were processed by a non linear anisotropic filter and LV edges were identified using an edge detection method to evaluate diastolic function. Our aim was to analyze the IVPG and diastolic function in HOCM patients with PTSA.

Methods: 19 patients had an echocardiogram performed at baseline and after PTSA treatment (mean follow up 5.8±1.2 months). Diastolic parameters were obtained using PW-Doppler through the mitral valve (E, A, DT) and tissue Doppler imaging (Ea). Color M-mode was used to obtain the flow propagation velocity (Vp) through the mitral valve and to calculate IVPG offline using custom written software. LV dimensions were obtained. LV outflow tract (VGT) gradients were obtained.

Results: LVOT gradient decreased from 62±10 to 29±5 mmHg (p<0.001), severity of mitral regurgitation (MR) decreased from 2.1±0.2 to 1.3±0.2 (p=0.01). The septal size decreased from 2.3±0.9 to 1.9±0.6 mm (p<0.01). Diastolic results are shown in the Table.

Conclusion: This study is the first prospective determinant of the natural history of aspirin resistance, documenting a greater than 2-fold increase in the risk of major adverse events associated with aspirin resistance.

4:15 p.m.

Loss of Thrombomodulin Expression Impairs Vein Graft Thromboreistance

Antony Y. Kim, Kenneth Lee Baughman, Peter L. Wallismy, Frank D. Kologod, C. E. Sian, Jason Speny, Clayton Deming, Eric Peck, Jay Shake, Gregory Ang, Charles Esmon, Renu Virmani, Jeffrey Rade, Johns Hopkins School of Medicine, Baltimore, Maryland.

Background: Thrombosis is the major cause of early vein graft failure. The aim of this study was to determine whether alterations in the expression of the anticoagulant proteins, thrombomodulin (TM) and the endothelial cell protein C receptor (EPCR), impair endothelial thromboreistance and contribute to vein graft failure.

Methods: Immunohistochemical analysis of autogenous rabbit vein grafts revealed that the expression of TM, but not EPCR, was reduced by 98% 3 days after implantation, with a near 80% reduction in bound thrombin activity on day 7, to levels comparable to normal veins (0.4 vs. 5.2 ± 1.1 μM/cm², respectively, p=0.74). Surprisingly, neointima formation was not affected by this inhibition of local thrombin generation. Conclusions: These results suggest that early loss of TM expression enhances local thrombin generation that contributes to early vein graft failure due to thrombosis, but does not contribute significantly to late vein graft failure due to neointimal hyperplasia.

4:30 p.m.

Improvement in Diastolic Suction in Patients With Hypertrophic Obstructive Cardiomyopathy After Septal Ablation

Alabrandi Porrer, Maria Sitges, Rebecca Smith, Neil L. Greenberg, Imren Vlassak, Takahiro Shiota, Murtu E. Tuzcu, Nichoress Smidt, Harry M. Laver, James D. Thomas, Mario J. Garcia, Department of Cardiovascular Medicine, The Cleveland Clinic Foundation, Cleveland, Ohio.

Background: Septal alcohol ablation (PTSA) is an effective treatment in hypertrophic obstructive cardiomyopathy (HOCM). Intraventricular pressure gradient (IVPG) is a novel method to evaluate diastolic function. Our aim was to analyze the IVPG and diastolic function in HOCM patients with PTSA.

Methods: 19 patients had an echocardiogram performed at baseline and after PTSA treatment (mean follow up 5.8±1.2 months). Diastolic parameters were obtained using PW-Doppler through the mitral valve (E, A, DT, pulmonic venous flow (S), mitral annulus tissue Doppler imaging (Ea). Color M-mode was used to obtain the flow propagation velocity (Vp) through the mitral valve and to calculate IVPG offline using custom written software. LV dimensions were obtained. LV outflow tract (VGT) gradients were obtained.

Results: LVOT gradient decreased from 62±10 to 29±5 mmHg (p<0.001), severity of mitral regurgitation (MR) decreased from 2.1±0.2 to 1.3±0.2 (p=0.01). The septal size decreased from 2.3±0.9 to 1.9±0.6 mm (p<0.01). Diastolic results are shown in the Table.

Conclusions: IVPG is a reliable indicator of diastolic function improvement in HOCM after PTSA as indicated by the increase in IVPG and change in other diastolic parameters.

4:45 p.m.

