901-24 Safe Magnetic Resonance Imaging of Pacemaker Patients

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Most authorities feel MRI of pacemaker (PM) patients is absolutely contraindicated.

Methods: We collected data on institutional practices regarding MRI of PM patients and identified previously unreported PM patients who have undergone MRI through a survey of cardiologists (n = 106) and radiologists (n = 86) at predominantly tertiary care (92%) institutions. Forty-one % (n = 78) of the 192 surveys were returned over a 2 month period.

Results: Practice policies varied, with 33% of cardiologists responding they would perform MRI if needed and 93% of radiologists completely prohibiting MRI in PM patients. We identified 19 patients who were scanned a total of 20 times. A variety of strategies were suggested or used to help scan PM patients safely, including avoiding MRI of PM dependent patients, reprogramming the PM to asynchronous or non-capture mode, having PM specialists present and use of enhanced non-invasive monitoring. A variety of unipolar, bipolar, single and dual chamber PM underwent MRI including models from CPI, Intermedics, Medtronic, Siemens Pacesetter, and Telectronics. Sixteen of the scans occurred without the knowledge that the patient had a pacemaker. Seventeen scanning events occurred "without apparent consequence" to the patient or PM. One unmonitored PM patient died who was inadvertently scanned. One patient felt discomfort at the PM pocket, and one patient developed a rapid heart rate during MRI. These latter two patients were unharmed after the MRI was aborted. All of the PM interrogated afterward were found unaffected.

Conclusions: The results highlight attitudinal differences regarding the perceived safety of scanning PM patients. PM patients may safely undergo MRI if appropriate strategies are employed that account for potential ill effects on the PM by MRI.

CARDIAC TRANSPLANTATION — BASIC AND CLINICAL

901-25 The Paradox of Donor Stimulation of Endothelial-induced Smooth Muscle Growth

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Cardiac allograft vasculopathy (CAV) is the major cause of long-term morbidity and mortality in cardiac transplant recipients. It appears to be related to immune damage to the coronary endothelial cells, resulting in intimal proliferation. In order to delineate the mechanisms by which CAV can occur, a co-culture model of human endothelial cells (EC) and smooth muscle cells (SMC) obtained from the donor at the time of organ procurement was utilized. These cells were separated by collagenase digestion, and cultured for four passages. EC and SMC were then grown to confluence in the separate chambers of a co-culture plate separated by a 0.45 micron Millipore filter. Preserved lymphocytes (LYMPH) obtained from the donor and pooled blood lymphocytes from the recipient 3–4 weeks following transplant were added to the EC well so as to cause an immunologic stimulation of the EC. None of the recipients were exposed to monoclonal or polyclonal antibodies to lymphocytes. All cultures and assays were done in triplicate. Results are as follows:

Patient#	% Increase in donor lymph H^3 thymidine	p Value
Donor 1	+51	0.04
Donor 2	+45	0.05
Donor 3	+104	0.05
Donor 4	+25	0.01
Donor 5	−19	NS

The donor EC/donor LYMPH co-culture stimulated SMC growth measured by H^3 thymidine incorporation in 4 of 5 patients. The donor EC/recipient LYMPH co-culture did not result in significant SMC H^3 thymidine incorporation. *Conclusion:* These paradoxical findings of a lack in significant SMC proliferation in the recipient stimulated donor cells continue to raise questions in relation to the effects of circulating lymphocytes on the development of cardiac allograft vasculopathy.

901-26 Coexpression of Vimentin and Ki 67 Indicates Cardiomyocyte Regeneration After Acute Rejection in Human Cardiac Allografts

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Electron microscopy suggests that after acute cardiac allograft rejection (AR) injured adult human cardiomyocytes regenerate. They dedifferentiate and reacquire the condition of embryonic myocytes. A close connection between regeneration and vimentin (vim) expression was shown in an animal model. Vim is an intermediate filament expressed in muscle tissue during development and replaced lateron by desmin. In this study, immunohistochemistry (alkaline phosphatase-, peroxidase- and fluorescence-techniques) was used to further analyze this regeneration process. We obtained endomyocardial biopsies (EMB) of 41 heart transplant patients (9 f, 32 m, mean age 46, 1–60 months postop) on 104 occasions. EMB were graded for AR (ISHLT) and (double-)stained with vim, desmin and Ki67, a proliferating cell marker.

