Influence of Left Ventricular Mass on the Diagnostic Accuracy of Myocardial Perfusion Imaging Using Positron Emission Tomography with Dipyridamole Stress

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This study assesses the influence of left ventricular hypertrophy (LVH) on the accuracy of myocardial perfusion imaging using pharmacologic coronary vasodilation. Seventy-five patients without previous infarction, and with known coronary anatomy, were studied by echocardiography and PET. LVH (defined by mass >131 g/m^2 in males or >100 g/m^2 in females) was identified in 25 patients; this group did not differ significantly from the remainder in terms of clinical or angiographic parameters. Twenty patients with hypertrophy had significant coronary artery stenoses, which were identified correctly by PET in 11 (55%), in contrast to 29 of 34 patients (85%, p = 0.03) with coronary disease but normal LV mass. Normal perfusion images were obtained in three of five patients (60%) with hypertrophy but no coronary disease; in contrast, 14 of 16 patients without either coronary disease or hypertrophy (88%, p = ns) had normal scans. The accuracy of PET was 14/25 (56%) in those with hypertrophy, and 43/50 (86%, p = 0.01) in patients with normal LV mass. In this group, the presence of hypertrophy was associated with reduction in the diagnostic accuracy of PET using dipyridamole stress. These findings may account for the phenomenon of "dipyridamole nonresponsiveness" in some patients.

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The identification of coronary artery disease in patients with left ventricular hypertrophy (LVH) is a frequent clinical problem. Such individuals frequently demonstrate resting abnormalities which compromise the accuracy of electrocardiographic stress testing (*1*), so myocardial perfusion imaging is often employed for the diagnosis of coronary artery disease. This investigation may be performed safely and effectively using intravenous dipyridamole (*2*), especially if individuals are unable to exercise (*3*), or if imaging is performed using ^{82}Rb PET (*4*). The underlying principle of this approach is that regions supplied by a flow-limiting coronary stenosis are characterized by less dipyridamole-induced enhancement of tracer uptake than are normal regions (*5*). However, the enhancement of coronary flow is also altered by LVH (*6,7*), which is characterized by reduction in the capacity of normally perfused myocardium to mount a coronary vasodilator response (*8*). The purpose of this study was to examine the influence of these phenomena on the accuracy of dipyridamole-stress perfusion imaging.

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MATERIALS AND METHODS

METHODS

Patient Selection

Individuals referred for ^{82}Rb PET with recent or planned coronary arteriography had two-dimensional echocardiography performed and were eligible for enrollment into the study. The decision to undergo catheterization was not contingent upon the perfusion imaging results, and most patients had PET performed after angiography. Echocardiography was generally arranged after a patient was identified as suitable and had undergone the other two tests. In addition, recent clinical echocardiograms of good quality were used because of difficulties in arranging an echocardiogram before intervention.

To avoid the detection of resting perfusion defects from influencing the sensitivity of myocardial perfusion imaging, patients with electrocardiographic or echocardiographic evidence of previous myocardial infarction were excluded. Individuals were not studied if they had asthma or unstable angina.

After the exclusion of 5 patients with technical inadequacies which precluded the interpretation of one or other technique, the study population consisted of 75 noninfarct patients with angiographic, perfusion, and echocardiographic data. The mean age was 62 ± 11 yr, and 26 (35%) were female.

Echocardiography

All patients had two-dimensional echocardiography performed in proximity to myocardial perfusion imaging without any intercurrent cardiac events (interval 2.7 ± 1.3 mo). Measurements of

ventricular dimensions were made from M-mode images recorded on paper at 50 mm/sec. In the situation of unfavorable cardiac orientation for satisfactory M-mode images, the two-dimensional images of the parasternal long-axis view of the left ventricle (recorded on ½" VHS video tape) were digitized and measured (DataVue, Nova Microsonics, Indianapolis, IN). Septal thickness (S), ventricular end-diastolic dimension (EDD), and left ventricular posterior wall (PW) thickness were measured just below the level of the mitral valve leaflets in accordance with the recommendations of the American Society of Echocardiography (*9*). Left ventricular mass was then calculated using the formula (*10*):

