Results: 1) Right after coronary ligation, MR was only trace with limited increase in LV end-systolic volume (26 ± 7 vs. 1[illegible] ± 5 ml, p < 0.01). 2) After 8 weeks, moderate MR developed as the LV dilated (44 ± 8 ml, p < 0.01 vs. acute), without change in EF (36 ± 7% to 39 ± 3%). 3) As MR developed, the PMs shifted posteriorly and mediolaterally, especially the ischemic medial PM (p < 0.0001), and the annulus dilated (p < 0.01). The only independent predictor of MR was the increase in the tethering distance from PMs to anterior annulus (r^2 = 0.86).

Conclusion: LV and PM ischemia without dilatation fails to produce important MR. Ischemic MR with LV remodeling relates strongly to changes in the 3D geometry of the PM and annular mitral valve attachments.

1222-22 Chordal Force Distribution Determines Systolic Mitral Leaflet Configuration and Severity of Functional Mitral Regurgitation

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Functional mitral regurgitation (MR) is caused by changes in several elements of the mitral valve apparatus. Interaction among these have to comply with the chordal force distribution defined by the chordal coapting forces (F_C) created by the transmitral pressure difference (LVP-LAP), which close the leaflets and the chordal tethering forces (F_T) pulling the leaflets apart.

Five porcine mitral valves were investigated *in vitro* to test the hypothesis that functional MR is directly related to alterations of the chordal force balance, $\Sigma[F_C-F_T]$. Independent variations of the spatial positions of the papillary muscles and LVP-LAP allowed direct measurements of F_T and F_C with chordal force transducers attached to the primary chordae. Chordae geometry, regurgitant orifice area (EOA) and occlusional leaflet area (OLA) needed to cover the leaflet orifice for a given leaflet configuration were assessed by 2D echo and reconstructed three-dimensionally. OLA was used as expression for incomplete leaflet coaptation. Regurgitant fraction (RF) was measured with an electromagnetic flowmeter.

Using ANOVA linear correlations were tested between $\Sigma[F_C-F_T]$ and OLA (regression coefficient (min-max) r^2 = 0.115 – 0.65 mm²/N; p < 0.001), EOA (r^2 = 0.45 – 0.11 mm²/N; p < 0.001) and RF (r^2 = 0.06 – 0.01 10^{-2}/N; p < 0.001). Increasing F_T by papillary muscle malalignment restricted leaflet mobility resulting in an apical and posterior shift of the coaptation line. Anterior leaflet coapting forces increased due to mitral leaflet remodeling generating a nonuniform EOA.

Altered chordal force distribution caused functional MR based on tented leaflet configuration observed clinically. The data suggest that chordal reconstruction should be considered to repair ischemic MR.

1223 Cardiac Surgery With Severe Left Ventricular Dysfunction

Wednesday, April 1, 1998, 3:00 p.m.–5:00 p.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 3:00 p.m.–4:00 p.m.

1223-41 Hospital Recovery After Cardiomyoplasty Surgery: Relationship to Surgeon Experience in a Multi-Center Trial

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Introduction: Hospital mortality in previous FDA studies of cardiomyoplasty has been 12–31%. Lower hospital mortality is likely to be required for acceptable risk/benefit. In order to minimize experience as a factor in hospital mortality, the Cardiomyoplasty - Skeletal Muscle Assist Randomized Trial (C-SMART) instituted a formal training program for all new surgeons.

Methods: Surgical training included hands-on animal experience, observation of a clinical case, and an experienced surgeon at the first case. Between September 1994 and July 1997, 33 treatment patients received surgery.

Results: There was 1 early (within 30 days) witnessed sudden death after discharge from initial hospitalization. Median (range) length of surgery and hospitalization were:

	1st case (52%)	others (48%)	p-value
skin-to-skin time (hrs.)	5.6 (2.5–11.4)	6.1 (4.0–9.2)	0.428
ICU days	4.5 (2–11)	5 (2–7)	0.904
days hospitalized	15 (7–29)	11.5 (6–20)	0.027

Conclusion: Despite over 50% of surgeries being the surgeon's initial cardiomyoplasty experience, early mortality has remained low (3%), and operative time and duration of ICU stay were not effected by surgeon experience. This supports C-SMART's organized surgical training as an effective tool to improve early outcomes after cardiomyoplasty.

