# Correlation of Echocardiographic Left Atrial Abnormality With Myocardial Ischemia During Myocardial Perfusion Assessment in the Presence of Known Left Ventricular Hypertrophy

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**Left atrial (LA) abnormality, an easily quantifiable parameter of left ventricular (LV) diastolic dysfunction, has been associated with cardiovascular risk similar to that of LV hypertrophy. The correlation between LV hypertrophy and LA abnormality among patients undergoing myocardial perfusion (MP) study has not been described. We prospectively studied 78 consecutive patients with LV hypertrophy who underwent MP study after screening for electrocardiographic and echocardiographic LA abnormality over a 6-month period. Of those, 48 had a positive MP imaging result, and 30 did not. LA size ($p = 0.002$) and P-wave duration ($p = 0.017$) were significantly increased in the former. The differential change in LA size (no defect = 35 ± 4, mild = 36 ± 5, moderate = 38 ± 5, severe = 44 ± 5 mm; $p < 0.0001$) and P-wave duration (no defect = 107 ± 14, mild = 110 ± 17, moderate = 113 ± 15, severe = 127 ± 22 ms; $p = 0.003$) was greatest when the MP study defect exceeded moderate severity. In conclusion, the presence of LA abnormality could assist during MP study interpretation among patients with LV hypertrophy when such markers appear to be correlated with the severity of the MP study defect.**  **(Am J Cardiol 2013;112:416−419)**

Myocardial perfusion (MP) single-photon emission computed tomography is an established cardiovascular modality used in the detection of myocardial ischemia or infarction. In the presence of its inherent limitations such as attenuation, scatter, poor spatial resolution, and depth-dependent loss of resolution, the presence of certain clinical cardiac risk factors such as left ventricular (LV) hypertrophy can sometimes introduce visual and quantitative challenges in assessing the underlying myocardial ischemia.[1−3] Left atrial (LA) abnormality, defined as widening of P wave ≥120 milliseconds on electrocardiographic tracing, an easily quantifiable morphophysiologic parameter of LV diastolic dysfunction, has also been strongly associated with increased risk of cardiovascular events because it often serves as an early marker for myocardial ischemia.[4−7] We thus studied the correlation of this cardiovascular marker with myocardial ischemia during MP study among a subset of patients with LV hypertrophy.

## Methods

We prospectively studied all consecutively hospitalized patients at Saint Vincent Hospital, Worcester, Massachusetts, over a 6-month period with previously identified LV hypertrophy on an electrocardiogram who underwent MP study. Patients were then screened for 2-dimensional transthoracic echocardiograms performed within 14 days (range 1 to 13 days; mean 4 days) of MP study. Because coronary artery disease and hypertension are highly prevalent correlates of LA abnormality, both otherwise unselected groups were matched for these co-morbidities. MP study reports were then assessed for reversible perfusion abnormalities consistent with myocardial ischemia. Reports with nonreversible defects suggesting a myocardial scar were excluded from this investigation. All patients were referred by either cardiologists or primary care physicians who were directly involved in their care for evaluation of myocardial ischemia. We also appraised common medical co-morbidities documented in patients' medical records, consistent with current classifications and guidelines for disease definition and diagnosis outlined by American College of Cardiology/American College of Physicians/American Society of Internal Medicine Task Force on Practice Guidelines.

Resting 12-lead electrocardiograms were recorded in supine position from a Marquette 2000 electrocardiograph (Marquette Electronics Inc., Milwaukee, Wisconsin) standardized at 25 mm/s and 10 mm/mV. Electrocardiograms were evaluated for previously identified LV hypertrophy ≥1 month (range 1 to 27 months; mean 20 months) to be considered for this study, using the Cornell gender-specific criteria: S wave in lead $V_3$ + R wave in lead aVL >28 mm in men and >20 mm in women.

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See page 418 for disclosure information.

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Table 1
Differences between patients with positive versus negative myocardial perfusion imaging studies

| Variable | MP Imaging Study | | |
|---|---|---|---|
| | Negative (n = 30) | Positive (n = 48) | p Value |
| Age, mean ± SD (yrs) | 69.4 ± 15.5 | 68.6 ± 10.4 | 0.792 |
| Women | 12 (40%) | 23 (48%) | 0.494 |
| Men | 18 (60%) | 25 (52%) | |
| LA enlargement | 4 (13%) | 19 (40%) | 0.013 |
| Diabetes mellitus | 7 (23%) | 16 (33%) | 0.346 |
| Hyperlipidemia | 8 (27%) | 26 (54%) | 0.017 |
| Atrial fibrillation | 3 (10%) | 6 (13%) | 0.737 |
| Valvular heart disease | 5 (17%) | 14 (29%) | 0.211 |
| COPD | 5 (17%) | 13 (27%) | 0.288 |
| Dilated cardiomyopathy | 8 (27%) | 16 (33%) | 0.535 |
| Beta adrenergic blockers | 18 (60%) | 39 (81%) | 0.040 |
| ACE inhibitors | 8 (27%) | 19 (40%) | 0.243 |
| Angiotensin receptor blockers | 7 (23%) | 11 (23%) | 0.966 |
| Statins | 8 (27%) | 22 (46%) | 0.091 |
| P-wave duration (ms) | 107 ± 14 | 117 ± 19 | 0.017 |
| LA size (mm) | 35 ± 4 | 40 ± 6 | 0.002 |
| LV ejection fraction (%) | 44 ± 12 | 43 ± 11 | 0.662 |
| Interventricular septal thickness (mm) | 10 ± 2 | 11 ± 2 | 0.014 |
| LV end-systolic volume (ml) | 42 ± 8 | 44 ± 9 | 0.290 |
| LV end-diastolic volume (ml) | 111 ± 16 | 115 ± 11 | 0.293 |
| MP imaging LV ejection fraction (%) | 44 ± 13 | 45 ± 12 | 0.959 |

ACE = angiotensin converting enzyme; COPD = Chronic obstructive pulmonary disease.