$$\text{Echocardiographic LV mass (g)} = 1.04\ \{(S + EDD + PW)^3 - EDD^3\}.$$

Left ventricular mass values consistent with necropsy data were obtained by application of the formula (*11*):

$$\text{True LV mass} = 0.8\ (\text{echocardiographic LV mass}) + 0.6.$$

LVH was then defined by LV mass ≥131 g/m^2 in males, and ≥100 g/m^2 in females. The study population was thus divided into subgroups of patients with and without LVH.

Coronary Arteriography

Arteriograms were performed using the Sones or Judkins techniques and recorded on cine film. Films were interpreted by a reviewer blinded to the echocardiographic and scintigraphic data, and stenosis severity was assessed with the assistance of digital calipers. Significant disease was defined by the presence of >50% diameter stenosis in a major epicardial vessel.

Dipyridamole-Stress Technique

A conventional dipyridamole-handgrip stress protocol was employed before stress-perfusion imaging (*12*). Patients were instructed to attend the laboratory in the fasting state, with abstention from caffeine for 12 hr, and after cessation of theophylline for at least a day. No changes were made to anti-anginal therapy. Dipyridamole was infused intravenously to a dose of 0.56 mg/kg over a 4-min period, and following 2-min delay, handgrip was performed at 25% of maximum grip strength for 4 min. Patients were monitored clinically and electrocardiographically throughout the stress period, and the hemodynamic and electrocardiographic responses to stress were analyzed for each sub-group.

PET

Myocardial perfusion was evaluated on a Posicam camera (Positron Corp., Houston, TX) following intravenous injection of 40–60 mCi ^{82}Rb from a generator (Cardiogen, Squibb Diagnostics, Princeton, NJ) using a previously described protocol (*13*). The imaging sequence commenced with a 20-min transmission scan performed with a ^{68}Ga filled ring, after which resting imaging was performed over 7 min, the patients underwent dipyridamole stress, and finally stress rubidium imaging.

Images were interpreted by observers blinded to the echocardiographic and angiographic data in accordance with previously published guidelines, with a stress-induced defect being defined by >20% reduction of counts with stress, and a resting defect defined by regional counts >20% below maximum (*13*). This interpretation was performed using a quantitative color scale and polar map display produced by the Posicam Data Acquisition System (PDAS) and VAX 11/750 computer.

Data Analysis

Abnormal coronary perfusion corresponding to a stenosis of greater than 50% in a major epicardial coronary artery was classified as true-positive finding, and the absence of a perfusion defect corresponding to a significant stenosis was defined as false-negative. In the absence of significant coronary stenosis, the presence of abnormal perfusion was classified as a false-positive and the presence of normal perfusion constituted a true-negative. Sensitivity, specificity, and accuracy were calculated in the usual fashion. An unpaired t-test was used to compare continuous variables in the groups with and without LVH and noncontinuous variables were compared using Fisher's exact test or a chi-square test, depending on sample sizes.

RESULTS

Left Ventricular Hypertrophy

Left ventricular mass was increased in 25 patients and was normal in the remaining 50. The clinical characteristics of these patients are described in Table 1. There were no significant differences in age, sex, body habitus or medical therapy between the groups. Nineteen of these patients (70%) with LVH had an identifiable underlying cause, including hypertension (n = 17) or valvular heart disease (n = 2); similarly, 25 patients (50%) of those with normal LV mass had hypertension (p = ns).

