Original Article

Estimation of Myocardial Cell Damage on the Basis of Mean Electrocardiographic Voltage and Anatomical Left Ventricular Mass

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Left ventricular mass (LVM) as assessed by magnetic resonance imaging (MRI, LVM_{MRI}) and electrocardiographic (ECG) voltage reflect different pathological features. We hypothesized that ECG voltage is related to the electrical potential of cardiac muscle cells (electrical LVM) and to anatomical LVM as evaluated by MRI, and that the divergence between electrical LVM and anatomical LVM reflects the degree of myocardial damage. Because adipose tissue has high electrical resistance, we previously found a very strong correlation between body-fat-corrected mean ECG voltage (Vfm) and LVM as estimated by echocardiography in patients with essential hypertension. In this study we compared LVM_{MRI} , Vfm, the ratio of $Vfm \times 10^2/LVM_{MRI}$, and the results of ^{99m}Tc tetrofosmin scintigraphy in patients with and without myocardial infarction (MI). We studied 33 patients without MI and 26 patients with MI. Vfm significantly correlated with LVM_{MRI} in patients without MI ($r=0.71$, $p<0.01$). The ratio of $Vfm \times 10^2/LVM_{MRI}$ apparently reflected the relation between electrical LVM and anatomical LVM. $Vfm \times 10^2/LVM_{MRI}$ in patients with MI was smaller than that in patients without MI (0.98 ± 0.28 vs. 1.42 ± 0.29 , $p<0.01$). $Vfm \times 10^2/LVM_{MRI}$ decreased as ^{99m}Tc score increased ($r=-0.66$, $p<0.01$). Our results indicate that Vfm is a useful index of electrical LVM and that $Vfm \times 10^2/LVM_{MRI}$ reflects the electrical potential of the viable myocardium in total anatomical LVM. (*Hypertens Res* 2002; 25: 19–24)

Key Words: myocardial damage, LVM, ECG, MRI, ^{99m}Tc tetrofosmin

Introduction

The extent and degree of myocardial cell injury after myocardial infarction (MI) is a strong predictor of outcome (1, 2). Although many electrocardiographic (ECG) criteria have been used to estimate infarct size or predict outcome in patients with MI (3–6), little attention has been paid to the ECG potential of the salvaged myocardium and the association between mean ECG voltage and anatomical left ventricular mass (LVM), a potential index of myocardial damage.

We previously used body-fat-corrected mean ECG voltage (Vfm) to estimate LVM and compared the results with those

obtained by echocardiography (7). Because adipose tissue has high electric resistance, the amount of body fat influences ECG voltage. Vfm is more strongly correlated with LVM obtained by echocardiography than other variables in patients with mild essential hypertension.

Anatomical LVM as assessed by magnetic resonance imaging (LVM_{MRI}) includes interstitial substances and therefore does not accurately reflect cardiac muscle cell volume. On the other hand, R- and S-wave voltages on ECG are related to the electrical potential of cardiac muscle cells (electrical LVM). LVM_{MRI} and ECG voltage reflect different pathological features. To test the hypothesis that the divergence between electrical LVM and anatomical LVM reflects

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myocardial damage, we examined the relations among Vfm, LVM_{MRI}, and the results of rest ^{99m}Tc scintigraphy in patients with and without MI.

Methods

Patients

We studied 33 patients (16 men and 17 women; mean age, 65 ± 10 years) without MI and 26 patients (19 men and 7 women; 62 ± 11 years) with MI. The patients without MI were receiving treatment for diabetes mellitus, hypertension, hyperlipidemia, or hyperuricemia. All patients with MI had a documented history of MI and had received reperfusion therapy, as confirmed by ECG and coronary angiography. Patients with right or left bundle branch block, pulmonary emphysema, pericardial effusion, general edema, or metal implants were excluded, as were patients with posterior MI. All patients underwent measurement of body fat, standard 12-lead ECG, and cine MRI. Informed consent to undergo resting ^{99m}Tc tetrofosmin single photon emission computed tomography (SPECT) was obtained from all patients with MI and 12 patients without MI. Patients with MI were examined by standard 12-lead ECG at least 4 weeks after MI (8) and by cine MRI at least 1 week after MI.

Body Fat-Corrected Mean ECG Voltage (Vfm)

Body fat (kg) was measured with an impedance meter (AD6311; A&D Co., Tokyo, Japan) by means of the tetrapolar bioelectric impedance method (9, 10).

