

913-78 Influence of Coronary Microvascular Dysfunction on Left Ventricular Contractile Response to Catecholamine in Dilated Cardiomyopathy

Makoto Onishi, Tomohiro Kondo, Masato Morita, Tohru Arii, Nagao Yasutomi, Tadaaki Iwasaki. *Hyogo College of Medicine, Nishinomiya, Japan*

We attempted to clarify the influence of coronary microvascular dysfunction on left ventricular contractile response to dobutamine (DOB) administration in 12 patients with dilated cardiomyopathy (DCM) using DOB stress 201 thallium-SPECT (DOB-Tl) and myocardial contrast echocardiography (MCE). Methods: 1) The echocardiographic assessment of left ventricular contractility was done by centerline method before and after DOB(10 μ) administration at mid-papillary muscle level. 2) MCE: The sonicated 5% human serum albumin (2 ml) was injected into left coronary artery, and the background subtracted peak intensity (Δ PI) and the washout half-time (T1/2) of contrast enhancement were measured at the same level. 3) DOB-Tl (DOB: 10 μ): Tl uptake was scored from 0 (defect) to 3 (normal). Total Tl uptake score of left ventricle in which was divided into 9 segments was calculated before and after DOB infusion. Results: 1) The contractility responded normally to DOB in 7 of 12 patients (Group 1). The remaining 5 patients (Group 2) showed impairment of response to DOB. However, no significant difference was found between the 2 groups in left ventricular contractility before DOB infusion. 2) MCE: The higher Δ PI and the shorter T1/2 were observed in Group 1 compared with those in Group 2 ($p < 0.01$). 3) Tl uptake score immediately after DOB stress was significantly lower in Group 2 than in Group 1 (22 ± 4 vs. 16 ± 3 , $p < 0.01$). Tl uptake score did not differ between the 2 groups before DOB infusion.

Conclusion: These results suggest that myocardial ischemia based on microvascular dysfunction is involved in reduced response to catecholamine in DCM.

913-79 The Relation Between Thallium-201 Uptake and Contractile Reserve Elicited With Isolated or Combined Adrenergic and Adenosinergic Stimulation in Viable Myocardium

Albert Varga, Miodrag Ostojic, Rosa Sicari, Alessandro Pingitore, Alessia Gimelli, Ana Djordjevic-Dikic, Ivana Nedeljkovic, Marco Torres, Paolo Marzullo, Eugenio Picano. *CNR, Institute of Clinical Physiology, Pisa, Italy*

We have previously shown that infra-low (0.28 mg/Kg) dipyridamole (DIP) added to low dose (up to 10 mcg/Kg/min) (DOB) recruits an inotropic reserve in asynergic segments which were non-responder either after DOB or DIP alone and destined to recover following revascularization. In order to investigate the relationship between radiolabeled and echocardiographic markers of myocardial viability, 24 patients (mean age 60 ± 9 years) with previous myocardial infarction (> 3 months), angiographically assessed coronary artery disease (5 with 1-, 10 with 2- and 9 with 3-vessel disease) and resting dysfunction (mean ejection fraction $37 \pm 12\%$) underwent rest-redistribution planar 201-Thallium scintigraphy (Th), and low dose pharmacological stress echo with DOB, DIP and combined DIP and DOB (DIDO). Criteria for viability in a 13 segment model were: percentage peak activity < 55% for Th; decrease in wall motion score ≥ 1 grade (1 = normal to 4 = dyskinesic) for stress echo. A regional resting dyssynergy was observed in 167 segments; of these, 128 were viable by Th (78%) 93 by DOB (56%; $p < 0.001$ vs Th), 85 by DIP (51%; $p < 0.001$ vs Th), and 120 by DIDO (72%, $p = ns$ vs Th). The rate of agreement between Th and stress echo was 71% for DIP, 73% for DOB and 86% for DIDO ($p < 0.001$ vs DIP and vs DOB). In conclusion DIDO recruits an inotropic reserve in a significant proportion of segments with resting dysfunction which were non-responders either after DIP or DOB alone and preserved Th uptake.

