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QUANTITATIVE EXERCISE ECHOCARDIOGRAPHY IN NORMALS: TEMPORAL CHANGES IN REGIONAL WALL THICKENING AFTER EXERCISE

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Treadmill exercise produces qualitatively normal or hyperkinetic wall motion in regions of normal myocardium. However, there are few data or quantitative criteria for normal wall thickening after exercise. Our purpose was to measure normal wall thickening after exercise with echo and to determine the temporal changes in wall thickening during the immediate post-exercise period. Eight subjects (age 29±4y) underwent an exercise echo treadmill test (Bruce protocol). Rate pressure product (RPP) increased from 8,989 to 29,351 at peak exercise. End-diastolic (ED) and end-systolic (ES) frames were digitized (512 x 240 pixel resolution) on a Nova Microsonics digital work station. Wall thickness (cm) was measured in ED and ES from parasternal long axis (4 regions) and short axis (6 regions) views at baseline (pre) and during 2 minute intervals after exercise. Percent systolic wall thickening (%Th) was calculated. Results (mean ± SD, [Range]):

	Pre	0-2 mins	2-4 mins	5-7 mins
ES	1.36±0.24 [0.92-1.86]	1.64±0.29* [1.09-2.18]	1.48±0.29 [0.97-1.99]	1.36±0.26 [0.90-1.91]
%Th	56±20 [15-86]	85±25* [45-135]	68±25 [24-110]	57±25 [10-112]
RPP	8,989	20,134*	15,327	11,824

*p<0.05 vs. Pre and 5-7 mins (ANOVA)

Conclusion: Maximal wall thickening occurs within 2 minutes of peak exercise and returns to normal after 5 minutes. The lower end of the normal ranges (end-systolic thickness greater than 1.1 cm and/or percent systolic thickening greater than 45%) may be useful in determining a normal regional wall thickening response to exercise.

EARLY ASSESSMENT OF CORONARY ARTERY BYPASS GRAFT PATENCY BY HIGH DOSE DIPYRIDAMOLE ECHOCARDIOGRAPHY

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To assess the role of high dose (up to 0.84 mg/kg in 10') dipyridamole echocardiography test (DET) in the evaluation of coronary artery bypass graft (CABG) patency early after surgery 18 consecutive pts with angina underwent DET and coronary angiography (CA) before CABG and 10 days afterward. All pts had multivessel disease at CA. A total of 53 grafts were performed. Before CABG 14 pts had a positive and 4 a negative DET. No complication occurred during the test performed early after CABG. Of the 14 pts with positive pre-surgery DET 10 had negative and 4 positive post-CABG DET. All 4 pts with negative pre-surgery DET had a negative test after CABG. In the 4 pts with positive DET, before and after CABG, dipyridamole time (i.e the time from the onset of dipyridamole infusion to the development of frank asynergy) increased from 5.8±5 to 9.3±0.9 after the procedure and wall motion score index at peak dipyridamole improved from 1.55±0.2 to 1.28±0.3. Forty-nine of 53 grafts were patent by CA. DET was positive in 4 of 5 pts who had at least 1 obstructed graft or native vessel obstructed distal to graft insertion. DET was negative in all the 13 pts who had complete revascularization. In the 4 pts with positive DET, the test correctly identified the localization of the diseased bypass graft. These data suggest that: 1) DET can be easily and safely performed after CABG; 2) it reliably detects and identifies diseased bypass grafts.

EFFECTS OF A PATENT INFARCT-RELATED CORONARY ARTERY ON LEFT VENTRICULAR MORPHOLOGY AND REGIONAL FUNCTION

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Although thrombolytic therapy benefits clinical outcome after acute myocardial infarction, the relationship between vessel patency and post-infarct LV remodeling is less understood. Two dimensional echocardiography was performed within 48 hrs and 3 months in 30 pts treated with intravenous thrombolysis for their first myocardial infarction. Using a previously validated method the 3 dimensional LV total endocardial surface area (ESA) and the extent of abnormal wall motion (%AWM) were quantitated. Coronary angiography performed at a mean of 5 days after thrombolytic therapy was used to divide pts into those with a patent (Group A, n=20), or those with an occluded (Group B, n=10) infarct-related artery. ESA index (ESA_i=ESA/BSA;cm^2/m^2), %AWM, and Δ%AWM (mean±SD) were compared between and within the groups.

Results:

	Group A		Group B
ESA_i(entry)	60±15		52±7
ESA_i(3 mos)	62±13	p<0.02	62±10
%AWM(entry)	28±15		21±11
%AWM(3 mos)	18±16	p<0.005	24±17
Δ%AWM	-9±13	p<0.03	+2±12

Conclusions: A patent infarct-related artery after reperfusion is associated with improvement in regional LV wall function and attentuates LV dilatation.

THE SAFETY OF HIGH DOSE INTRAVENOUS DIPYRIDAMOLE-ECHOCARDIOGRAPHY TEST

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Clinical data on 6077 high dose (up to 0.84 mg/Kg over 10') dipyridamole-echocardiography test (DET) performed on 4810 pts were prospectively collected from 31 echocardiographic laboratories. All pts were studied for known or suspected coronary artery disease; 1181 early (<18 days) after an acute myocardial infarction; 223 had unstable angina. The dipyridamole infusion protocol was: 0.56 mg/Kg over 4' (low dose), followed by 4' of no dose and, if still negative for echocardiographically assessed myocardial ischemia, 0.28 mg/Kg over 2'. Major adverse reactions occurred in 5 cases (0.08%). There were 3 cases of severe bradycardia progressing to cardiac asystole (one of them was aminophylline resistant, prolonged and complicated by an acute myocardial infarction and coma, which evolved to death after 23 days), 1 pulmonary edema, 1 sustained ventricular tachycardia. In other 20 DET studies the high dose was not given in spite of the echocardiographic negativity, for limiting side effects due to the lower dose: hypotension and bradycardia (13 pts), excessive tachycardia (2), headache (2), restlessness (1), vomiting (1), acute bronchospasm (1). In all cases, side effects promptly subsided after aminophylline.

Thus, high dose DET is reasonably safe and well tolerated, even early after an acute myocardial infarction and in pts with unstable angina, when selectively applied in those pts in whom the lower dose did not induce echocardiographic signs of ischemia or limiting side effects.