Vfm strongly correlates with left ventricular mass as assessed by echocardiography in patients with essential hypertension ($r = 0.85$) (7). To determine Vfm, the following formula was used.

$$Vfm = 0.175 \times FAT^{1/3} \times Vm + 0.5 \text{ (mV)}$$

$$Vm = (RV_5 + 2R_{EC} + 3SV_3) / 3$$

$$R_{EC} = R_E / \cos[60^\circ - \theta]^{1/2}$$

Vm, mean ECG voltage; RV₅, R wave voltage in lead V₅ (mV); R_{EC}, R_E corrected by θ ; SV₃, S wave voltage in lead V₃ (mV); R_E, length of Einthoven arrow; θ , angle of electric axis; FAT, body fat weight (kg).

Magnetic Resonance Imaging

All images were acquired with a 1.0 T MRI unit using a flexible body coil (Siemens Harmony Sysem; Siemens, Erlangen, Germany). Patients were in the supine position. Cine MRI images were acquired at base-apex short-axis locations with no slice gap during repeated breath-holds, using a segmented turbo-FLASH sequence. Typical imaging conditions were as follows: repetition time, 56 ms; echo time, 6.1 ms; flip angle, 20°; voxels, 2.96 × 1.56 × 10 mm.

LVM Measurements

Image analysis was performed with an ARGUS software package (Siemens), using Simpson's method to determine LVM. The myocardial border was outlined manually at end-diastole and end-systole by a single observer blinded to the data. The myocardial area was measured in each slice, and the area was summed to calculate myocardial volume. The volume was multiplied by myocardial density (1.05 g/cm³) to derive myocardial mass.

Radionuclide Imaging

SPECT imaging was done at rest 60 min after intravenous injection of ^{99m}Tc tetrofosmin (740 MBq). A dual-head gamma camera (Millenium VG; GE, Milwaukee, USA), equipped with a low-energy, high-resolution collimator was used. Acquisition time was 60 s per view, with 30 views collected over 180° from the right anterior oblique to the left posterior oblique projection. Images were reconstructed into a 64 × 64 matrix by the filtered back-projection method without attenuation or scatter correction.

SPECT Image Analysis

The left ventricle was divided into 20 segments; short-axial slices at 3 different levels (basal, mid-ventricular, and apical, 6 segments each) and an apical portion of the vertical long-axial slice (2 segments). Segment tracer uptake was visually scored by 2 experienced observers who were blinded to the data, using a 5-point scoring scheme: 0 = normal, 1 = equivocal, 2 = mild defect, 3 = moderate defect, 4 = severe defect. The total score was calculated for each patient.

Statistical Analyses

Variables are expressed as means ± SD. Categorical data were analyzed by the χ^2 test. Continuous variables were analyzed by the unpaired Student's *t*-test. Correlation was assessed by linear regression analysis. A *p* value of less than 0.05 was considered to indicate statistical significance.

Results

Patient Characteristics

The clinical characteristics of the patients with and without MI are summarized in Table 1. There were no significant differences between the two groups in coronary risk factors, body mass index, or body fat (11). Table 2 shows the measured variables in men and women according to group. Table 3 shows the measured variables in patients with and without hypertension according to group.

Table 1. Clinical Characteristics of Subjects

	With MI (<i>n</i> = 26)	Without MI (<i>n</i> = 33)	<i>p</i> value
Age (years)	62 ± 11	65 ± 10	0.20
Male (%)	73.1	48.5	0.06
Body mass index (kg/m ²)	23.7 ± 3.5	24.4 ± 4.5	0.51
Body fat (kg)	12.4 ± 3.2	14.3 ± 5.1	0.11
Diabetes mellitus (%)	11.5	15.2	0.69
Hypertension (%)	53.8	69.7	0.21
Blood pressure (mmHg)			
Systolic	123.4 ± 17.6	146.1 ± 22.0	< 0.01
Diastolic	66.6 ± 16.1	79.6 ± 10.7	< 0.01
Hyperlipidemia (%)	38.5	57.6	0.14
Hyperuricemia (%)	23.1	30.3	0.54
Medication (%)			
ACE inhibitor	57.7	18.2	0.02
ARB	7.7	9.1	0.85
β-blocker	30.8	30.3	0.97
Ca antagonist	15.4	39.4	0.04
Diuretic	11.5	15.2	0.69
α-blocker	3.8	0.0	0.26
Vfm (mV)	1.23 ± 0.04	1.68 ± 0.04	< 0.01
LVM _{MRI} (g)	133.0 ± 20.0	121.0 ± 30.5	0.09
MI area (%)			
Anterior	61.5		
Inferior	34.6		
Lateral	3.8		
Posterior	0.0		

Data are means ± SD or percent of patients. MI, myocardial infarction; Vfm, body fat-corrected mean RCG voltage; LVM_{MRI}, left ventricular mass obtained by MRI; ACE, angiotensin converting enzyme; ARB, angiotensin II receptor blocker.

