

**ARTERIAL DENERVATION AND CYCLOSPORINE THERAPY DIMINISH
ENDOTHELIUM-DEPENDENT RELAXATION**

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Impaired endothelium-dependent vasodilation to acetylcholine has been documented in the coronary arteries of patients following cardiac transplantation. No clinical features, including cardiac rejection, appear to correlate with this impairment. The aim of this study was to determine if denervation and cyclosporine therapy contribute to endothelial dysfunction after transplantation. In organ chambers experiments, isolated aortic rings were studied from controls and from rabbits after a two week period of chemical denervation with 6-hydroxydopamine. Supersensitive vasoconstrictor responses to norepinephrine and methacholine occurred in the denervated arteries. In denervated rings the sensitivity and maximum endothelium-dependent relaxations to methacholine (38 ± 14 vs. $76 \pm 3\%$ controls) and substance P (18 ± 8 vs. $56 \pm 7\%$) were significantly diminished. Relaxations to sodium nitroprusside were not impaired. In rabbits treated for one week with Cyclosporin A (35 mg/kg/sc) there were diminished relaxations to methacholine (54 ± 10 vs. $79 \pm 4\%$) but not to nitroprusside. Diminished relaxations to methacholine ($64 \pm 7\%$) were also observed in vehicle (olive oil) treated rabbits. These studies suggest that arterial denervation and cyclosporine therapy cause endothelial dysfunction after transplantation and may contribute to the coronary arteriopathy that follows transplantation.

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Poster Displayed: 2:00PM-5:00PM

Author Present: 4:00PM-5:00PM

Hall C, New Orleans Convention Center

Cardiac Surgery: Miscellaneous

TOTAL SIMULTANEOUS AORTIC REPLACEMENT.

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At our Institution, in September 1985 simultaneous total aortic resection from valve to bifurcation was started and since then through December 1988 thirteen patients were operated on. Median sternotomy was used with extension into the left chest; a thoracoabdominal extraperitoneal incision was also added. Cardiopulmonary bypass was instituted with arterial return in a femoral artery and venous drainage with a cannula advanced to the right atrium through the right femoral vein. While the body's temperature was going down, under hypothermic cardioplegia, the ascending aorta was opened; after removing the aortic valve, a composite valve graft was set in place, the coronary arteries were reconnected, and the valved conduit graft was lengthened to the size of the entire aorta. As soon as temperature dropped under 20°C circulation was stopped with the patient in head down position. The carotid arteries were clamped; the aorta was completely exposed and opened from arch to bifurcation. First of all the aortic arch was replaced by anastomosing the carotid arteries to the side of the graft, then the low intercostal arteries were attached and finally the abdominal aorta was replaced. At the end of procedure cardiopulmonary bypass and rewarming were started. Two patients died of hemorrhage, paraplegia was not registered. Early (30 day) survival was 84.6% (11/13) and 10 patients are still alive at latest follow up 6 months to 44 months after operation. There was one late death due to renal insufficiency.

**HEMODYNAMIC STUDY DURING UPRIGHT ISOTONIC EXERCISE BEFORE AND
SIX MONTHS AFTER DYNAMIC CARDIOMYOPLASTY: PRELIMINARY RESULTS**

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The hemodynamic effects of cardiomyoplasty (CM) during exercise has not been already documented. Three male patients (pt), age 46, 48, 44 years, with heart failure class III (N.Y.H.A.) were studied invasively at upright (U) position and during isotonic upright exercise, before (B) and six months (M) after (A) CM. (three M with maximal muscle flap stimulation). The left ventricular ejection fraction (%L) by radienuclide ventriculography of each pt B CM was 19, 28 26 and A CM 30, 41, 40, respectively. The drugs and clinical treatment remained the same or reduced A CM. The following parameters were obtained with maximal O₂ uptake (VO₂ Max): CO (l/min), heart rate (HR) (bpm), pulmonary vascular resistance (PVR) and systemic vascular resistance (SVR) (wood units), pressures (mmHg) of AD, main pulmonary artery (PA), PA wedge (W) and mean systemic artery (AP). Results: (1st, 2nd and 3rd pt. respectively).

CO				PA				AD			
U		EX		U		EX		U		EX	
A	B	A	B	A	B	A	B	A	B	A	B
4.2	3.4	7.0	11.1	93	97	103	130	6	1	26	15
2.6	2.6	5.5	7.7	87	77	110	97	6	1	15	15
5.0	4.2	9.2	10.2	93	90	102	100	5	2	15	0

AP				W				VO ₂ max	
U		EX		U		EX		A	B
A	B	A	B	A	B	A	B		
37	20	63	50	29	5.5	40	30	16	17
60	28	70	53	38	28	70	53	14	19
23	15	48	25	13	10	30	15	13	21

PVR				SVR			
U		EX		U		EX	
A	B	A	B	A	B	A	B
1.9	3.4	3.2	1.8	21	26	11	7.7
8.7	5.4	5.5	4.5	35	30	17	11
2.0	1.2	1.9	0.5	18	22	9.5	5

Comparing the results B CM and A CM: there were reduction of RA, W, AP, PA at U and EX. The PVR, SVR reduced at EX. The VO₂ max and CO increased during EX but not at U, suggesting that improvement after CM was more evident during exercise.

**TETRALOGY OF FALLOT WITH ABNORMAL CORONARY
ARTERY: THE SEARCH FOR AN OPTIMAL STRATEGY OF
REPAIR**

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From 1968 to 1988, 1 775 pts underwent surgery for Tetralogy of Fallot (TOF), 50 of these pts (2.8%) had an abnormal coronary artery (ACA). Mean age was 15.8 months for the 9 pts who underwent palliative surgery and 5.7 years for the 41 complete repairs. Diagnosis of ACA was made by angiographic study in 22 cases, and intraoperatively in others. 36 pts (72%) presented a left anterior descending artery arising from the right coronary artery, and 5 (10%) a single right coronary vessel. When diagnosis was angiographic, palliative surgery was done, especially in small infants and when the ACA crossed the pulmonary outflow tract at the level of the stenosis. When diagnosis was intraoperative, surgical options were: patch reconstruction, conduit placement from the right ventricle to the pulmonary artery (RV-PA) or lowering of the PA on to the infundibular incision. Hospital mortality was 14% (7 pts) including: 3 fatal infarctions related to coronary injury (2 compressions by an RV-PA conduit and 1 inadvertent division), 2 associated with others lesions (mitral anomaly, hypoplastic pulmonary artery bed), 1 residual ventricular septal defect and 1 congestive heart failure after a palliative procedure. One pt had anterolateral infarction due to unintentional injury of the ACA. 4 pts underwent reoperation for early or late RV-PA conduit obstruction. Mean of follow-up was 10.3 years; there was no late mortality; only 3 pts had asymptomatic RV dilatation at echography. Management of TOF with ACA depends of: the circumstances of discovery of the abnormal vessel, the age of the pt, the level and severity of pulmonary outflow tract stenosis, and the contiguity of the ACA to this obstruction. Individualized approach without RV-PA conduit seems the best option to reduce mortality and morbidity of this association.