OUTCOME OF ABO COMPATIBLE VERSUS IDENTICAL BLOOD TYPE MATCHING IN CARDIAC TRANSPLANT RECIPIENTS ON TRIPLE IMMUNOTHERAPY

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ABO identical (Id) blood type matching in cardiac transplant (Ctx) pts has been reported to yield better survival than ABO compatible (Cp) matching in pts on double immunotherapy (cyclosporine, prednisone). We conducted a retrospective analysis in 82 consecutive pts undergoing Ctx 1/86-2/88 given triple immunotherapy (cyclosporine, prednisone, azathioprine) to assess the effects of ABO blood type matching on survival, cardiac function, rejection, and coronary arteriopathy. There were no differences between Id matched pts (n=69) and Cp matched pts (n=13) in 18 mo survival, and 1 yr cardiac output, echo LVEF, rejection episodes per patient and coronary arteriopathy. In the Id pts, 22% had no rejection compared to 23% of Cp pts.

			One Year			
	N	18 mo surv	Echo LVEF	CO L/min	Rej/pt	Coronary arteriopathy
Cp	13	92%	65±5%	5.2±1.5	0.8	8%
Id	69	85%	63±11%	5.9±1.5	0.8	7%

Conclusion: ABO Cp matched pts on triple immunotherapy appear to have equivalent cardiac function, rejection frequency and 18 month survival when compared to ABO Id pts. Current preference given to ABO Id matched hearts for pts on waiting lists may not be warranted by consideration of outcomes.

Wednesday, March 6, 1991
Poster Displayed: 2:00PM–5:00PM
Author Present: 3:00PM–4:00PM
Hall F, West Concourse
Myocardial Ischemia: Experimental

LATE REPERFUSION LIMITS MYOCARDIAL INFARCT EXPANSION AND ANEURYSM FORMATION WITHOUT SALVAGING MYOCARDIUM

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Reperfusion of infarcted myocardium limits infarct expansion, although the time after infarction that reperfusion produces this benefit and the permanency of this effect are unknown. Rats were randomized into four groups: 1) infarction with reperfusion after 1-2 hours 2) infarction with reperfusion after 6-8 hours 3) infarction without reperfusion 4) sham operation. Surviving rats were killed and their hearts morphologically analyzed at either 7 days, when infarct expansion has plateaued, or 21 days, when infarct healing and aneurysm formation are complete. In rats killed at 7 days, reperfusion after 1-2 hours did not reduce infarct size or transmural necrosis compared to permanent occlusion, but did significantly limit infarct expansion as measured by an index based equally on infarct endocardial segment lengthening and infarct wall thinning (expansion index: no reperfusion 2.73±0.25, n=13 vs. 1-2 hr reperfusion 1.56±0.13, n=23; p<0.001). Late reperfusion after 6-8 hours was also effective in limiting infarct expansion (1.78±0.15, n=16; p=0.002). There was no significant difference in effect on expansion between reperfusion at 1-2 hours and 6-8 hours after infarction (p=0.289). Late reperfusion also limited aneurysmal dilation in those rats killed at 21 days when infarct healing and aneurysm formation are complete (expansion index: no reperfusion 3.45±0.39, n=13; 1-2 hr reperfusion 2.21±0.24, n=15, p=0.01; 6-8 hr reperfusion 2.02±0.20, n=9, p=0.01). This effect was again independent of myocardial salvage; reperfusion did not significantly reduce infarct size or transmural necrosis.

CONCLUSIONS:

1) Late reperfusion reduces acute infarct expansion and later aneurysm formation independent of myocardial salvage.

2) The time after infarction in which reperfusion limits expansion and aneurysm formation is substantially longer than previously established.

NITROGLYCERIN-INDUCED CORONARY DILATION IS IMPAIRED EARLY AFTER CARDIAC TRANSPLANTATION

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Previously we demonstrated tyramine (T)-induced cardiac norepinephrine release ($\Delta[NE]=\Delta[NE]_{coronary\ sinus}-\Delta[NE]_{aorta}$) late after human cardiac transplantation (CT), suggesting sympathetic reinnervation (SR). To determine whether SR affects resting epicardial coronary arterial tone, we studied 53 patients (pts) without prior nitrate therapy: 10 pts <4 months after CT (early), 40 pts >1 year after CT [28 with NE release after T (SR) and 12 without (denervated, D)], and 3 normal patients. Resting coronary tone was assessed by measuring the diameter of a proximal or mid coronary artery (40 RCA, 7 LAD, 6 LCx) at end-diastole using quantitative angiography (Reiber/CAAS, SEE=0.06 mm in our lab) at rest and after maximal vasodilation with nitroglycerin (NTG, 200µg IC).

RESULTS: (mean±SEM)

Patients	n	Δ[NE] (pg/ml)	Coronary Diameter (mm) Rest	NTG	%Δ	HR (%Δ)	MAP (%Δ)
Early	10	24±18	4.3±.3	4.5±.5	7±2	0±0	-9±2*
Late-D	12	46±16	3.7±.2	4.2±.2	15±3	3±2	-5±2*
Late-SR	28	706±79†	3.5±.2	4.0±.1	15±2†	1±1	-7±2*
Normal	3	2212±547†	2.9±.2	3.5±.2	22±7†	1±0	5±6

*p<0.05 vs. Normal; †p<0.05 vs. Early; The response to NTG in the LAD, LCx, and RCA was similar. HR=heart rate; MAP=mean arterial pressure.

These data demonstrate that nitroglycerin-induced epicardial coronary vasodilation is impaired early after CT, subsequently returns to the normal range, and is not influenced by sympathetic reinnervation. These findings suggest that resting epicardial coronary tone is not significantly mediated by sympathetic innervation and that early after CT coronary arteries have reduced tone or a defect in responsiveness to nitroglycerin.

CORONARY VENOUS RETROINFUSION OF DEFEROXAMINE, AN OXYGEN FREE RADICAL SCAVENGER, REDUCES INFARCT SIZE

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The effects of oxygen free radical scavengers on infarct size (IS) are still controversial. One explanation for the contradictory results may be the mode of drug administration. To compare the efficacy of the hydroxyl scavenger deferoxamine (DF) given either by coronary venous retroinfusion (CVR) or systemic intravenously (I.V.), 24 pigs underwent 60 min LAD coronary occlusion followed by 3 hrs reperfusion. Five minutes infusion of DF (10 mg/kg) was given either by CVR (n=8) or systemic I.V. (n=8) beginning 15 min prior to reperfusion. Control pigs (n=8) received saline I.V. Regional myocardial function, risk area (RA) and IS were assessed by sonomicrometry, blue dye and tetrazolium staining, respectively.

IS expressed as percentage of RA was 73.9±13.5% in the control group, 70.6±16.4% in the systemic I.V. treated group and 48.5±21.4% (mean±SD, p<0.05) in the CVR treated group, but there were no differences in hemodynamics or regional myocardial function between the groups.

Conclusion: The oxygen free radical scavenger deferoxamine reduced infarct size significantly when given by coronary venous retroinfusion but not by systemic intravenous administration. The lack of hemodynamic effects suggests a direct cardioprotective effect of the drug. The discrepancy between the positive effect on infarct size reduction and lack of effect on regional myocardial function may be due to "reperfusion stunning" of the myocardium.