Table 2. Measured Variables in Men and Women

Patients without MI	Men (<i>n</i> = 16)	Women (<i>n</i> = 17)	<i>p</i> value
Vfm (mv)	1.70 ± 0.03	1.68 ± 0.05	0.89
LVM _{MRI} (g)	123.0 ± 28.9	119.2 ± 32.7	0.73
Vfm × 10 ² /LVM _{MRI} (mV/g)	1.42 ± 0.30	1.43 ± 0.30	0.92
Tc score	0.0 (<i>n</i> = 6)	0.0 (<i>n</i> = 6)	
Patients with MI	Men (<i>n</i> = 19)	Women (<i>n</i> = 7)	<i>p</i> value
Vfm (mV)	1.24 ± 0.03	1.39 ± 0.03	0.32
LVM _{MRI} (g)	135.1 ± 21.0	127.6 ± 17.5	0.41
Vfm × 10 ² /LVM _{MRI} (mV/g)	0.93 ± 0.26	1.11 ± 0.30	0.15
Tc score	18.5 ± 14.4	13.1 ± 8.4	0.37

Data are means ± SD. MI, myocardial infarction; Vfm, body fat-corrected mean ECG voltage; LVM_{MRI}, left ventricular mass obtained by MRI; Tc score, Tc SPECT summed score.

Comparison between Vfm and LVM_{MRI}

Figure 1 shows the relation between Vfm and LVM_{MRI}. In patients without MI, Vfm significantly correlated with LVM_{MRI} ($r = 0.71$, $p < 0.01$). In patients with MI, Vfm was smaller than in patients without MI.

Ratio of Vfm × 10²/LVM_{MRI}

We hypothesized that Vfm is related to anatomical LVM and to the electrical potential of salvaged myocardial cells in patients with MI, and that the divergence between Vfm and anatomical LVM might reflect myocardial damage. The ratio of Vfm × 10²/LVM_{MRI} in patients with MI was significantly smaller than that in patients without MI (0.98 ± 0.28 vs. 1.42 ± 0.29 , $p < 0.01$) (Fig. 2).

Table 3. Measured Variables in Patients with and without Hypertension

Patients without MI	With HT (n = 23)	Without HT (n = 10)	p value
Vfm (mv)	1.73 ± 0.04	1.59 ± 0.04	0.37
LVM _{MRI} (g)	122.3 ± 31.7	118.1 ± 29.0	0.72
Vfm × 10 ² /LVM _{MRI} (mV/g)	1.45 ± 0.29	1.37 ± 0.30	0.52
Tc score	0.0 (n = 9)	0.0 (n = 3)	
Patients with MI	With HT (n = 15)	Without HT (n = 11)	p value
Vfm (mV)	1.37 ± 0.03	1.17 ± 0.04	0.15
LVM _{MRI} (g)	137.1 ± 22.5	127.5 ± 15.5	0.24
Vfm × 10 ² /LVM _{MRI} (mV/g)	1.02 ± 0.28	0.92 ± 0.28	0.38
Tc score	15.5 ± 14.3	19.0 ± 11.7	0.51

Data are means ± SD. HT, hypertension; MI, myocardial infarction; Vfm, body fat-corrected mean ECG voltage; LVM_{MRI}, left ventricular mass obtained by MRI; Tc score, Tc SPECT summed score.

Fig. 1. Correlation between Vfm and LVM_{MRI}. Linear regression line is for patients without myocardial infarction (MI). LVM_{MRI}, left ventricular mass obtained by MRI; Vfm, body fat-corrected mean ECG voltage.

Fig. 2. Ratio of Vfm × 10²/LVM_{MRI}. Error bars indicate means (± SD). The ratio Vfm × 10²/LVM_{MRI} in patients with myocardial infarction (MI) was significantly smaller than that in patients without MI (0.98 ± 0.28 vs. 1.42 ± 0.29, ** p < 0.01). LVM_{MRI}, left ventricular mass obtained by MRI; Vfm, body fat-corrected mean ECG voltage.

