1004-57 Regional Left Ventricular Function by Intraventricular Ultrasound in Patients with Myocardial Infarction

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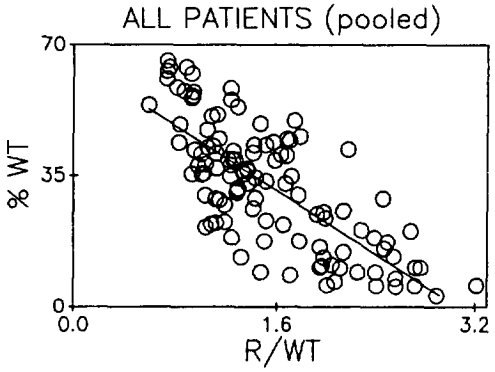
Regional left ventricular (LV) dysfunction induced by ischemia/infarction is accompanied by increased end-systolic stress because the ischemic LV wall is unable to generate enough tension to contribute effectively to systole. To explore the possibility of assessing regional LV dysfunction as changes in LV wall stress we performed intraventricular echocardiography in 10 patients with a 6.2 french/12.5 MHZ catheter at the time of cardiac catheterization. Cross-sectional images obtained at the level of the papillary muscles were analyzed by computer aided system to assess left ventricular wall thickness and radius of curvature (RC) in 16 equi-angular segments. End-systolic segmental endocardial radius of curvature divided by LV wall thickness obtained as segment area divided by the average of endo and epicardial arc lengths was utilized as an index of regional LV performance proportional to segmental LV wall stress. Percent wall thickening (WT%) was reduced (20.7 ± 14.5%) in the territory perfused by the stenosed artery determined at catheterization, when compared with WT% obtained from territory perfused by normal coronaries (34.4 ± 15.8%, $p < 0.05$). In addition, systolic wall thickening was inversely related to the ratio of RC to WT at end-systole ($r = 0.75$, %WT = 65.5 − 21.4 (RC/WT), $p < 0.05$) reflecting regional systolic dysfunction with increased circumferential end-systolic wall stress in those regions. In conclusion, intraventricular echocardiography correctly detects regional left ventricular dysfunction and its geometric consequences to local LV performance induced by ischemic myocardial damage. This technique may play an important role in monitoring myocardial injury by ischemia during invasive interventional procedures.



1004-58 Quantitative Intravascular Ultrasound Imaging: Evaluation of an Automatic Approach

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Intravascular ultrasound (IVUS) imaging allows a detailed interpretation and a precise quantitation of vascular cross-sections. To reduce the existing observer variabilities, quantitative intravascular ultrasound (QIVUS) needs to be made available.

We have developed a fully automatic edge detection technique, based on adaptive active contour models and called ADDER (Adaptive Damping Dependent on Echographic Regions), that allows the quantitation of the vascular luminal area (LA). Using a 20 and 30 MHz mechanically rotated transducer mounted at the tip of a 4.8 F or 3.5F catheter, 53 normal and pathological arterial segments (from coronary, renal, splenic, iliac, and carotid arteries) were imaged in vitro. These images were analyzed by two experts, E1 and E2, who manually traced the intra-luminal contour twice for each image, as well as with ADDER.

Intra-observer variabilities for LA's were found to be excellent (−1.7 ± 3.3% for E1, 1.3 ± 6.5% for E2). The inter-observer variability was: 2.1 ± 4.3%. The success factor for ADDER was 98.1%. Its intra-observer variability was null, since the method always finds a unique contour. The correlation between the automatically found LA and the manual LA was: $r = 0.99$ ($y = 1.03x + 0.89$ mm^2). Morphometric variations between manually and automatically traced contours, analyzed by the centerline method, were 98 ± 139 μm on average (N = 52 images).

In conclusion, the ADDER automatic contour detection applied to IVUS images is robust and characterized by small systematic and random errors; therefore, QIVUS is a useful tool in clinical research trials.

