# Resting Asynchronous Left Ventricular Contraction Abnormality Analyzed by a Phase Method in Spastic Angina Pectoris

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Quantitative phase analysis of equilibrium ventriculography was performed to study the character of left ventricular (LV) wall motion abnormalities in patients with spastic angina pectoris, who may have clinically and electrocardiographically silent ischemia combined with myocardial stunning, during rest and hyperventilation stress testing. **Methods:** Phase analysis of the left ventricle at rest was performed by equilibrium radionuclide ventriculography in 13 control subjects and 36 patients with spastic angina pectoris. First-pass methodology along with hyperventilation stress testing was performed to assess spasm occurrences. Phase analysis of equilibrium multigated blood-pool scintigrams was performed to evaluate LV asynchrony at rest. **Results:** The mean s.d. of LV phase distribution in the patients with variant and vasospastic angina was greater than that in the healthy control subjects (11.28 ± 1.79 and 10.02 ± 1.57 degrees versus 6.16 ± 1.07 degrees). In addition, the mean s.d. of LV phase distribution in the variant angina group was greater than that in the vasospastic angina group. Furthermore, a linear correlation was found between the s.d. of LV phase distribution at rest and the percent decrease in ejection fraction during hyperventilation stress. **Conclusion:** Asynchronous LV contraction without significant hypokinesis was detected at rest in spastic angina pectoris. The severity of this asynchronous contraction corresponded well with decreases in ejection fraction during hyperventilation stress testing. Thus, analysis of the s.d. of LV phase distribution at rest is expected to provide useful information regarding LV asynchrony in spastic angina pectoris.

**Key Words:** variant angina pectoris; vasospasm; phase analysis; hyperventilation stress test; myocardial stunning

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Coronary artery spasm in the absence of fixed organic coronary artery obstruction causes the pathophysiologic syndrome known as spastic angina pectoris (*1–4*). The identification of angina due to vasospasm is clinically important because coronary artery spasm may cause subjectively silent myocardial ischemia, which is sufficient to produce detectable ventricular dysfunction, wall motion abnormalities and life-threatening arrhythmias (*1–2,5–6*). Identifying vasospasm-induced angina is often difficult. The diagnosis of spastic angina is based on clinical history, electrocardiograms (ECG) at the onset of anginal attack, Holter ECG and coronary arteriography with ergonovine or acetylcholine provocation. Radionuclide ventriculography in combination with various types of stress testing has been widely recognized as a useful diagnostic method for coronary artery disease (CAD) (*3,7–9*). Left ventricular (LV) dysfunction during stress testing is strongly correlated with obstructive coronary lesions. The hyperventilation stress test has been used to detect spastic angina because of its high sensitivity and noninvasiveness (*3,9–16*).

Since 1979, Fourier analysis has been used in multigated cardiac blood-pool scintigraphy (*17–24*). It provides valuable information regarding the onset and sequence of ventricular contraction. One of the major clinical applications of phase analysis is the detection of asynchronous ventricular contraction, as occurs in bundle branch block (*19–21*), Wolff-Parkinson-White (WPW) syndrome (*21–22*) and CAD (*23–25*). Analysis of s.d. of LV phase distribution has been reported to improve the diagnostic sensitivity for coronary artery disease (*25*), but phase analysis has not been used previously for diagnosis of vasospastic angina.

This study uses quantitative phase analysis to examine the existence of asynchronous LV contraction and its characteristics at rest in patients with spastic angina pectoris; it also compares phase analysis with the occurrance of abnormal LV wall motion induced by hyperventilation stress.

## METHODS

### Patients

The study population consisted of 36 spastic angina patients and 13 normal controls. The spastic angina patients were subdivided as those with variant angina pectoris and vasospastic angina based on coronary arteriography and ECG results as follows: (1)

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in the variant angina pectoris group, 22 patients had 75% or higher coronary spasm and transient ST-segment elevation; (2) in the vasospastic angina group, 14 patients had 50%–75% coronary spasm and transient ST-segment depression.

Twenty-nine of the 36 patients with spastic angina were men and 7 were women, age range 37 to 71 yr (mean 56.6 ± 8.9 yr). No patient had previous myocardial infarction, congestive heart failure or valvular heart disease. The 13 control subjects (7 men, 6 women) were examined to establish normal values for phase analysis. Their ages ranged from 22 to 70 yr (mean 53 ± 14.4 yr). None of them presented any evidence of ECG disease on history, physical examination, resting and stress ECG, echocardiogram, stress $^{201}$Tl myocardial scintigram or during equilibrium radionuclide ventriculography of diastolic LV function.

### Hyperventilation Test

The hyperventilation stress test was performed by requiring the subjects to breathe as deeply and rapidly as possible (at least 40 inspirations per min) for 5 min following a metronome. A 12-lead ECG and blood pressure were recorded at 1-min intervals during the examination. A positive stress test was signified by characteristic electrocardiographic changes (ST-segment elevation or depression ≥1 mm at 0.08 sec after the J point) with or without chest pain during the examination. Using the first-pass method, imaging of the LV was performed before HV stress and at 3 min after the end of the stress. If angina or ST-segment changes appeared, stress images were obtained as soon as possible.