Comparison between Ratio of Vfm × 10²/LVM_{MRI} and Summed Score of SPECT

An increase in the summed score of SPECT indicates increased myocardial damage. Patients without MI had low SPECT scores, including attenuation. In the study group as a whole, the summed scores of SPECT ranged from 0 to 50, and Vfm × 10²/LVM_{MRI} decreased as the summed score of SPECT increased ($r = -0.66$, $p < 0.01$) (Fig. 3).

Discussion

Myocardial damage is associated with decreased ECG voltage (12). Few studies have evaluated the relation between LVM criteria and myocardial damage. We here examined the relations among mean ECG voltage, anatomical LVM as evaluated by MRI, and SPECT imaging score in patients with and without MI.

The extent and degree of myocardial cell injury after MI is a strong predictor of outcome (1, 2). Several studies have examined ECG criteria defining myocardial infarct size (13–15). In those studies, ST-segment elevation and QRS characteristics on admission received the most attention. As for QRS characteristics, poor R-wave progression indicates myocardial damage (10). Other investigations have shown that patients with non-Q wave infarction have a better outcome than those with Q wave infarction (16, 17). We focused our attention on decrease in the viable myocardium as indicated by R- and S-wave ECG voltages. Because R-wave voltage changes in the early phase after MI (8), a standard 12-lead ECG was performed at least 4 weeks after MI.

LVM_{MRI} and ECG voltage reflect different pathological features. In addition to hypertrophy of cardiac muscle cells, higher concentrations of interstitial substances such as fibroblasts and collagen can increase LVM_{MRI}. Although left ventricular wall thickness can reflect a damaged myocardium

Fig. 3. Correlation between $V_{fm} \times 10^2 / LVM_{MRI}$ and Tc SPECT summed score (Tc Score). MI, myocardial infarction; V_{fm} , body fat-corrected mean ECG voltage; LVM_{MRI} , left ventricular mass obtained by MRI.

after MI (18), this variable sometimes does not decrease in patients with non-transmural infarction. In such patients, the size of the anatomical myocardium differs from that of the viable myocardium. On the other hand, R- and S-wave ECG voltages are related to cardiac muscle action potential. In patients without MI, V_{fm} is related to the size of the non-damaged myocardium (Fig. 1). It is difficult to estimate myocardial damage solely on the basis of V_{fm} in patients with severe left ventricular hypertrophy who have a small myocardial infarction. The quantity of anatomical V_{fm} must be adjusted to evaluate the relation between V_{fm} and myocardial damage. We therefore used the ratio of $V_{fm} \times 10^2 / LVM_{MRI}$ to assess myocardial damage. The correlation between $V_{fm} \times 10^2 / LVM_{MRI}$ and left ventricular ejection fraction obtained by MRI ($r = 0.45$, $p < 0.01$) was stronger than that between V_{fm} and left ventricular ejection fraction ($r = 0.26$, $p < 0.05$).

The use of ^{99m}Tc tetrofosmin for scintigraphy provides better quality images than the use of ^{201}Tl (19). We used ^{99m}Tc tetrofosmin, which indicates myocardial perfusion, to estimate myocardial damage. The ratio of $V_{fm} \times 10^2 / LVM_{MRI}$ in patients with MI was significantly lower than that in patients without MI (Fig. 2). Furthermore, $V_{fm} \times 10^2 / LVM_{MRI}$ decreased as SPECT summed score increased (Fig. 3), suggesting that $V_{fm} \times 10^2 / LVM_{MRI}$ reflects the amount of viable myocardium. Some patients with MI underwent early recanalization. These patients had low SPECT scores, and $V_{fm} \times 10^2 / LVM_{MRI}$ was smaller than that in patients without MI, suggesting that V_{fm} reflects latent myocardial damage not detected on Tc SPECT. However, the size of anterior MI was larger than that of inferior MI (21.5 ± 14.6 vs. 9.8 ± 5.9 , $p < 0.05$), and $V_{fm} \times 10^2 / LVM_{MRI}$ of anterior MI was smaller

than that of inferior MI (0.86 ± 0.25 vs. 1.21 ± 0.14 , $p < 0.01$). The size of anterior MI therefore might be overestimated on the basis of $V_{fm} \times 10^2 / LVM_{MRI}$.

