

1040 Contrast Echocardiography: Clinical Applications

Wednesday, March 27, 1996, 3:00 p.m.—5:00 p.m.
Orange County Convention Center, Hall E
Presentation Hour: 4:00 p.m.—5:00 p.m.

1040-61 Real-Time, Three Dimensional Echocardiography With Saline Contrast Enhancement: Methods and Possibilities

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To test the hypothesis that real-time, three-dimensional echocardiography (RT3D) images of the right ventricular chamber (RV) could be obtained with echo contrast, peripheral intravenous injections of agitated saline were made into 14 volunteers using the second generation successor to the Duke RT3D system. The device uses a matrix phased array transducer (2.2 MHz: 40 × 40 elements) and a 16:1 receive/transmit parallel processing scheme to develop a 4,096 line scan which interrogates everywhere in a 60 degree pyramidal volume at 22 frames/sec. Images are simultaneously comprised of two steerable, intersecting conventional B-scan sector arcs with multiple (usually three) C-scan planes parallel to the transducer face and adjustable anywhere in range within the scan volume. This requires no gating of ECG and respiration or off line reconstruction techniques. The added capability to capture an entire pyramidal scan volume allows retrospective examination of any target within the volume by adjusting B and/or C scans. Contrast images were readily made in all 14 subjects (29 injections) and the right chambers and RV outflow tract were identified in all.

A previously unsuspected patent foramen ovale was detected in one subject. Calculation of RV volumes were attempted by outlining the areas of contrast within the C-scan (variably adjusted in the captured volume). Volumes ranged from 68.0 to 94.5 ml. These data indicated that RT3D contrast injections are possible and opens the opportunity for more precise testing and application of contrast methods for the development of new descriptors of cardiac flow or volume.

1040-62 Contractile Versus Microvascular Reserve for the Determination of the Extent of Myocardial Salvage After Reperfusion: The Effect of Residual Stenosis

Jiri Sklenar, Gustavo Camarano, Suad Ismail, N. Craig Goodman, Sanjiv Kaul. *University of Virginia, Charlottesville, VA*

To test the hypothesis that microvascular reserve is a better indicator of the extent of viable myocardium postinfarction than the presence of contractile reserve, we studied 15 open-chest dogs undergoing coronary occlusion followed by reperfusion. Contractile reserve was measured using two-dimensional echocardiography and microvascular reserve was measured using myocardial contrast echocardiography, both before and after placement of a stenosis. Infarct size was measured using triphenyl tetrazolium chloride. In the absence of a stenosis, the relationship between infarct size and wall thickening improved with increasing doses of dobutamine ($r = 0.62$, $r = 0.88$, and $r = 0.92$ for 5, 10, and 15 $\mu\text{g}/\text{kg}/\text{min}$, respectively). In the presence of a stenosis, however, the relationship became worse with increasing doses of dobutamine ($r = 0.53$, $r = 0.53$, and $r = 0.38$ for 5, 10, and 15 $\mu\text{g}/\text{kg}/\text{min}$, respectively). There was a fair correlation between infarct size and perfusion defect size on myocardial contrast echocardiography after reperfusion ($r = 0.82$) with the defect size underestimating infarct size by approximately 20%. This relationship improved significantly during infusions of both adenosine ($r = 0.99$) and dobutamine ($r = 0.94$) in the absence of stenosis. The correlations remained good ($r = 0.95$ for adenosine and $r = 0.81$ for dobutamine) also in the presence of stenosis. It is concluded that microvascular reserve is superior to contractile reserve for defining the spatial topography of necrosis and hence, the extent of viable myocardium within the infarct bed after reperfusion, particularly when a stenosis is present on the infarct-related artery.

