

DIPYRIDAMOLE ELECTROCARDIOGRAPHIC TEST CAN DETECT SEVERE CORONARY ARTERY STENOSIS OF 90% IN THE LUMINAL DIAMETER

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The aim of this study was to investigate the relationship between the severity of coronary artery stenoses and the dipyridamole-induced ST depression. The subjects were 100 patients without myocardial infarction who underwent coronary arteriography for chest pain. They were divided into 4 subgroups by the maximal percent stenosis in the luminal diameter of the coronary arteries (%stenosis); 0-49%, 50-69%, 70-89%, and 90-100%. Body surface ECG mapping of 87 leads was performed both in the dipyridamole injection test (D) and in the symptom-limited treadmill exercise test (T). ST depression ≥ 0.1 mV was considered significant. The incidence of ST depression were;

%stenosis	0-49%	50-69%	70-89%	90-100%	total
D (%)	5**	13*	25*	84	49**
T (%)	47	47	63	92	72

(** p<0.01 vs. T, * p<0.05 vs. T)

In patients with coronary artery stenosis $\geq 90\%$, D had as high incidence of ST depression as T; while in patients with less severe stenosis, D had lower incidence than T. For the diagnosis of severe coronary stenosis $\geq 90\%$, D had as high sensitivity (84% vs. 92%), higher specificity (86% vs. 52%, p<0.01) and higher predictive accuracy (85% vs. 70%, p<0.05) compared to T. Dipyridamole-induced ST depression is thought to be a sensitive and specific marker of severe coronary artery stenosis.

PREDICTIVE VALUE OF ISCHEMIA AFTER MYOCARDIAL INFARCTION: HOLTER MONITORING VERSUS EXERCISE TESTING

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The presence of myocardial ischemia (IS) during Holter monitoring (HM) and exercise testing (EX) is considered to be of prognostic significance in post-MI-patients. To investigate the relation between HM and EX we prospectively studied 24h-HM and submaximal EX 15 days after acute MI in 135 patients. Criteria for IS in HM were ST-depressions > 1 mV > 1 min duration, in EX were ST-depressions > 1 mV in > 2 precordial leads. The patients were divided into 4 groups based on the presence of IS. The groups were comparable with regard to age, sex, MI-localisation, extent of vessel disease and medication. Follow-up was 9 ± 3 months.

Results:

	Group A EX+/HM+	Group B EX+/HM-	Group C EX-/HM+	Group D EX-/HM-
IS:				
pts (n)	16	26	16	77
death	3	2	0	4
ischemic event	12(75%)	9(35%)	n.s. 3(19%)	n.s. 24(31%)

ischemic event = cardiac/sudden death, recurrent MI, unstable or new onset of angina with or without CABG or PTCA; * = p<0.05; ** = p<0.01

Conclusion:

Ischemia on HM or EX alone is not a sensitive indicator of worse prognosis after acute MI. However ischemia detected by both methods identifies a group of patients with unfavorable clinical outcomes.

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Poster Displayed: 2:00PM-5:00PM

Author Present: 3:00PM-4:00PM

Hall F, West Concourse

Echocardiography

CUMULATIVE LEFT VENTRICULAR DYSFUNCTION MAY OCCUR DURING CORONARY ANGIOPLASTY

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To evaluate during PTCA the effects of balloon inflation (BI) before control wall motion (CWM) has restored following the previous one, we studied 11 anesthetized pts with normal LV function and proximal left anterior descending stenoses, using transesophageal echo. All pts underwent at least 3 consecutive BI's. In 5 pts (group A) serial BI's were performed after CWM had restored and in 6 pts before CWM had restored (group B). Parameters evaluated at the level of the papillary muscles, just before last deflation: LV end-diastolic area (EDA), end-diastolic endocardial length (EDEL) of anterior segment (i.e. between papillary muscles), and ejection fraction of ischemic region (REF); also time of CWM recurrence after last deflation was assessed.

Results **	GROUP A	GROUP B
EDA (cm ²)	18±3	* 23± 4
EDEL (cm)	10.4±1.9	* 12.9± 1.8
REF (%)	9±8	* 4±11
time-CWM(sec)	21±5	* 36± 7

*p<0.01; ** mean ±SD

Conclusion; LV geometry and recovery are particularly affected when serial BI's are performed before CWM has restored.

DYSFUNCTION OF MORE THAN 40% OF LEFT VENTRICULAR MYOCARDIUM IS REQUIRED TO CAUSE CARDIOGENIC SHOCK
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Previous studies have suggested that myocardial infarction (MI) of 40% or more of the LV can result in cardiogenic shock (CS). However these results were obtained using a clinical diagnosis of CS and autopsy determination of the extent of MI. Whether the same conclusions apply using a more precise hemodynamic definition of CS and *in vivo* determinants of the extent of myocardial dysfunction (MD) is uncertain. To address this issue, we employed a previously validated echo method of quantitating the extent of MD in 27 pts with isolated LV MI and with Swan-Ganz catheters. Pts were classified according to Forrester hemodynamic subsets (Class I= CI>2.2, PCW<18; Class II= CI>2.2, PCW>18, Class III= CI<2.2, PCW<18.8; Class IV(CS)= CI<2.2, PCW>18). The echo MD mapping technique employs data from apical 2 chamber, apical 4 chamber and 3 parasternal short axis views to display and quantitate the total endocardial surface of the LV with regions of MD superimposed. The extent of MD can be expressed in absolute terms or as a percent of the total endocardial surface area.

RESULTS:

Class	I	II	III	IV
XMD(mean±sd)	44±12	65±29	64±16	79±9

No pt with CS had less than 60% MD (p <.001 vs Class I). While pts in CS tended to have larger MI than Classes II and III, there was overlap. Of multiple parameters evaluated, only the presence of more than 1 MI separated CS pts from those in classes II and III (p<.02).
CONCLUSION: When a precise hemodynamic definition is used, all pts with CS have more than 60% MD.