Electrocardiographic evidence of LVH was present in five of the patients with increased LV mass, comprising 28% of the patients in this group with electrocardiograms suitable for interpretation (the remaining seven patients having conduction disturbances or pacemakers). Such

TABLE 1
Clinical Features of Groups With and Without LVH

	LVH group	Non-LVH group	p
n	25	50	
Female	11 (44%)	15 (30%)	ns
Age (yr)	63 ± 10	61 ± 14	ns
Weight (kg)	79 ± 14	81 ± 12	ns
Hypertension, valve disease	17 (68%)	25 (50%)	ns
Anti-anginal therapy	19 (76%)	35 (70%)	ns
ECG evidence of LVH	5/18	3/44	0.04
LV septum (mm)	13 ± 2	11 ± 2	0.001
LV posterior wall (mm)	13 ± 2	11 ± 2	0.001
LV diastolic diameter (mm)	56 ± 9	45 ± 6	0.001
LV mass index (g/m^2)	162 ± 31	91 ± 15	0.001
Significant CAD	20 (80%)	34 (68%)	ns
Multivessel disease (%CAD)	11 (55%)	16 (47%)	ns
Severe (≥70%) stenoses (%CAD)	16 (80%)	25 (71%)	ns

CAD = coronary artery disease and ECG = electrocardiographic.

changes were only apparent in three patients in the group without LVH (p = 0.04). The mean LV mass in the group with hypertrophy was 162 ± 31 g/m^2, compared with 91 ± 15 g/m^2 in patients without hypertrophy. This reflected significantly higher values for both LV chamber dimensions and mean LV wall thickness in patients with LVH (Table 1).

Twenty of the 25 patients (80%) with LVH had significant coronary artery disease, compared with 34 of the 50 patients (68%, p = ns) without LVH. The prevalence of multivessel disease (11/20 versus 16/34) and the proportion of patients with severe (>70% diameter) stenoses (16/20 versus 25/34) were comparable in each group.

The hemodynamic effects of influence of dipyridamole-handgrip stress in patients with and without LVH are summarized in Table 2. Heart rate increased to 130% of that at rest and rate-pressure product to 140% in those with LVH, with equivalent changes in those with normal LV mass. Electrocardiographic features of stress-induced ST-segment changes were present in 3 of the 20 patients with coronary stenoses and LVH and in 9 of the 34 patients with coronary disease and normal LV mass (p = ns).

Myocardial Perfusion Imaging

The effects of LVH on the sensitivity, specificity, and accuracy of myocardial perfusion imaging are portrayed in Figure 1. Perfusion defects were present in 29 of 34 patients with coronary disease but without LVH (sensitivity 85%). Purely stress-induced defects were present in 21 patients, with the remainder showing partial defects at rest. In contrast, 9 patients with both coronary disease and LVH showed no perfusion defects by myocardial scintigraphy, so that perfusion imaging identified the presence of coronary artery disease in only 11 instances (55%, p = 0.03). Three of these were purely stress induced, the remainder having some impairment in local resting perfusion.

The features of the 14 patients with false-negative perfusion imaging findings are described in Table 3. Antianginal therapy was present in 11 (79%) compared with 29 of the 40 with correct perfusion imaging (73%, p = ns). Eight of the 14 patients with false-negative scans had severe (≥70%) stenoses (57%) compared with 33 of the 40 patients with true-positive scans (83%, p = ns). Multivessel disease was present in 7 of the 14 false-negative group (50%) and 20 of the 40 patients with true-positive scans. The mean increments of heart rate and blood pressure in patients with false-negative scans (1.3 ± 0.2 and 1.4 ± 0.2, respectively) were not significantly different from those with true-positive results (1.3 ± 0.2 and 1.5 ± 0.3).

Sixteen patients had neither coronary disease nor LVH and 14 of this group had normal perfusion imaging (specificity 88%). However, only three of five patients (60%) without coronary disease but with LVH had normal scans (Fig. 1). The diagnostic accuracy of myocardial perfusion imaging in patients without LVH was significantly higher (43/50) than in those with this problem (14/25, p = 0.01).