1040-63 Intramyocardial Heterogeneity of Integrated Backscatter Cyclic Variations Blunting Induced by Atrial Pacing in CAD Patients

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Integrated backscatter cyclic variations (IBScv) reflect regional myocardial contractility and are blunted during myocardial ischemia. Experimental studies have shown that subendocardial thickening is greater than the subepicardial

one and that acute myocardial ischemia mainly impair the former. Accordingly, we hypothesized that stress induced myocardial ischemia mainly affects subendocardial IBScv. To verify this hypothesis multiplane transesophageal echocardiography and simultaneous atrial pacing were performed in 5 normal subjects and in 9 pts with significant ($\geq 50\%$ coronary narrowing) CAD. 28 myocardial segments with adequate image quality were considered for analysis: 12 in normal subjects and 16, supplied by a narrowed coronary artery, in pts with CAD. In each pt a transgastric two-chamber view was acquired and IBScv were calculated in subendo- and subepicardium both at rest ($74 \pm 9 \text{ b/m}^2$) and at peak-pacing ($136 \pm 15 \text{ b/m}^2$). A prototype (AD system, Hewlett-Packard Co.) was used to acquire and analyse densitometric images. **Results:**

		Rest (dB)	Peak-pacing (dB)
Normals (12 segments)	Subendocardium	7.7 ± 2.2	7.8 ± 2.5
	Subepicardium	5.3 ± 3.1	5.5 ± 3.0
CAD (16 segments)	Subendocardium	7.7 ± 1.6	$5.9 \pm 2.0^{**}$
	Subepicardium	5.2 ± 3.0	6.0 ± 1.8

* $p < 0.05$; ** $p < 0.005$ vs rest.

Conclusion: IBScv are greater in the subendocardium than in the subepicardium. Atrial pacing stress test does not affect IBScv in normals, while it blunts them in CAD pts; blunting exclusively occurs in the subendocardium. Heterogeneity of IBScv intramyocardial changes caused by stress induced ischemia must be taken into account when using IBScv for investigating myocardial ischemia.

1040-64 Further Evidence That the Myocardial Transit Rate of Microbubbles Reflect Endothelial Function: An Evaluation in a Reperfusion Model With a Constant Flow Rate

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We have previously shown that the transit rate of sonicated albumin microbubbles (AM) is slowed in the presence of crystalloid cardioplegia (CC) and we have hypothesized that this is because of microbubble adherence to the endothelium which become dysfunctional in the presence of CC. To further explore this issue, we studied myocardial AM and red blood cell (RBC) transit rates in an arrested heart perfused at a constant flow rate alternating with arterial blood and CC. AM transit rate was derived using myocardial contrast echocardiography (MCE) in 8 open-chest dogs where the AM were injected as a bolus into the cross-clamped aorta. RBC transit rates were derived from time-activity curves obtained from the myocardium using a Cs^{125} miniature γ -detector. The AM transit rate was 1.5 ± 0.5 during baseline blood perfusion and slowed to 0.3 ± 0.2 during CC infusion. Although it improved during blood reperfusion compared to CC (1.0 ± 0.2), it was markedly slower ($p < 0.001$) compared to baseline blood perfusion. In comparison, the transit rate of radiolabeled RBCs remained unchanged (0.20 ± 0.09 , 0.19 ± 0.07 , 0.16 ± 0.03 , $p = 0.40$). AM transit rate was not influenced by the total protein content, osmolality, pH, pO_2 , pCO_2 , K^+ concentration, or temperature of either the perfusate or the dog blood. We conclude that the transit rate of AM can be used for in-vivo real-time measurement of microvascular endothelial function. This potential application of MCE appears to be without parallel in cardiac imaging.

1040-65 Coronary Flow Velocity Immediately After Reperfusion Reflects Myocardial Microcirculation in Canine Acute Myocardial Infarction Models

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To examine the relationship between coronary blood flow pattern immediately after reperfusion of acute myocardial infarction (AMI) and myocardial microcirculatory injury, coronary flow velocity variables were compared with myocardial contrast echocardiographic images in 10 canine AMI models (LCX 4 hrs. clamp procedure). We measured coronary flow velocity 10 minutes after reperfusion using Doppler guide wire (FlowWire). Averaged peak velocity was $14.6 \pm 6.3 \text{ cm/s}$, averaged systolic peak velocity was $9.0 \pm 3.5 \text{ cm/s}$, averaged diastolic peak velocity was $19.9 \pm 6.9 \text{ cm/s}$ and diastolic to