We used Simpson's method to evaluate anatomical LVM on MRI scans. MRI can accurately and reliably measure anatomical LVM but has some limitations—*i.e.*, it is expensive and cannot be used in patients with metal implants, such as pacemakers, artificial valves, or stents (20). We excluded such patients from our study. Echocardiography is widely used to measure LVM. It is moderately expensive and has few limitations, but is expertise-dependent and may give erroneous results in patients with distorted ventricles (20). LVM is difficult to measure by echocardiography in patients with coronary artery disease, because the damaged myocardium may be thin. LVM cannot be calculated on the basis of only one dimension. Furthermore, as compared with MRI, echocardiography tends to overestimate LVM (21, 22). Therefore, despite the cost and labor involved, MRI remains a useful and accurate technique for evaluating LVM.

Left ventricular hypertrophy is a strong predictor of cardiovascular morbidity and mortality (23–25). Clinically, evaluation of left ventricular hypertrophy facilitates risk stratification. ECG is a practical, inexpensive method providing highly reproducible data; however, ECG cannot accurately evaluate anatomical LVM (20, 26). Various criteria have been developed to evaluate LVM by ECG (3–6). However, these criteria have not been validated by MRI and have not been corrected by electrical resistance. Electrical potential attenuates before reaching electrodes, and the high electrical resistance of adipose tissue necessitates correction on the basis of body fat. Therefore, we previously described a method to measure V_{fm} (7). In this study, we demonstrated a significant correlation between V_{fm} and LVM_{MRI} (Fig. 1).

There were few women among the patients with MI in the present study. Among patients with MI, the infarct size of women was smaller than that of men, and the $V_{fm} \times 10^2 / LVM_{MRI}$ of women was larger than that of men (Table 2). Although the patients with MI showed a trend toward left ventricular hypertrophy (Table 3), their blood pressure was significantly lower than that in the patients without MI (Table 1). Because most patients with MI received ACE inhibitors in line with AHA recommendations irrespective of the presence or absence of hypertension, the blood pressure of some patients after MI might have been lower than that before MI.

Our study had several limitations. First, various factors influence electrical resistance between the heart and electrodes (5, 26). It is well known that R- and S-wave voltages are decreased in patients with emphysema and obesity (5, 26). Because V_{fm} cannot correct for these factors, patients with pulmonary emphysema, pericardial effusion, and generalized edema were not included in this study. Second, because the R-wave voltage of the right precordial leads increases in patients with posterior MI, it is difficult to accurately estimate V_{fm} in such patients. These patients were also excluded.

Third, although pathological fluctuations after myocardial infarction are lower with cine MRI and SPECT than with ECG (27), patients with MI should have undergone cine MRI and SPECT at least 4 weeks after MI. In patients without MI there were no significant diurnal variations in ECG. The time-lag between ECG and MRI was 7 days (mean) in patients with MI and 28 days (mean) in patients without MI. The time-lag between ECG and SPECT was 28 days and 62 days, and that between SPECT and MRI was 32 days and 64 days, in patients with MI and without MI, respectively.

In conclusion, this study indicates that V_{fm} is a useful index of electrical LVM and that $V_{fm} \times 10^3 / LVM_{MRI}$ reflects the electrical potential of the viable myocardium in total anatomical LVM.