L-Arginine Protects Human Heart Cells From Simulated Anoxia and Reoxygenation

Subodh Verma, Nontsuke Shiono, Ren-Ka Li, Donald A. Mickie, Paul W. Fedek, Richard D. Weisel, University of Toronto, Toronto, Ontario, Canada.

The present study was conducted to evaluate the direct effects of L-arginine in a human ventricular heart cell model of simulated ischemia and reperfusion independent of alternate cell types such as endothelial cells, neutrophils, platelets or fibroblasts. Human ventricular cell cultures were subjected to 90 minutes of low volume ischemia and 30 minutes of reperfusion. L-arginine (0.5 mM) was administered during the pre-ischemic period or during the reperfusion phase. Nitric oxide synthase (NOS) activity, nitric oxide (NO) production, cGMP levels and cellular injury were assessed. The effects of L-arginine on cell signaling, the expression of the endothelial nitric oxide synthase (iNOS), the endothelial nitric oxide synthase (eNOS), protein kinase (PKG), aPKG antagonist (glibenclamide) were examined. Our data indicate, that ischemia and reperfusion increased NO activity and facilitated the conversion of L-arginine to NO, 

4:00 p.m.

A Prospective, Blinded Determination of the Natural History of Aspirin Resistance Among Stable Cardiac Patients

Patricia A. Gum, Kandica Kottke-Marchant, Eric J. Topol, The Cleveland Clinic Foundation, Cleveland, Ohio.

Background: Aspirin resistance, as defined by comprehensive, ex vivo platelet function testing as well as presumed clinical unresponsiveness to aspirin, has been previously reported by our group and others. However, little information exists linking the laboratory detection techniques and is based on a model of the vessel and uses knowledge of the morphologic structures. The stent contour is detected based on the high intensity of the stent struts as compared to other structures and their expected location in the vessel wall based on an approximately circular model. The detection method itself is able to perform correlations based on a continuously three-dimensional structure.

Methods: The (semi-)automated approach is a combination of transversal and longitudinal contour detection techniques and is based on a model of the vessel and uses knowledge of the morphologic structures. The stent contour is detected based on the high intensity of the stent struts as compared to other structures and their expected location in the vessel wall based on an approximately circular model. The detection method itself is able to perform correlations based on a continuously three-dimensional structure.

Results: In a set of 150 slices from different pullback series (acquired with Boston Scientific and EndoSonics equipment) the automatically detected stent boundaries were compared to manually drawn stent boundaries. The comparison of the cross-sectional stent areas for the manually traced and automatically detected boundaries resulted in a r value of 0.99. In a set of 50 pullback runs (acquired with EndoSonics equipment) the entire stent segment was analyzed automatically and the most distal and the most proximal slice in a continuous series of slices containing stent struts was detected to assess the stent length. The comparison of these distances with the original stent lengths resulted in an average overestimate of 0.35 +/- 1.42 mm by IVUS. The start and end point of a stent is not well defined because the catheter moves in and out the stent, due to cardiac motion. Conclusion: Due to the flexible use of more than two longitudinal cutplanes and the advanced knowledge-guided contour detection approach, the new IVUS analysis system has proven to be suitable for clinical research studies. The inclusion of the stent boundary is very useful for stent studies e.g. to study the effect of drug coated stents.

4:00 p.m.

L-Arginine Protects Human Heart Cells From Simulated Anoxia and Reoxygenation

Subodh Verma, Nontsuke Shiono, Ren-Ka Li, Donald A. Mickie, Paul W. Fedek, Richard D. Weisel, University of Toronto, Toronto, Ontario, Canada.

The present study was conducted to evaluate the direct effects of L-arginine in a human ventricular heart cell model of simulated ischemia and reperfusion independent of alternate cell types such as endothelial cells, neutrophils, platelets or fibroblasts. Human ventricular cell cultures were subjected to 90 minutes of low volume ischemia and 30 minutes of reperfusion. L-arginine (0.5 mM) was administered during the pre-ischemic period or during the reperfusion phase. Nitric oxide synthase (NOS) activity, nitric oxide (NO) production, cGMP levels and cellular injury were assessed. The effects of L-arginine on cell signaling, the expression of the endothelial nitric oxide synthase (eNOS), protein kinase (PKG), aPKG antagonist (glibenclamide) were examined. Our data indicate, that ischemia and reperfusion increased NO activity and facilitated the conversion of L-arginine to NO, 

4:00 p.m.