

STRESS ECHOCARDIOGRAPHY *Chairman: Petros Nihoyannopoulos***VALIDATION OF DOBUTAMINE STRESS DIGITAL ECHOCARDIOGRAPHY DURING CARDIAC CATHETERISATION.**

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The diagnostic accuracy of dobutamine stress echocardiography using digital analysis (DSDE) was determined in a series of 56 male patients, mean age (\pm SD) 55.7 \pm 10.8y., at the time of routine diagnostic coronary arteriography (CA). β -blockers were discontinued for >72 hours prior to the study. Immediately after CA, right heart catheterisation was performed and dobutamine was infused intra-venously in incremental doses of 5-40 μ g/kg/min in eight stages of four minutes duration while monitoring haemodynamic changes. At the end of each stage on-line digital acquisition was used to record four standard 2D echocardiographic images. A 12-lead electrocardiogram (ECG) was recorded at two minute intervals and serial left ventriculography (LVG) was performed at baseline, mid-point and end-point infusion of dobutamine. All the patients had a significant increase in rate pressure product. End-points included new or worsening regional wall motion abnormalities, ST depression \geq 3mm, or significant angina. All patients had a modified Bruce protocol treadmill test performed for comparison (ETT). The diagnostic accuracy of the ECG, LVG, DSDE and ETT were compared with the results of CA. Coronary artery disease (>50% stenosis) was present in 46 patients (82.1%).

	ECG	LVG	DSDE	ETT
Sensitivity	32.6% (15/46)	67.4% (31/46)	82.6% (38/46)	54.3% (25/46)
Specificity	90% (9/10)	90% (9/10)	90% (9/10)	60% (6/10)
Predictive accuracy	93.7% (15/16)	96.9% (31/32)	97.4% (38/39)	86.2% (25/29)

In conclusion (i) the overall diagnostic accuracy of DSDE was greater than any of the other methods investigated, (ii) electrocardiography is insensitive for the detection of ischaemia during dobutamine infusion and (iii) LVG was more sensitive and specific than ETT but not as sensitive as dobutamine stress echocardiography.

Haemodynamic Changes During Dobutamine Stress Echocardiography: Implications for the detection of viable myocardium

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Introduction: The use of dobutamine stress echocardiography to identify myocardial ischaemia and viability relies on its inotropic and chronotropic actions. The detection of viability is thought to be complete at low doses of infusion (10 mcg/kg/min). However, there is surprisingly little data on the haemodynamics of routine dobutamine stress echocardiography.

Method: We therefore studied 61 patients referred for investigation of ischaemic heart disease with graded dobutamine stress echocardiography, dose range 5 to 40 mcg/kg/min rising in 3 minute stages. Their mean age was 58 yrs, (6 female, 55 male). Forty five per cent of patients had a resting wall motion abnormality. Stroke volume (SV) was calculated using the product of the left ventricular outflow tract cross sectional area and the systolic velocity integral of a continuous wave Doppler signal obtained from the apex.

Results: Mean cardiac output (CO) rose from 4.9 \pm 1.4 l/min at rest to 6.0 \pm 2.3 l/min at 10 mcg/kg/min (p <0.001) and to 9.2 \pm 2.2 l/min at peak. The early rise was due mainly to an increase in SV from 68 \pm 19 ml at rest to 77 \pm 23 ml at 10 mcg/kg/min (p <0.001). The late rise in CO was due to heart rate which rose from 73 \pm 12 bpm at rest to only 78 \pm 17 bpm at 10 mcg/kg/min (p =0.06) but 120 \pm 23 bpm at peak (p <0.001). Stroke volume continued to rise throughout the study to 80 \pm 17 ml at peak with only 33% of patients attaining maximal SV by 10 mcg/kg/min and 68% by 20 mcg/kg/min. Furthermore, of 8 patients in whom viability was detected, 5 (62%) showed a response during the high dose phase (>20 mcg/kg/min) of the study.

Conclusion: We conclude that higher doses of dobutamine than previously reported may commonly be required to identify viable myocardium.