1223-42 Bridge to Recovery: Improved Survival in Patients With Acute Viral Myocarditis Using Temporary External Pulsatile Ventricular Support

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Acute viral myocarditis (AVM) can cause rapid cardiac decompensation leading to a spectrum of clinical outcomes, from full recovery to complete heart failure. Conventional therapy with steroids, aggressive diuresis, and inotropic support offers the possibility of complete myocardial recovery, but is often ineffective with subsequent poor outcomes. We examined the ABIOMED voluntary world registry with the use of a temporary external pulsatile ventricular assist device, the BVS 5000 (BVS), in the setting of AVM.

16 patients with AVM underwent device implantation. 11 (69%) underwent biventricular support, 4 (25%) underwent left ventricular support and 1 (6%) underwent right ventricular support for an average of 9 days (range 1–62). The average age for this group was 24 ± 11 years. Complete hemodynamic data were available for 9 AVM patients and are tabulated below:

Variable	PRE-BVS	ON BVS	OFF BVS
Mean BP (mmHg)	60 ± 13	80 ± 12	74 ± 14
Left Atrium (mmHg)	24 ± 4	12 ± 5	15 ± 3
CVP (mmHg)	21 ± 8	13 ± 6	14 ± 4
CI (L/min/m²)	1.4 ± 0.2	2.8 ± 0.6	3.6 ± 0.7
BUN (mg/dl)	52 ± 43	36 ± 21	28 ± 11
Creatinine (mg/dl)	2.2 ± 1.5	1.7 ± 0.2	1.1 ± 0.5
SGOT (IU/l)	506 ± 577	160 ± 398	72 ± 131

Overall, in this cohort, 5/9 (56%) patients had infectious complications, 2/9 (22%) had embolic events, and 5/9 (56%) experienced perioperative renal insufficiency. However, nearly half of these complications developed prior to BVS assistance. 9/16 (56%) AVM patients were both successfully weaned from device support and discharged home.

AVM represents one of the growing indications for which temporary mechanical ventricular assistance may be most effective. Failure to achieve full ventricular recovery while on device support still allows for other surgical alternatives, including implantation of a long term implantable ventricular assist device, or cardiac transplantation.

1223-43 Improved Survival Rates Support Left Ventricular Assist Device Implantation Early After Myocardial Infarction

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Implantation of left ventricular assist devices (LVADs) early after acute myocardial infarction (MI) has traditionally been thought to be associated with high mortality rates. A recovery period of two to three weeks is thus often imposed prior to LVAD implantation, thereby allowing for hemodynamic and end-organ stabilization.

We investigated the impact of preoperative MI on the survival rates of patients who received a Thermo Cardiosystems Incorporated (TCi) LVAD either < 2 weeks (EARLY) or >2 weeks (LATE) after MI.

	EARLY*	LATE*
Total Number	11	9
Age (years)	52.7 ± 7.8	54.7 ± 6.9
Women/Men	1:10	2:7
Pneumatic/Electric	4:7	5:4
Time from MI (Days)	6.8 ± 5.1	22.9 ± 6.4
Intraaortic Balloon Pump	8 (72.7%)	6 (66.6%)
Pulmonary Capillary Wedge (mmHg)	28.0 ± 5.3	28.6 ± 5.8
Cardiac Index (L/min·m²)	2.1 ± 0.5	1.8 ± 0.4
Concomitant Right Ventricular Assist Device	1 (9.1%)	1 (11.1%)
Nitric Oxide	2 (18.2%)	0
Percent Transplanted	6 (54.6%)	5 (55.6%)
Percent Explanted	1 (9.1%)	0
Mortality	2 (18.2%)	4 (44.4%)

*p < 0.05 EARLY vs. LATE for all variables analyzed

In the EARLY group, one patient died of multi-system organ failure 4 days after TCI LVAD insertion, and another died of a pulmonary embolus 3 days after TCI LVAD insertion. One additional patient in the EARLY group, who

received a CABG one day post-MI and an LVAD seven days thereafter, was successfully weaned from device support and underwent device removal 101 days later. He is alive and well 24 months after device removal, with a 50% estimated ejection fraction by postoperative MUGA scan.