Results: vim was expressed in 1–5% of cardiomyocytes in 19/104 EMB (vim+). Double staining with desmin proved the muscular origin of vim+ cells. Coexpression of vim and Ki67 indicated myocyte proliferation. Of the 19 vim+ EMB all had AR ≥ IA (ISHLT) either in this or in the preceeding EMB (14 ± 7 days). Of the 85 vim negative EMB 59 had no AR, 26 had AR ≥ IA (ISHLT) in this or the preceeding EMB.

Conclusion: This is the first report to show that vim can be expressed by adult cardiomyocytes. Expression of vim and Ki67 appears to be related to AR, but was not found in all EMB with evidence for AR, either due to sampling error or lack of regeneration in some AR. The data suggest that myocytes damaged by AR may gain myofibrillar regeneration by going through a sequence of embryological development as indicated by expression of vimentin and Ki67. Immunohistochemistry is a useful tool to investigate cardiac regeneration.

901-27 Regional Wall Motion Analysis by Dobutamine Stress Echocardiography in Heart Transplant Recipients with Normal Coronary Angiographic Findings: Comparison with Intravascular Ultrasound

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Coronary angiography (ANGIO) is still the reference method for diagnosis of coronary allograft vasculopathy (CAV). To investigate, if normal ANGIO excludes CAV, 42 patients (P, 51 ± 7 yrs, 49 ± 22 months after heart transplantation) were studied prospectively by ANGIO, intravascular ultrasound (IVUS, quantitative analysis of degree and extension of intimal hyperplasia; modified Stanford grading, grades 1–6) and dobutamine stress echocardiography (DSE, 5–40 mcg/kg/min, 5 min stages). Regional wall motion abnormalities (WMA) were assessed qualitatively (2-D-echo, 16 segment model) and quantitatively (M-Mode, systolic wall thickening of septum (IVS) and LV posterior wall (LPW). P with allograft rejection (>grade 1A ISHLT) were excluded from analysis. ANGIO was completely normal in 33 P; these P were allocated according to mean IVUS grading (mean, 4.1 coronary segments per P): group1, mild-to-moderate intimal hyperplasia (mean grade <3.5), n = 20; group2, marked-to-severe intimal hyperplasia (mean IVUS grade ≥3.5), n = 13. No group1 P had WMA at rest, 2/20 P developed WMA during DSE (6/320 segments). In group2, WMA at rest were found in 6/13P (26/208 segments). During DSE, WMA increased in these 6P and newly developed in 6 P (total, WMA in 12/13 group2 P in 72/208 segments during DSE). Mean systolic thickening of IVS (group2 vs. 1: rest, 23 vs. 33%, $p < 0.01$; max.DSE, 32 vs. 62%, $p < 0.01$) and LPW (rest, 38 vs. 58%, $p < 0.01$, max.DSE, 65 vs. 96%, $p < 0.01$) were signif. smaller in group2 than group1. In total, in 15/33 P with normal ANGIO, evidence of CAV was found (IVUS ≥ 3.5 and WMA at DSE, n = 12; IVUS ≥ 3.5 only, n = 1; WMA at DSE alone, n = 2).

Conclusion: A normal ANGIO does not rule out CAV. WMA during DSE are associated with marked to severe intimal changes as assessed by IVUS. These changes appear to preceede ANGIO changes. Analysis of WMA by DSE is a feasible noninvasive method for early detection of CAV. Costly and invasive ANGIO, in particular when performed without IVUS, may be not necessary at regular annual intervals in heart transplant recipients with normal DSE findings.