Although it did not reach significant level, serum VEGF in A4 tended to be higher than A3 compared level of serum VEGF among groups. Data were expressed as median and inter-quartile range in parenthesis. RESULTS: Serum VEGF was significantly negatively correlated with arterial saturation (r=-0.8, p<0.01). Serum VEGF in A1 was significantly higher than those in A2 and A3 (372±299 pg/ml in A1 vs. 196±151 pg/ml in A2 and 186±87 pg/ml in A3, respectively), but not higher than VEGF in A2 (252±293 pg/ml)

Background: We have revealed massive expression of Ca binding protein migration inhibitory factor-related protein (MRP) on granulocyte in acute phase of Kawasaki disease. Newton et al. [1 Immunol 1986 100:1427] reported MRP reinforce the ability of adhesion molecule MAC-1, which suggest the relationship between MRP/MAC-1 and vasculitis. We quantified leukocytes MAC-1 expression in Kawasaki disease, and evaluated the adhesion ability between cultured human coronary artery endothelial cell and Kawasaki disease patient's peripheral leukocyte. Furthermore, we evaluate the leukocyte-endothelial cell adhesive ability using the patients' plasma, before and after gamma globulin therapy, to see the gamma globulin effect for this system. (Materials and Methods) We quantified MAC-1 expression from the patients' plasma (the patients' plasma before Kawasaki disease patient's leukocyte (p=0.1) and was converted to cDNA by RT-PCR, and Mac-1 expression was evaluated by quantitative PCR (Applied Biosystems; GeneAmp 7700). Patients' leukocytes, labeled with BCECF-AM, exposed to cultured human coronary artery endothelial cell, and leukocyte adhesion assay were performed. Leukocyte adhesion assay was also carried out using the patients' plasma, pre/post gamma globulin therapy. (Result) Mac-1 expression was a peak on acute phase of Kawasaki disease and significantly decreased after 1 month of onset. The patients' leukocytes adhesion to endothelial cell was significantly increased, which was significantly inhibited by addition of anti-Mac-1 antibody. The patients' plasma before gamma globulin therapy significantly increased leukocyte-endothelial cell adhesion, which was abolished by the plasma of post gamma globulin therapy. We postulated Mac-1 play the key role for leukocyte invasion into endothelium, which is the milieus for vasculitis development. Antigen gamma globulin therapy is effective through inhibiting leukocyte-endothelial cell adhesion.

POSTER SESSION
1142 Pediatric Electrophysiology and Intervention
Monday, March 18, 2002, 3:00 p.m.-5:00 p.m.
Georgia World Congress Center, Hall G
Presentation Hour: 4:00 p.m.-5:00 p.m.

1142-97 Termination of Intracardial Reentrant Tachycardia Using Implanted Pacemakers in Patients Following Atrial Switch Repair of Transposition of the Great Arteries
Gerald A. Savar, David J. Bradley, Peter S. Fishbach, Kristen A. George, Sarah S. Lacy, MacDonald Dick, II, University of Michigan Congenital Heart Center, Ann Arbor, Michigan.

Background: Intracardial reentrant tachycardia (IART) has been associated with patients (pts) following atrial switch operation (ATS) for transposition of the great arteries who have sick sinus syndrome (SSS). Anti-tachycardic pacing (ATP) has been used to convert IART in these pts but its efficacy using implanted pacemakers and safety have been questioned.

Methods: Clinical records of pts who had undergone atrial or dual chamber pacemaker placement for SSS were retrospectively reviewed. Implant details, number and results of ATP attempts, medications, and outcomes were recorded.

Results: Of the 29 pts studied, 19 were endocardial in 25 and epicardial in 4 and was guided by pacing threshold data with no attempt to induce and convert IART at implant. 21 (72%) had 1 or more episodes of IART and all were taking digoxin, 5 on beta-blockers, and 2 on amiodarone. 4 of 21 had IART prior to pacemaker implant without recurrence post implant. 17 underwent ATP for conversion of I to 30 episodes per patient (median 3 episodes) 7 pts experiencing 47 episodes had automatic anti-tachycardic pacemakers. ATP therapy consisted of 15 to 40 beats at a cycle length of 50% to 80% of the IART cycle length (median 67%, minimum 150 ms) delivered at an amplitude and pulse width twice the bradycardia pacing settings. ATP cycle length began at 60% of the IART cycle length and was decreased until conversion. In most episodes were converted after many induced attempts. 1 ATP attempt increased the IART rate but did not increase the ventricular rate. 4 ATP attempts in 2 pts caused atrial fibrillation that reverted spontaneously to sinus rhythm. All pts were hemodynamically stable in IART before and during ATP.