References

- Gersh BJ, Anderson JL: Thrombolysis and myocardial salvage: result of clinical trials and the animal paradigm. *Circulation* 1993; **88**: 296–306.
- Geltman EM: Infarct size as a determinant of acute and long-term prognosis. *Cardiol Clin* 1984; **2**: 95–103.
- Casale PN, Devereux RB, Alonso DR, *et al*: Improved sex-specific criteria of left ventricular hypertrophy for clinical and computer interpretation of electrocardiograms: validation with autopsy findings. *Circulation* 1987; **75**: 565–572.
- Sokolow M, Lyon TP: The ventricular complex in left ventricular hypertrophy as obtained by unipolar precordial and limb leads. *Am Heart J* 1949; **37**: 161–186.
- Rautaharju PM, Manolio TA, Siscovick D, *et al*: Utility of new electrocardiographic models for left ventricular mass in older adults. *Hypertension* 1996; **28**: 8–15.
- Robert JS, William CR: Electrocardiographic observations in severe aortic valve stenosis: correlative necropsy study to clinical, hemodynamic, and ECG variables demonstrating relation of 12-lead QRS amplitude to peak systolic transaortic pressure gradient. *Am Heart J* 1982; **103**: 210–221.
- Tochikubo O, Miyajima E, Shigemasa T, *et al*: Relation between body fat-corrected ECG voltage and ambulatory blood pressure in patients with essential hypertension. *Hypertension* 1999; **33**: 1159–1163.
- Bashour TT, Kabbani SS, Brewster HP, *et al*: Transient Q wave and reversible cardiac failure during myocardial ischemia: electrical and mechanical stunning of the heart. *Am Heart J* 1983; **106**: 780–783.
- Lukaski HC, Bolonchuk WW, Hall CB, *et al*: Validation of tetrapolar bioelectrical impedance method to assess human body composition. *J Appl Physiol* 1986; **60**: 1327–1332.
- Lukaski HC, Johnson PE, Bolonchuk WW, *et al*: Assessment of fat-free mass using bioelectrical impedance measurements of the human body. *Am J Clin Nutr* 1985; **41**: 810–817.
- Matuo K, Mikami H, Itoh M, Ogihara T, Tuck ML: Sympathetic activity and body mass index contribute to blood pressure levels. *Hypertens Res* 2000; **23**: 303–310.
- Zema MJ, Kligfield P: Electrocardiographic poor R wave progression II: correlation with angiography. *J Electrocardiol* 1979; **12**: 11–15.
- Haisty WK, Pahlm O, Wangner NB, *et al*: Performance of automated complete Selvester QRS scoring system in normal subjects and patients with single and multiple myocardial infarction. *J Am Coll Cardiol* 1992; **19**: 341–346.
- Willems JL, Willems RJ, Willems GM, *et al*: Significance of initial ST segment elevation and depression for the management of thrombolytic therapy in acute myocardial infarction. *Circulation* 1990; **82**: 1147–1158.
- Birnbaum Y, Kloner RA, Sclarovsky S, *et al*: Distortion of the terminal portion of the QRS on the admission electrocardiogram in acute myocardial infarction and correlation with infarct size and long-term prognosis (thrombolysis in myocardial infarction 4 trial). *Am J Cardiol* 1996; **78**: 396–403.
- Barbagelata A, Califf RM, Sgarbossa EB, *et al*: Thrombolysis and Q wave versus non-Q wave first acute myocardial infarction: a GUSTO-I substudy. *J Am Coll Cardiol* 1997; **29**: 770–777.
- Matetzky S, Barabash GI, Rabinowitz B, *et al*: Q wave and non-Q wave myocardial infarction after thrombolysis. *J Am Coll Cardiol* 1995; **26**: 1445–1451.
- Baer FM, Voth E, Schneider CA, *et al*: Comparison of low-dose dobutamine-gradient-echo magnetic resonance imaging and positron emission tomography with [^{18}F]fluorodeoxyglucose in patients with chronic coronary artery disease. *Circulation* 1995; **91**: 1006–1015.
- Zaret BL, Rigo P, Wackers FJT, *et al*: Myocardial perfusion imaging with ^{99m}Tc tetrofosmin. *Circulation* 1995; **91**: 313–319.
- Devereux RB, Pini R, Aurigemma GP, *et al*: Measurement of left ventricular mass: methodology and expertise. *J Hypertens* 1997; **15**: 801–809.
- Missouris CG, Forbat SM, Singer DRJ, *et al*: Echocardiography overestimates left ventricular mass: a comparative study with magnetic resonance imaging in patients with hypertension. *J Hypertens* 1996; **14**: 1005–1010.
- Bottini PB, Carr AA, Prisant M, *et al*: Magnetic resonance imaging compared to echocardiography to assess left ventricular mass in the hypertensive patient. *Am J Hypertens* 1995; **8**: 221–228.
- Levy D, Garrison RJ, Savage DD, *et al*: Prognostic implications of echocardiographically determined left ventricular mass in the Framingham heart study. *N Engl J Med* 1990; **322**: 1561–1566.
- Haider AW, Larson MG, Benjamin EJ, *et al*: Increased left ventricular mass and hypertrophy are associated with increased risk for sudden death. *J Am Coll Cardiol* 1998; **32**: 1454–1459.
- Devereux RB, Roman MJ: Left ventricular hypertrophy in hypertension: stimuli, patterns, and consequences. *Hypertens Res* 1999; **22**: 1–9.
- Levy D, Labib SB, Anderson KM, *et al*: Determinants of sensitivity and specificity of electrocardiographic criteria for left ventricular hypertrophy. *Circulation* 1990; **81**: 815–820.
- Tanaka R, Nakamura T: Time course evaluation of myocardial perfusion after reperfusion therapy by ^{99m}Tc -tetrofosmin spect in patients with acute myocardial infarction. *J Nucl Med* 2001; **42**: 1351–1358.