PRESENCE OF MYOCARDIAL VIABILITY DOES NOT PREDICT GLOBAL IMPROVEMENT IN LEFT VENTRICULAR FUNCTION IN CHRONIC ISCHAEMIC CARDIOMYOPATHY

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The identification of viable myocardium has important therapeutic and prognostic implications in patients (pts) with chronic ischaemic left ventricular (LV) dysfunction. Improvement in regional LV wall thickening after low-dose dobutamine (DOB) in pts with segmental akinesia or severe hypokinesia is indicative of myocardial viability. In 36 pts with chronic ischaemic LV dysfunction with LV ejection fraction (EF) <35% echocardiography/Doppler was performed before and after DOB infusion (5-10 mcg/kg/min). Semiquantitative segmental LV wall motion analysis was performed by a panel of 3 from dynamic image display. Improvement in regional wall motion by at least one grade (score: 0-5 in ascending scale) in 2 or more segments was considered evidence of viability, whereas an increment in stroke volume (SV) by 10% was considered as improvement in global LV function. In 27 out of 36 patients (75%) there was evidence of myocardial viability and none showed deterioration of wall motion with DOB. Improvement in wall motion score correlated with the increment in SV ($r=0.77$; $p<0.0001$). However, in the 27 pts with improvement in wall motion SV did not improve in 5 (19%). Assessment of myocardial viability in chronic ischaemic cardiomyopathy should be combined with global LV function assessment, since this combination may predict those likely to improve after successful revascularisation.

MYOCARDIAL ISCHAEMIA DURING DOBUTAMINE STRESS ECHOCARDIOGRAPHY INDUCES 'DYNAMIC' MITRAL REGURGITATION.

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Patients with coronary artery disease may be limited by breathlessness rather than chest pain, usually due to a transient decrease in left ventricular function secondary to ischaemia. Mitral regurgitation (MR) may contribute to the exercise induced breathlessness experienced by these patients. We tested the hypothesis that patients may develop transient or 'dynamic' MR during acute myocardial ischaemia. Dobutamine stress transoesophageal echocardiography was performed in 13 patients (12 men, mean age 61 years) with angiographically proven coronary artery disease and morphologically normal mitral valves. MR was graded 0-4 by jet area/left atrial area ratios. Dobutamine (5 μ g/kg/min increasing by 5 μ g/kg/min every 5 minutes) was administered up to 25 μ g/kg/min or until ischaemia developed (new LV dyskinesia or significant ST segment changes) or until the patient was limited by symptoms.

In 6 patients (46%), MR increased by at least 1 grade; all developed a new area of LV dyskinesia (5 septal, 1 anterior). In 3 (23%), MR improved despite development of a new area of dyskinesia; this was associated with improved overall LV function. In 4, MR was unchanged; 3 of these had a new area of dyskinesia.

Dynamic mitral regurgitation may be induced by acute ischaemia, but is not seen when LV volume is reduced by the intervention. In this study MR was most commonly associated with septal dyskinesia.

TRANSMITRAL FLOW PATTERN DURING SHORT EXERCISE: A PREDICTOR OF VENTRICULAR CONTRACTILE RESERVE FOLLOWING MYOCARDIAL INFARCTION

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To examine the effects of short steady-state exercise on Doppler indices of left ventricular (LV) function, we studied 20 patients (pts) (age 62 ± 7 years) with an old myocardial infarction (MI) and 20 age-matched normal control subjects. All pts had no post-MI angina and also had a negative treadmill exercise test. Pts and controls were exercised on a supine bicycle ergometer for 3 min at a workload of 60 Watts. Heart rate and arterial pressure monitoring and Doppler recordings of transmitral and LV outflow tract velocities were made at baseline and during bicycle exercise. Heart rate increased by $32 \pm 18\%$ in MI pts and by $37 \pm 23\%$ in controls ($p=ns$) with bicycle exercise. Systolic arterial pressure increased by $25 \pm 7\%$ in MI pts and $28 \pm 6\%$ in controls ($p=ns$). Peak early filling velocity (E) increased with supine exercise vs baseline both in MI pts (80 ± 18 vs 58 ± 20 cm/s, $p < 0.001$) and normals (79 ± 14 vs 59 ± 9 cm/s, $p < 0.001$). Peak late filling velocity (A) increased with exercise vs baseline in normals (62 ± 14 vs 40 ± 15 cm/s, $p < 0.001$) but did not change overall in MI pts (56 ± 18 vs 52 ± 12 cm/s, $p=ns$). E/A ratio decreased at peak exercise vs baseline in controls (1.3 ± 0.3 vs 1.6 ± 0.4 , $p < 0.01$) but increased in MI pts (1.7 ± 0.9 vs 1.3 ± 0.3 , $p < 0.01$). All controls and 11 (55%) MI pts increased A, however 9 (45%) MI pts decreased A at peak exercise compared with baseline. LV end-diastolic diameter (53 ± 4 vs 52 ± 7 mm, $p=ns$) and left atrial diameter (35 ± 4 vs 36 ± 4 mm, $p=ns$) were not different between pts who increased and pts who decreased A on exercise. Peak LV outflow tract velocity increased with exercise by an average of $22 \pm 20\%$ in MI pts and by $26 \pm 16\%$ in normals ($p=ns$). However, pts who increased A on exercise showed a greater increase in LV outflow velocity ($32 \pm 12\%$) compared to those who decreased A ($14 \pm 19\%$), ($p < 0.05$). In addition, maximal exercise capacity expressed by METS achieved during treadmill exercise was greater in pts who increased A (7.5 ± 3.7 METS) compared with those who decreased A (4.2 ± 2.3 METS) ($p < 0.05$) during bicycle exercise.