Conclusion: Bilateral pacing failed to control IART in some pts. By implanted pace makers is safe and effective in converting IART in pts post ATS procedure who are not severely hemodynamically compromised by IART avoiding the need for DC cardioversion. IART induction is not required at pacemaker implant. Patients with atrial overdrive suppression may not be stimulant at maximum output. Use of post ATS WSS as the incidence of IART is high and ATP is highly effective and safe.

1142-98 Comparison of Adenosine-Sensitive and Adenosine-Resistant Accessory Pathways in Children
David J. Bradley, Loren M. Madden, Christopher B. Stefanelli, Elizabeth V. Saarel, Jeremy D. Aronson, Gerald A. Saber, Peter S. Fishbach, MacDonald Dick, II, University of Michigan Congenital Heart Center, Ann Arbor, Michigan.

Background: Adenosine (Adn) is routinely used during electrophysiology study (EPS) of patients with supraventricular tachycardia (SVT) to assess presence of accessory atrioventricular pathways (AP) and verify success of radiofrequency ablation (RFA). Adenosine blocks conduction in the atrioventricular node, but generally not in APs. We investigated the incidence and properties of Adn-sensitive and Adn-resistant APs.

Methods: The electrophysiology patient database at the University of Michigan Congenital Heart Center was queried for all patients with accessory pathways (APs) and SVT undergoing EPS between 1996 and 2001. Clinical records and EPS tracings were reviewed. Adn was used in 132 patients. Adn was administered during ventricular pacing at a stable infranormal dose of 200μg/kg with a maximum dose of 12mg was used. All patients were under 21 years of age (median 11.8 years).

Results: A total of 151 patients with APs and SVT were identified; Adn was used in 132 patients (137 APs). Of these, 20 APs (15%) blocked with adenosine; RFA was performed on 85 (117/137) APs, and was successful in 94%, (115/118). Adn-sensitive, v 92% (33/101) Adn-insensitive APs (p=0.01). Adn-sensitive and insensitive APs did not differ significantly with regard to patient age (10.6±5.8 v11.4±7.4 years), gender (47% v. 55% boys), or SVT cycle length (314±59 v 315±35ms). Procedure times, total RF application sites, and erapsulation success, Adn-sensitive and resistant pathways did not correlate with septal v wall free-wall (35% v 28% septal, p=0.16), or left v right-sided (43% v. 60% left, p=0.36) locations. In contrast, Adn-sensitive APs were less likely to demonstrate pre-excitation in sinus rhythm (37% v 61%, p=0.04), and had significantly longer antral electrographic effective refractory periods (ERP) when pre-excitation was present (340±42 vs 278±58ms, p=0.05). Retrograde ERP also trended longer in Adn-sensitive pathways (270±57 vs 259±43) though this difference was not statistically significant (p=0.10).

Conclusions: Adn-sensitive APs occur frequently in pediatric patients, Adn is not an absolute test of AP presence. Adn-sensitive APs are significantly less likely than Adn-resistant APs to cause pre-excitation, and have longer antegrade ERGAPs when they do.

1142-99 Biologic Response to the HELEX® Septal Occluder Implantation in the Canine Heart

Background: The HELEX™ device has recently been used in humans for transcatheter secundum atrial septal defect (ASD) closure. We report the data on the biologic response to implantation of this device in the canine heart. Methods: Histologic data from 29 animal implants in non-randomized prospective studies conducted at W.L. Gore & Associates Inc and The Cleveland Clinic Foundation were analyzed. Animals had either a surgically created or a percutaneously created ASD and immediate implantation of a HELEX™ device. Animals were sacrificed at intervals from 2 days to 1 year. Devices were examined grossly, with multiple sections stained using hematoxylin and eosin. Distal edge of all devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage. Distal devices were assessed for fibrous connective tissue (FCT) and endothelial-like cell coverage.