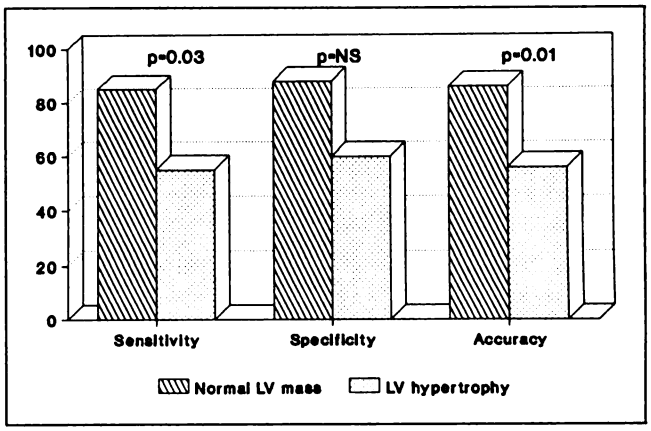

FIGURE 1. Sensitivity, specificity and accuracy of dipyridamole-stress ^{82}Rb PET in patients with and without LVH.

TABLE 2
Response to Dipyridamole-Handgrip Stress in Populations With and Without LVH

	LVH group	Non-LVH group	p
Resting heart rate	65 ± 14	63 ± 12	ns
Resting RPP (×103)	9.1 ± 2.7	8.5 ± 2.1	ns
Heartrate response (× rest)	1.3 ± 0.2	1.4 ± 0.2	ns
RPP response (× rest)	1.4 ± 0.3	1.5 ± 0.3	ns
Rb rest:stress ratio	1.1 ± 0.2	1.2 ± 0.2	ns
ECG evidence of ischemia	3/20 (15%)	9/34 (26%)	ns
Rb-PET sensitivity	11/20 (55%)	29/34 (85%)	0.03
Rb-PET specificity	3/5 (60%)	14/16 (88%)	ns

ECG = electrocardiographic and RPP = rate-pressure product.

DISCUSSION

In this study, the presence of LVH was associated with reduction of the sensitivity of dipyridamole-stress myocardial perfusion imaging for the detection of coronary artery disease. Indeed, of the variables studied, only the presence of hypertrophy distinguished between patients with true-positive and false-negative perfusion imaging results.

Study Design

The study population was derived from a group of patients with planned or recent coronary arteriography referred for ^{82}Rb PET. As the majority of these patients had chest pain or other cardiac symptoms, the group was biased towards patients with coronary artery disease, so that the sample size for assessment of specificity was relatively small. The groups with and without LVH were comparable in other clinical, therapeutic, and angiographic respects.

In order to prevent resting defects from falsely inflating

TABLE 3
Clinical, Angiographic, and Stress Findings in Patients with False-Negative Scans

Patient no.	Sex	LVH	Other diseases	Medical therapy	Coronary angiography	Stress response		
						ECG	HR	RPP
1	F	LVH	ht, as	Ca	70% RCA	no change	1.2	1.2
2	M	LVH	ht	Beta	65% LCX, 60% RCA	no change	1.6	1.7
3	F	LVH	ht	Ca, Beta	50% RCA	no change	1.1	1.2
4	F	LVH	ht	Ca, Beta, nitr	50% LAD, 70% RCA	no change	1.2	1.1
5	F	LVH	none	none	80% LCX, 65% LAD	no change	1.3	1.5
6	M	LVH	ht	Ca	75% LAD	no change	1.4	1.5
7	M	LVH	ht	Ca, Beta	100% DI, 90% RCA	no change	1.8	1.9
8	M	LVH	none	none	80% LAD, 60% LCX	no change	1.3	1.2
9	M	LVH	none	Ca, nitr	60% LAD, 70% DI	no change	1.2	1
10	F	NLV	ht	Ca, nitr	70% LAD	no change	1.4	1.5
11	M	NLV	none	Beta	60% RCA	no change	1.2	1.6
12	F	NLV	none	Beta	60% RCA	no change	1.2	1.1
13	M	NLV	none	Ca	65% LAD, 65% RCA	no change	1.3	1.2
14	M	NLV	ht	none	55% LAD	no change	1.3	1.3