We conclude that following MI the ability to increase A during exercise is associated with better LV systolic performance and better exercise capacity. Doppler recording of the transmitral flow velocity profile during short steady-state supine exercise may be a method to assess LV contractile reserve following MI.

Colour Doppler Imaging of Skeletal Muscle and Myocardium after Dynamic Cardiomyoplasty

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Dynamic cardiomyoplasty involves wrapping latissimus dorsi muscle around the ventricles to act as a biomechanical cardiac assist device. After a period of incremental electrical training the muscle is stimulated to contract on alternate cardiac beats by a pulse burst initiated by a synchronisable myostimulator. It is important to optimise the muscle stimulation voltage and the synchronisation delay between the R wave and the stimulator burst to give maximum systolic assistance whilst preserving myostimulator lifespan. Optimum stimulation voltages and synchronisation delays vary between patients and, to date, no practical non-invasive method has been found to optimise settings for muscle contraction.

Colour Doppler tissue imaging is a new non-invasive ultrasound technique which uses color Doppler to interrogate individual velocities and accelerations within muscle groups. This technique has the ability to interrogate both cardiac and skeletal muscle. To determine whether this new technique might be of potential value in optimising graft function, we have examined the effect of different myostimulator settings in three patients following cardiomyoplasty. In each case, a series of cardiac cycles were stored on digital cine loop and systolic frames were examined in assist and non-assist beats. Velocities were encoded within the latissimus dorsi muscle and posterior wall of the left ventricle. Assist beats were shown to have higher intramural velocities in systole in the native posterior wall. The direction of motion was reversed in the anterior wall indicating cardiac displacement. A change in stimulation voltage as low as 0.5 volts made an appreciable difference to wall motion and pedicle velocity, and differences were clearly noted by altering the synchronisation delay. We conclude that colour Doppler tissue imaging may prove to be a valuable non-invasive technique in optimising latissimus dorsi function in patients after dynamic cardiomyoplasty.

DOES PAPILLARY MUSCLE ISCHAEMIA CAUSE 'DYNAMIC' MITRAL REGURGITATION?

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During acute ischaemia some patients with coronary artery disease develop transient or 'dynamic' mitral regurgitation (MR). This has been attributed to papillary muscle dysfunction. The posteromedial and anterolateral papillary muscles are supplied by the right and left circumflex coronary arteries respectively, so transient ischaemia in the territory of either artery may result in papillary muscle dysfunction and mitral regurgitation. To investigate whether regional ischaemia is important in the pathogenesis of dynamic MR, we studied patients during coronary angioplasty, as this offers an excellent controlled model of acute ischaemia.

37 men (mean age 58) were studied by transthoracic echocardiography before and during elective coronary angioplasty. With the patient lying supine, imaging was performed and the left atrium was interrogated using colour flow mapping. The optimal window was identified and marked on the patient's chest, then used throughout the study. The ratio of the area of the mitral regurgitant jet (MRJ) to the area of the left atrium (LA) was determined by off-line computerised planimetry. Annular diameter and the distance of the mitral leaflet coaptation point to the annular plane were also measured.

During angioplasty 26 patients developed ischaemia (ST segment depression and/or new regional wall motion abnormality). 16 of these (62%) developed dynamic MR. The average MRJ/LA ratio increased from $5.6\% \pm 5.2$ (mean \pm SD) to $20.7\% \pm 7.0$ ($p < 0.001$). 64% of patients undergoing angioplasty to the LAD developed dynamic MR, whereas only 33% of patients with RCA lesions and 20% of LCX lesions had increased regurgitation. 11 (30%) of patients had no ischaemia; none developed dynamic MR. The distance between the mitral leaflet coaptation point (MLCP) and the annular plane increased with ischaemia from $0.57\text{cm} \pm 0.2$ to 0.7 ± 0.3 ($p < 0.01$). There was no change in annular diameter before ($2.95\text{cm} \pm 0.6$) and during ($2.92\text{cm} \pm 0.9$) ischaemia.

These data suggest that dynamic mitral regurgitation is common during acute ischaemia. It occurs more frequently with LAD territory ischaemia than with RCA or LCX, and it is associated with apical displacement of the coaptation point. Mechanisms other than papillary muscle dysfunction are probably responsible for the development of dynamic MR.