The long-term outcome of patients after recent MI may be improved substantially with the prompt use of the TCI LVAD. These data support the early identification and timely application of this modality in post-MI LVAD candidates. The aggressive implementation of this strategy may additionally reveal a subgroup of patients for whom post-MI temporary LVAD insertion may allow for full ventricular recovery.

1223-44 Neuromuscular Function of the Latissimus Dorsi Muscle in Goats After Long-term Dynamic Cardiomyoplasty

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Background: Skeletal muscle deterioration is a limitation to long term dynamic cardiomyoplasty. The etiology of the skeletal muscle deterioration needs to be better understood if strategies for preservation of skeletal muscle are to be developed. Ischemia, decreased muscle preload, muscle overuse, and chronic electrical stimulation have been proposed as causes for muscle deterioration. Denervation of the muscle flap also has been suspected since dispersion of acetylcholine receptors has been reported after dynamic cardiomyoplasty.

Methods: To evaluate latissimus dorsi muscle neuromuscular function after long term dynamic cardiomyoplasty we performed neuromuscular functional analysis and histology on the latissimus dorsi muscle and thoracodorsal nerve of normal goats and goats after 6 months of dynamic cardiomyoplasty.

Results: Four of 6 goats in the cardiomyoplasty group had positive sharp waves or fibrillation potentials, or both, on electromyograms whereas 0 of 6 goats in the control group had these changes ($p = 0.034$). Conduction velocity of the thoracodorsal nerve of goats from the cardiomyoplasty group (58.31 ± 9.80 m/s) was decreased compared to the goats from the control group (71.48 ± 5.71 m/s, $p = 0.02$). Peak voltage amplitudes were decreased in the cardiomyoplasty group (first peak = 1.65 ± 1.17 mV, second peak = 1.78 ± 1.29 mV) compared to the control group (first peak = 27.10 ± 9.21 mV, second peak = 23.30 ± 8.32 mV, $p = 0.002$). Skeletal muscle histologic changes were compatible with denervation. Severe injury to the thoracodorsal nerve in goats from the cardiomyoplasty group was present on nerve histology.

Conclusion: Neurophysiologic and histologic changes after cardiomyoplasty suggest denervation injury.

1224 Pediatric Cardiac Surgery and Intensive Care

Wednesday, April 1, 1998, 3:00 p.m.–5:00 p.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 3:00 p.m.–4:00 p.m.

1224-154 VSD Repair With Fresh Autologus Pericardium, 10 Years Experience

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Background: The objective of this study was to determine the incidence of VSD patch aneurysm when autologus pericardium was used to close a ventricular septal defect.

Pre-operative, early and late post-operative echographic data for each patient were reviewed to evaluate the presence of patch aneurysm.

From February 1986 to March 1997, the charts from 243 patients, under 15 years of age, who were available for follow-up were reviewed. 137 were male, 106 were female. Mean age was 2.12 years (± 0.16) ranging from 4 days to 14.9 years.

Pathologies were: Ventricular septal defect (121), Tetralogy of Fallot (59), Atrioventricular septal defect (41), Transposition of the great artenes (15), Double outlet right ventricle (7).

Follow-up ranged from 1 month to ten years with a mean of 5.89 years (± 1.32).

Results: Only 2 patients were found to have patch aneurysm (~1%). One was a 3 months old girl with an isolated VSD. A residual VSD was detected by echo early post-operatively. The patch became aneurysmal within three months and gradually enlarged. Reoperation was performed 14 months later. There was no aneurysm found 3 years post-operatively. The second patient had an atrioventricular septal defect repair. One month post-operatively the VSD patch was aneurysmal. Subsequent follow-up failed to reveal any enlargement of the patch.

Conclusion: VSD patch closure with autologus pericardium is a safe procedure. Aneurysmal formation was not found to be a problem up to 10 years post-operatively. Autologus pericardium is an excellent alternative for VSD closure.

