HYPOTENSION DURING DOBUTAMINE STRESS ECHOCARDIOGRAPHY:SIGNIFICANCE OF THE ABSENCE OF REGIONAL HYPOKINESIS

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Hypotension (hypo) during exercise treadmill testing is correlated with coronary artery disease (CAD). Hypo induced during dobutamine echocardiography (DE) has not previously been reported. This report is the first to describe and correlate hypo during DE with CAD. We defined hypo response to DE as a >10mmHg fall in systolic blood pressure (BP) during dobutamine infusion. Hypo occurred in 17 of 67 consecutive pts who underwent DE (25%). The mean fall in BP was 25mmHg±15mmHg (range 15-54mmHg). All episodes resolved after stopping the infusion. Four tests were terminated because of hypo (mean peak infusion 25± 8mcg/kg/min). Five of 17 pts had no associated regional wall motion abnormalities (RWMA) or EKG changes. These pts were followed for a mean of 8.4 months and had no ischemic events. Twelve pts had caths; 11 pts had RWMA associated with hypo and also had significant CAD. A single pt with hypo despite absence of RWMA had cath and was shown to have no evidence for CAD.

Conclusion:
Hypo during DE in the absence of RWMA does not signify CAD or an adverse prognosis.

EFFECTS OF THEOPHYLLINE UPON THE ONSET AND DEGREE OF ISCHEMIA DURING EXERCISE THALLIUM-201 IMAGING.

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Theophylline (Theo), as a methylxanthine, counteracts the coronary vasodilatory effects of adenosine and may reduce exertional ischemia. However, the impact of Theo upon thallium-201 (TL) imaging following exercise has not been assessed. Nine patients with known ischemic coronary artery disease and angina pectoris participated in a double-blind, placebo-controlled crossover study using a Theo infusion (5 mg/kg; peak concentration 8.2 mg/L) prior to exercise thallium-201 imaging. Parameters measured included time to onset of ST depression, exercise duration (Dur) and TL defects measured semi-quantitatively (SQ) using segmental analysis and with computerized quantitation (CQ). Both utilized a thallium defect score based upon the area of ischemia. Values are expressed as mean ± S.D.

			Thallium Defect Score	
	ST(min)	Dur(min)	SQ	CQ
Placebo	4.1 ± 1.6	7.9 ± 1.6	8 ± 5	51 ± 29
Theo	5.7 ± 2.4	8.8 ± 1.6	9 ± 4	46 ± 26
P value	0.05	0.05	NS	NS

Following Theo infusion, time to ST depression and exercise duration were both significantly increased. However, there was no significant difference in the thallium defect score by either the visual observation or computerized analysis.
CONCLUSION: Theophylline successfully delays the onset of ischemia but does not affect the degree of ischemia produced by exercise.

RECOVERY-PHASE PATTERNS OF ST SEGMENT DEPRESSION IN THE HEART RATE DOMAIN CANNOT DISTINGUISH BETWEEN ANGINA PATIENTS WITH AND WITHOUT CORONARY ARTERY DISEASE

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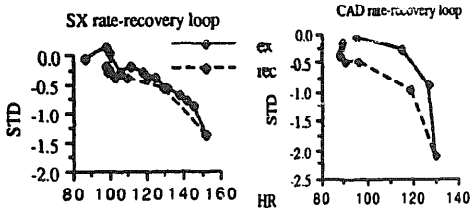
It has been suggested that patterns of recovery in HR after exercise can distinguish coronary artery disease (CAD) from normal subjects. To assess whether heart rate-recovery loops can differentiate CAD from syndrome X (SX) patients (pts) (typical angina, positive exercise test, normal coronary arteries and no coronary spasm), we studied the rate-recovery loops in 30 pts with SX and 75 pts with stable angina and CAD(>50% stenoses in≥1 coronary arteries). Loops were obtained by continuous plotting of ST segment depression (STD) and heart rate (HR) every minute throughout the treadmill exercise test and during 5 minutes of recovery. On average, STD was greater in the early recovery phase than at corresponding exercise HR in both SX and CAD pts (clockwise rate recovery loop). However, STD resolved more quickly related to HR recovery in SX pts compared to CAD (figure). In 68 of 75 CAD pts (90%) rotation was clockwise, whereas in SX pts rotation was clockwise in 12 (40%), anticlockwise in 9 (30%) and intermediate in 9 (30%)



Thus, heart-rate recovery loops do not appear to be able to distinguish individual patients with angina and SX or CAD.

Tuesday, March 5, 1991
Poster Displayed: 2:00PM–5:00PM
Author Present: 3:00PM–4:00PM
Hall F, West Concourse
Coronary Artery Disease: Basic Aspects

IDENTIFICATION OF A SPECIFIC MOLECULAR MARKER FOR ATHEROSCLEROTIC PLAQUE: A TIME COURSE STUDY IN THE RABBIT MODEL

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A specific molecular marker for atherosclerotic plaque, which we have named Z2D3, has been isolated from advanced human lesions and those of monkeys, pigs, and rabbits. According to molecular characterization, the Z2D3 marker appears to be a small, lipid containing molecule. It is probably not a sphingolipid because of its resistance to the usual acid hydrolysis conditions but is perhaps a neutral lipid or protolipid. A study was performed on New Zealand White Male Rabbits subjected to aortic ballooning and fed a high fat diet (2% cholesterol and 6% peanut oil). Aortic tissue samples from these animals were evaluated to determine both appearance of plaque and the origin of the molecular markers for Z2D3. The study showed that within the time sequence corresponding to the migration of both macrophages and lymphocytes to the early lesion, the Z2D3 antibody stained the cytoplasm and immediate pericellular regions of the medial smooth muscle cells located immediately beneath the elastic lamina of those areas of the artery wall that were thus involved. Slightly later in time, the smooth muscle cells were seen to penetrate the elastic lamina and migrate into the fatty streak area. Overall, the vast portion of staining within atherosclerotic plaque was found to be extracellular, diffusely manifest throughout the connective tissue matrix in addition to staining the cytosol of smooth muscle cells. The histologic assessment of the rabbit atherosclerotic lesions indicates that the Z2D3 molecule may be an important marker for studying the progression of atherosclerosis, as well as a specific target for in-vivo localization of imaging and/or therapeutic reagents.