913-80 Akinesis Becoming Dyskinesia at High-Dose Dobutamine Stress Echocardiography: A Marker of Poor Functional Recovery After Myocardial Revascularization

Abdou Elhendy, Jan H. Cornel, Jos R.T.C. Roelandt, Ron T. van Domburg, Galal M. El-Said, Mohammed M. Ibrahim, Paolo M. Fioretti. *Thoraxcenter, Rotterdam, The Netherlands*

Akinesis becoming dyskinesia at high dose dobutamine has been disregarded as a marker of myocardial ischemia. However, the relation between this pattern and myocardial viability has not been studied. 42 patients with old myocardial infarction underwent dobutamine stress echocardiography (up to 40 μ g/kg/min) before coronary artery bypass surgery, and resting echocardiogram 3 months after surgery. Viability in aknetic segments was considered if systolic thickening occurred at low-dose dobutamine (LDD). During high dose dobutamine, dyskinesia occurred in 35 of the 164 aknetic segments

(group A). The remaining 129 segments comprised group B. Segments of group B had a higher prevalence of viability pattern at LDD (18% vs 0%, $p < 0.01$) and functional improvement (20% vs 0%, $p < 0.005$) compared to group A. In absence of viability pattern at LDD, postoperative improvement occurred in 10% of segments in group B and in none in group A, resulting in a higher negative predictive value of LDD in group A versus B (100% vs 90%, $p < 0.05$).

Conclusion: Akinesis becoming dyskinesia at high dose dobutamine echocardiography is associated with poor functional outcome after revascularization. Observation of this pattern provides additional data to those obtained at LDD and improves the value of dobutamine echocardiography for prediction of functional improvement of aknetic segments.

913-81 Low Dose Dobutamine Stress Echocardiography Can Predict Improvement in Left Ventricular Ejection Fraction After Revascularization

David A. Cusick, Gorav Ailawadi, James W. Frederickson, Michael J. Vonesh, Robert O. Bonaw, Farooq A. Chaudhry. *Northwestern University, Chicago, IL*

Low dose dobutamine stress echocardiography (DSE) can identify potentially viable myocardium in pts with coronary artery disease and LV dysfunction. In such pts, LV dysfunction may improve after coronary artery bypass surgery (CABG). However, whether this translates into meaningful increase in overall LV ejection fraction (LVEF), and whether postop LVEF can be predicted, has not been demonstrated. We therefore studied 15 consecutive pts (mean age 63 yrs; range 47-81; 13 males, 2 females) with coronary artery disease and LV dysfunction who underwent DSE prior to CABG. LVEF was measured using a biplane area-length method at baseline, during low dose DSE, and after CABG (mean days after CABG 86). Two pts died perioperatively and thus post CABG LVEF is not available.

Results:

No	Mean LVEFL(%)		
	Baseline	Low Dose DSE	Post CABG
13	32 \pm 5	45 \pm 7*	43 \pm 6*

There was no significant difference in mean LVEF between low dose DSE and post CABG. Low dose DSE correlated with post CABG LVEF ($r = 0.87$, $p < 0.001$), and the mean difference between low dose DSE and post CABG LVEF was -2.5 ± 3.5 . **Conclusion:** These data indicate that low dose DSE can predict not only the recovery of LV segmental function but also whether a clinically relevant change in global LVEF will occur following CABG.

913-82 The Timing of Dobutamine Echocardiography After Acute Myocardial Infarction Does Not Alter the Accuracy for Detecting Reversible Dysfunction

Steven C. Smart, Thomas Knickelbine, John Wynsen, Kiran B. Sagar. *Medical College of Wisconsin, Milwaukee, WI*

Regional myocardia function has been shown to change during the first week after acute myocardial infarction (MI). To investigate if the timing of dobutamine echocardiography (DE) affects accuracy for reversible dysfunction, 115 patients (age 57 ± 13 yrs, 17 women/98 men, 66 with Q-wave infarction, 64 anterior infarction, and 80 treated with thrombolysis) underwent low dose DE (rest, 5 and 10 mg/kg/min in 5-10 min stages) at 5 ± 2 days (range 2-7) after acute MI. Follow-up echocardiography was performed at 2 ± 1 months after infarction. All echocardiograms were analyzed according to the standard model and scoring system. Reversible dysfunction was defined as improved infarction zone wall thickening at follow-up and dobutamine responsive as improved thickening during low dose infusion. Infarction zone dysfunction reversed at follow-up in 65 (57%) patients and responded to low dose dobutamine in 66 (57%). The sensitivity and specificity of DE for reversible dysfunction were 91% (50/65) and 86% (43/50), respectively. The interval between MI and DE was 2 days in 16 patients, 3 days in 24, 4 days in 24, 5 days in 12, 6 days in 16, and 7 days in 23. Hemodynamics were similar in each subset. The number of patients with reversible and fixed dysfunction and the sensitivity and specificity of DE according to the interval were as follows:

Interval (days)	2	3	4	5	6	7
Reversible (n)	11	15	12	7	6	14
Fixed (n)	5	9	12	5	10	9
Sensitivity (%)	82	93	92	100	83	93
Specificity (%)	80	100	87	80	100	89

There were no differences in sensitivity and specificity related to the interval to DE. In conclusion, the timing of DE after acute MI does not affect its sensitivity or specificity for reversible dysfunction.