AS = aortic stenosis, Beta = beta adrenoceptor antagonist, Ca = calcium antagonist, HR = heart-rate response to stress (multiple of rest), ht = hypertension, NLV = normal left ventricle, nitr = nitrates, and RPP = rate-pressure product (multiple of rest).

the sensitivity of myocardial perfusion imaging, patients with electrocardiographic or echocardiographic evidence of prior infarction were excluded from the study. Despite these measures, 16 patients had at least part of their post-stress defects apparent on the resting scan.

LVH was defined in this study by increased LV mass calculated by echocardiography (*9–11*). This approach constitutes a more sensitive determinant of LVH than electrocardiography (*14,15*), further evidenced here by the identification of this problem in only five patients by application of Estes' criteria (*16*). In this study, the calculation of LV mass was felt to be more appropriate than classification on the basis of wall thickness alone, as previous experimental observations (*17–20*) have linked LV mass (rather than wall thickness) to changes in coronary flow reserve.

PET is a highly sensitive myocardial perfusion imaging technique for the detection of coronary artery disease (*4, 21*). Due to the availability of attenuation correction, the energy of the positron annihilation photons, and high contrast resolution, it avoids some of the artifacts inherent in thallium imaging and has a higher accuracy (*13,22*). For the purpose of this in vivo study of the effect of LVH on myocardial perfusion imaging, its results are less prone to artifact than other currently available clinical techniques.

Effect of LVH on Coronary Flow

The development of LVH may be associated with inadequate growth of the coronary vasculature as well as alterations of vascular geometry (*23*). Consequently, despite increases of global myocardial blood flow above normal (*8*), resting regional flow per gram of tissue may be diminished (*24*). Several studies (*17–20*) have demonstrated that LVH is associated with reduction of maximum myocardial blood flow per gram of tissue (and thereby, flow reserve), although these findings have not been uniform, due partly to the use of vasodilator stimuli of variable efficacy, including treadmill exercise (*25*). These phenomena might act to limit the augmentation of tracer uptake into normal tissue after vasodilator stress; thus, the difference between a normal area and one supplied by a stenosed artery may be less apparent.

Influence of LVH on Perfusion Imaging

Although the impairment of coronary flow reserve in LVH has been elucidated, reduction in the accuracy of myocardial perfusion imaging due to these phenomena is less well established. The influence of LVH on specificity may involve the erroneous detection of either resting (*26*) or stress-induced defects (*27*). The latter reflect the propensity for hypertrophy to potentiate the occurrence of ischemia, even in the absence of major epicardial coronary artery disease (*27*). Such defects in the absence of significant coronary artery disease also have been described in patients with aortic stenosis (*28*). Our study, however, enrolled few patients without coronary disease (this is an inherent problem in the selection of patients after or with planned catheterization). The effect of LVH on the specificity of dipyridamole-stress perfusion imaging could be approached in another study design, for example in athletes at low probability of coronary disease.

The effect of LVH on the sensitivity of myocardial perfusion imaging for the detection of coronary artery disease may also be anticipated from existing data. Previous animal studies showing impaired coronary flow reserve in the presence of LVH correlate with recent scintigraphic findings that illustrate reduced augmentation of

myocardial rubidium activity after stress (*8*). This reduction of hyperemia in normal tissue impairs its distinction from myocardium that fails to increase rubidium uptake due to a significant coronary lesion and therefore compromises the sensitivity of the technique for the detection of coronary stenoses. In this respect, there is a paradox that although hypertrophy probably sensitizes the myocardium to ischemia, the ability to detect this with flow reserve-dependent techniques is reduced. This may not be true of exercise-stress methodologies. Standard doses of dipyridamole act as a coronary hyperemic stimulus, with metabolic phenomena of ischemia being induced relatively rarely (*29*), in contrast to exercise, where changes of regional coronary flow may be paralleled by the occurrence of ischemia. Ischemia may reduce regional coronary flow or decrease the uptake of tracer into the myocardium (*30*), augmenting the development of areas of reduced tracer uptake. Thus, despite impairment of coronary flow reserve, the sensitivity of perfusion imaging for the diagnosis of coronary disease may be maintained in exercise stress perfusion imaging by the occurrence of ischemia (*31*).