Table 2
P-wave duration and left atrial size versus degree of myocardial perfusion imaging study defect

| | Defect on MP Imaging Study Defect | | | | |
|---|---|---|---|---|---|
| | None (n = 30) | Mild (n = 17) | Moderate (n = 15) | Severe (n = 16) | p Value for Trend |
| P-wave duration (ms) | 107 ± 14 | 110 ± 17 | 113 ± 15 | 127 ± 22 | 0.003 |
| LA size (mm) | 35 ± 4 | 36 ± 5 | 38 ± 5 | 44 ± 5 | <0.0001 |

Two-dimensional transthoracic echocardiograms were performed on each patient in the left lateral recumbent position for parasternal long and short axes, apical 4- and 2-chamber views, and in the supine position for subcostal views using an ATI HDL 5000W imaging system with a P4-2 scan head and 2.6-MHz transducer (Phillips Medical Systems Company, Bothell, Washington). LA abnormality was considered when anteroposterior linear measurement from leading edge to leading edge during systolic frames exceeded 40 mm in men and 38 mm in women. Systolic frames were defined as the frame depicting the cardiac systolic event just before separation of the mitral valve tips. All echocardiograms were recorded and analyzed by experienced, board-certified echocardiographers who were blinded to both clinical presentation and electrocardiogram findings.

Each patient received an intravenous infusion of dipyridamole (0.56 mg/kg over 4 minutes) while undergoing continuous electrocardiographic monitoring under the supervision of board-certified cardiologist and qualified technicians per institutional protocols. Four and half minutes after completion of dipyridamole infusion, weight-based doses of $^{99m}$Tc-sestamibi were administered intravenously, and stress images were acquired 45 minutes later. Subsequently, another weight-based dose of $^{99m}$Tc-sestamibi was administered. Rest images were then obtained 45 minutes later. Single-photon emission computed tomography images were acquired using an ADAC scintillation gamma camera (ADAC Laboratories Inc./UGM Medical Systems Inc., Manchester, New Hampshire) with an all-purpose, high-resolution collimator by use of a step-and-shoot approach every 8° over a 180° clockwise circular orbit beginning at a right anterior oblique projection −39° and ending at left posterior projection 141°. Images were reconstructed using standard back projection algorithms (Butterworth filter, order 5, section frequency 0.4). Quantitative gating with Codonics R-wave electrocardiographic trigger gating (Codonics Inc., Middleburg Heights, Ohio) was also performed on stress images. Stress and rest images from the short axis, horizontal long axis, and vertical long axis slices were obtained according to the American Society of Nuclear Cardiology guidelines and were viewed by experienced board-certified readers. The myocardial radiopharmaceutical uptake was assessed by consensus for reversible perfusion abnormality as normal, mild defect, moderate defect, or severe defect in relation to the background uptake and quantified by size (large, medium, or small) when perfusion abnormalities were noted.

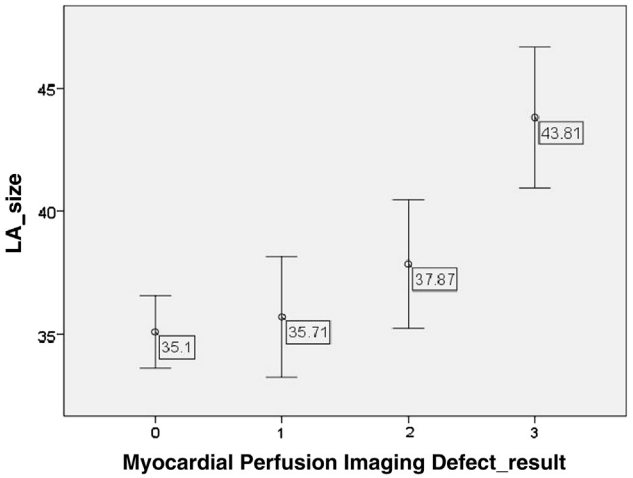


Figure 1. The differential change in left atrial size versus severity of MP imaging defect.

Data were expressed as mean ± SD for continuous variables and frequencies for categorical variables. Differences between groups were assessed using chi-square test for categorical variables and analysis of variance for continuous variables. A p value <0.05 was considered significant. Logistic multivariate regression analysis using significant variables was also performed. Statistical analyses were performed using SPSS Version 13.0 statistical software (SPSS Inc., Chicago, Illinois).

## Results

Seventy-eight consecutive patients qualified for the study. Clinical co-morbidities in the 2 groups based on MP