1004-59 Vascular Acoustic Emissions During Angioplasty: Potential Role in Identification of Induced Dissection

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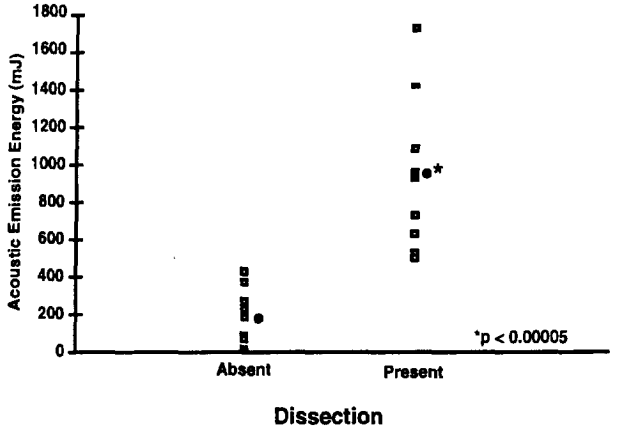
A fundamental mechanism of balloon angioplasty (BA) is plaque rupture. Rupture leading to dissection, however, has been implicated as an underlying factor responsible for both acute and chronic adverse outcomes. Acoustic emissions (AE) — transient sound waves generated by microstructural alterations of a material subjected to mechanical stress — may provide a novel means of characterizing BA-induced tissue trauma. Using a novel acoustic sensor system, we examined the relationship between cumulative AE energy released by human arterial tissue during BA and the observed pathologic injury. Post-mortem human arterial specimens (19) were subjected to identical BA with simultaneous monitoring of intraluminal pressure and AE. Sound energy was integrated throughout the pressurization period to obtain an estimate of the cumulative AE energy released during dilatation. Post-angioplasty inspection revealed a marked difference in AE energy released by specimens that experienced traumatic dissection vs. non-dissection dilatation:



Sound energy released by vascular tissue undergoing balloon angioplasty discriminates dissection from non-dissection tissue trauma. Given the deleterious role that dissection can play in BA, this novel system may provide a means of improving procedural outcome.

1004-60 Atheromas at the LAD Origin Develop Opposite the Flow Divider: Implications Regarding the Role of Hemodynamic Shear in Atherogenesis Derived from Intravascular Ultrasound

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The role of hemodynamic shear stresses in atherogenesis remains uncertain. Necropsy studies suggest that atherosclerotic lesions at the origin of the LAD form opposite the circumflex takeoff, rather than in the high-velocity high-shear bifurcation flow divider(FD) region. To determine whether this atheroma location occurs in vivo and is maintained across a range of disease severity, we analyzed high quality intracoronary ultrasound (IVUS) studies from 22 consecutive pts with disease somewhere in the LAD demonstrated by angiography. Atherosclerotic lesions at the LAD origin were present in all pts by IVUS and ranged from 30% to 78% stenosis. Percent area stenosis (%S) and maximum (MXT), minimum (MNT) and flow divider (FDT) intimal-medial thicknesses of the LAD were measured immediately after the takeoff of the circumflex. The angle (A) formed by the midpoint of the FD, the lumen center of gravity and the MXT was determined so as to position the lesion relative to the FD.

Results:

	%S	FDT (mm)	MNT (mm)	MXT(mm)	A
Mean ±SD	51±14	0.25±0.16	0.15±0.08	1.52±0.51	175±48°

MXT spared the arterial wall segment containing the FD in 100% of cases. Measurements of A indicated that maximal lesion size was located opposite the center of the FD segment. Lesion circumferential location and eccentricity (expressed as the MXT/MNT ratio) were unrelated to absolute plaque area or %S. Thus, we observed that atherosclerotic lesions at the origin of the LAD are located opposite the circumflex takeoff, spare the flow divider, and maintain this asymmetry across a wide range of lesion severity. These in-vivo