPTI x CaO_2/SPTI. Also myocardial ischemia was evidenced by depressed ST segments in eleven patients (40.7%) in this group. It is concluded from this study that patients with coronary artery disease, and poor left ventricular function as evidenced by an increased LVEDP do not compensate for the acute normovolemic anemia as the patients with normal LVEDP.