Practical Implications

The results of this study imply that a negative dipyridamole-stress perfusion scan may not effectively exclude coronary artery disease in patients with LVH. The coronary hyperemic response to standard doses of dipyridamole is attenuated in these patients, but higher doses may be more effective (this remains to be studied). Similarly, the ability of the scanner to image small areas at each extreme of high and low tracer uptake may assume critical importance when the distinction between zones of maximal and submaximal hyperemia is blunted. This study was performed using a current state-of-the-art device of comparable spatial resolution to alternative commercially available instruments, but the problems posed by LVH may be less prevalent using subsequent higher resolution devices.

CONCLUSIONS

In this study group, the sensitivity of dipyridamole-stress myocardial perfusion imaging was significantly lower in patients with LVH than in those with normal LV mass. This finding may be consistent with previous data showing a reduction of coronary flow reserve in patients with LVH. This observation may explain a proportion of patients showing the phenomenon of dipyridamole "nonresponsiveness".

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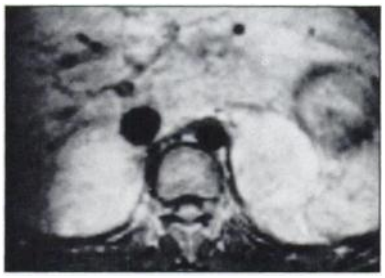
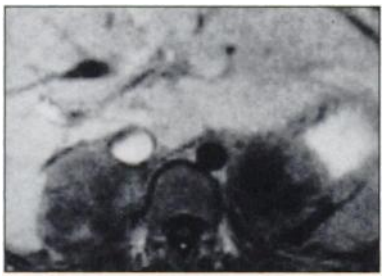
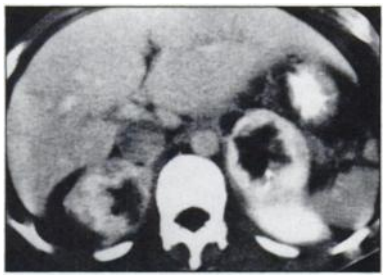
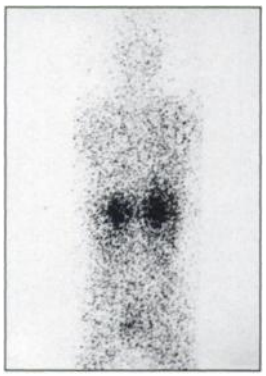
FIRST IMPRESSIONS









ACQUISITION INFORMATION
A 24-yr-old male with chest tightness, nausea and vomiting was referred for adrenal imaging to confirm bilateral pheochromocytoma. Bilateral adrenal mass was first found with ultrasonography. This patient was normotensive and had no family history. VMA (vanillylmandelic acid) level in 24-hr urine was elevated. Iodine-131-MIBG uptake in the adrenal glands was intense without discernible uptake in the heart and liver. Supplementary imaging studies of CT and MRI, which showed consistent findings of pheochromocytoma, were performed. Postoperative pathologic diagnosis was compatible with pheochromocytoma.

TRACER
[^{131}I]MIBG, 0.5 mCi

ROUTE OF ADMINISTRATION
Intraveneous injection

TIME AFTER INJECTION
24 and 72 hr

INSTRUMENTATION
Siemens 7500 Orbiter

CONTRIBUTOR
Seoung-Oh Yang

INSTITUTION
Dong-A University Hospital, Pusan, Korea