1224-155 Diastolic Ventricular Function Immediately Before and After Fontan Procedure

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Background: The role of diastolic ventricular function before and after Fontan operation has not been well understood. Considering the unique hemodynamics of the Fontan circulation, it may affect the acute postoperative course. We studied diastolic function immediately before and after Fontan operation.

Methods: Seven patients who underwent Fontan operation (4 with LV morphology and 3 with RV morphology, mean age 2.7 ± 0.4 years) were studied. Six patients with subpulmonary VSD with least shunt served as a control group. Ventricular pressure was recorded with a catheter transducer and simultaneous direct echocardiography was done 20 minutes before and after bypass. Relaxation rate (Tau) using the monoexponential model with a non-zero asymptote was derived from the pressure tracing. Myocardial stiffness constant (Km) in LV morphology heart was measured from diastolic dimension-stress relation. Rate-corrected mean velocity of circumferential shortening (mVcfc) was measured as a systolic performance.

	Tau (msec)		Km		mVcf	
	Pre	Post	Pre	Post	Pre	Post
Fontan	39.9 ± 4.1	103.8 ± 24.2[†§]	17.1 ± 3.8	30.2 ± 6.3[†§]	0.74 ± 0.13[§]	0.73 ± 0.18[§]
Control	34.1 ± 3.9	38.2 ± 9.8	19.0 ± 2.7	9.6 ± 2.3	1.07 ± 0.05	1.15 ± 0.1

† $p < 0.01$ vs Pre. § $p < 0.05$ vs Control

Results: Tau was not different between groups before bypass which significantly elongated after bypass in Fontan group, whereas it did not change in the control group. Km was not different between the groups before bypass which significantly increased after bypass in Fontan group and did not change in the control group. mVcfc was significantly lower in Fontan group before bypass and did not change in both group after bypass. Thus, ventricular relaxation was impaired and myocardial stiffness increased after bypass in Fontan group compared with the control group.

Conclusions: Impaired early relaxation as well as myocardial stiffness may have affected the hemodynamics in the early postoperative phase after Fontan procedure.

1224-156 Changes in Flow Patterns Detected by ECHO in Infants With Hypoplastic Left Heart on Subatmospheric Oxygen

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Supplemental nitrogen has been used to lower F_iO_2 below 0.21 in infants with hypoplastic left heart syndrome during the pretransplant period. "Subatmospheric oxygen" is expected to increase R_p, decrease Q_p, and increase Q_s. We prospectively performed ECHOs on 9 infants before, 1 hour after and 24–48 hours after initiation of supplemental nitrogen. By measuring vessel diameter on 2D ECHO and VTI on doppler ECHO, flow in various vessels was estimated in liters/min (mean ± SE). MPA, RPA and LPA flows decreased. Antegrade PDA flow was unchanged but retrograde flow decreased, so that net systemic flow increased. Innominate flow increased. Mesenteric flow was low but an increase reached significance for the larger celiac trunk. Even modest decreases in systemic saturation redistribute cardiac output from pulmonary to systemic circulation and may improve patient stability while awaiting transplantation.

	Pre	1 Hr	vs Pre	24–48 Hrs	vs Pre
MPA	3.00 ± 0.20	2.30 ± 0.09	$p < 0.01$	2.35 ± 0.26	$p < 0.01$
RPA	0.96 ± 0.13	0.75 ± 0.10	$p < 0.01$	0.72 ± 0.11	$p < 0.01$
LPA	0.83 ± 0.12	0.62 ± 0.07	$p < 0.02$	0.61 ± 0.07	$p < 0.02$
PDA-Ante	0.65 ± 0.08	0.66 ± 0.06	NS	0.66 ± 0.07	NS
PDA-Retro	0.34 ± 0.05	0.22 ± 0.05	$p < 0.01$	0.21 ± 0.04	$p < 0.01$
Innominate	0.15 ± 0.02	0.20 ± 0.05	$p < 0.01$	0.20 ± 0.03	$p < 0.05$
Celiac	0.07 ± 0.01	0.07 ± 0.01	NS	0.09 ± 0.01	$p < 0.05$
S Mesenteric	0.03 ± 0.01	0.04 ± 0.01	NS	0.04 ± 0.01	NS
Saturation	92 ± 1	84 ± 1	$p < 0.01$	82 ± 2	$p < 0.01$