### Radionuclide Technique

The first-pass method was used to evaluate the LV dysfunction produced by hyperventilation stress induced spasm. Multigated blood-pool scintigraphy was performed to assess the regional synchronicity at rest, because the statistical noise of the image is markedly reduced with this method. Six milligrams of stannous chloride pyrophosphate dissolved in 3 ml of saline was intravenously injected as pre-medication. The first-pass images were obtained following the bolus intravenous injection of 555–740 MBq (15–20 mCi) [$^{99m}$Tc]pertechnetate at rest. Soon after the resting first-pass examination, multigated equilibrium blood-pool scintigraphy was performed using in-vivo labeled to red blood cells. Hyperventilation stress first-pass examination was done following the bolus IV injection of 555 MBq (15 mCi) of $^{99m}$Tc-pertechnetate. Background subtraction was performed during stress with first-pass methodology. Images were obtained with a gamma camera (ZLC-7500, Siemens Co., Ltd.) using a slant-hole, high-resolution, low-energy collimator. First-pass images were obtained in the anterior position, and multigated equilibrium images were obtained in the modified left anterior oblique (LAO) position with a 15° caudal tilt to diminish the overlapping of cardiac structures. The following acquisition parameters were used: an energy window of 20%, a peak at 140 keV, a 40-msec interval after the R wave, and 10% rejection of abnormal beats (corresponding to about 600 beats). Data were processed using a SINTIPAC-700 computer system (Shimadzu Co. Ltd., Japan) and 64 × 64 matrix images were obtained. The resting and post-stress ejection fraction based on the first-pass image data (resting ejection fraction and hyperventilation ejection fraction) and the resting ejection fraction based on the equilibrium image data were calculated from time-activity curves. In our laboratory, a study is considered abnormal if ejection fraction decreases by more than 5% under the hyperventilation stress condition (*7–9*).

The percentage of change in ejection fraction under resting and post-stress conditions was calculated as follows:

$$\%\ \mathrm{EF} = \frac{\text{resting EF–HVEF}}{\text{resting EF}} \times 100\%, \qquad \text{Eq. 1}$$

where EF is ejection fraction and HVEF is hyperventilation ejection fraction.

### Phase Analysis of Equilibrium Blood Pool Images

Phase analysis of multigated equilibrium blood-pool scintigrams was performed by using the first component of Fourier harmonics to fit a cosine curve to the time-activity curve of each pixel. Phase histograms of the LV were constructed from the phase images obtained in the modified LAO projection. The borders of the LV were manually determined using an amplitude image, and the left atrium and pulmonary vessels were eliminated. The mean phase value and s.d. for the LV were then calculated from these histograms as absolute values in milliseconds. The s.d. in degrees was then calculated as follows:

$$\text{s.d. (degrees)} = \frac{\text{s.d. (ms)} \times 360}{\text{mean R-R interval (ms)}}. \qquad \text{Eq. 2}$$

### Coronary Arteriography

Coronary arteriography was performed using either the Judkins or Sones technique. The doses of intravenous methylergometrine were 1, 5 and 10 μg at the right coronary artery, and 1, 5, 10 and 30 μg at the left coronary artery at 5-min intervals. The severity of coronary spasm was determined by the ergonovine provocation.

### Statistical Analysis

All data are expressed as the mean ± s.d. Student's t-test for paired variables was used for comparisons, and linear regression analysis by the least squares method was performed to assess the relationship between the s.d. of the phase distribution of the left ventricle and percentage of ejection fraction. A p value of less than 0.05 was considered significant.

## RESULTS

### Left Ventriculography and Hyperventilation Stress Testing

Technetium-99m blood-pool scintigraphy (multigated equilibrium method) was performed on the 36 patients (22 variant angina pectoris and 14 vasospastic angina patients) and on the 13 control subjects. The ejection fraction at rest and after hyperventilation stress (resting ejection fraction and hyperventilation ejection fraction) were obtained by the first-pass method. There was no patient who had angina or significant ECG abnormality during the first-pass studies at rest and under hyperventilation stress, or during multigated equilibrium studies at rest.

A reduction of 5% of the ejection fraction under the hyperventilation stress condition was seen in 23 of the 36 spastic angina patients (64%). In the spastic angina patients, the resting ejection fraction determined by the multigated equilibrium method tended to be lower than that in control subjects, but the difference was not statistically significant (variant angina pectoris group, 57.9% ± 6.45%; vasospastic angina group, 58.3% ± 4.90%; controls, 62.4% ± 5.61%) (Fig. 1).

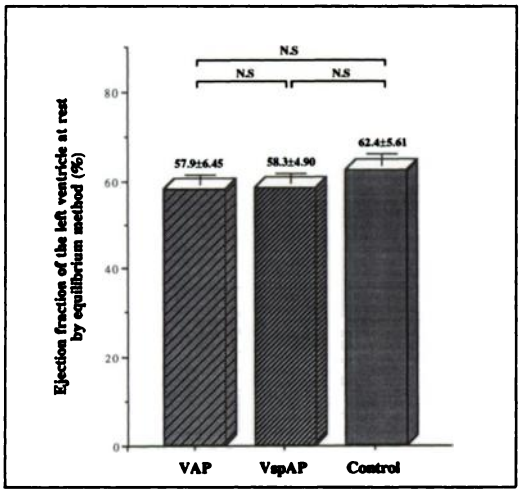


**FIGURE 1.** Mean ejection fraction obtained by multigated equilibrium method at rest in normal control subjects and in spastic angina patients (variant angina pectoris and vasospastic angina groups). In the angina patients, the resting ejection fraction tended to be lower than that in the control subjects, but there was no significant difference.

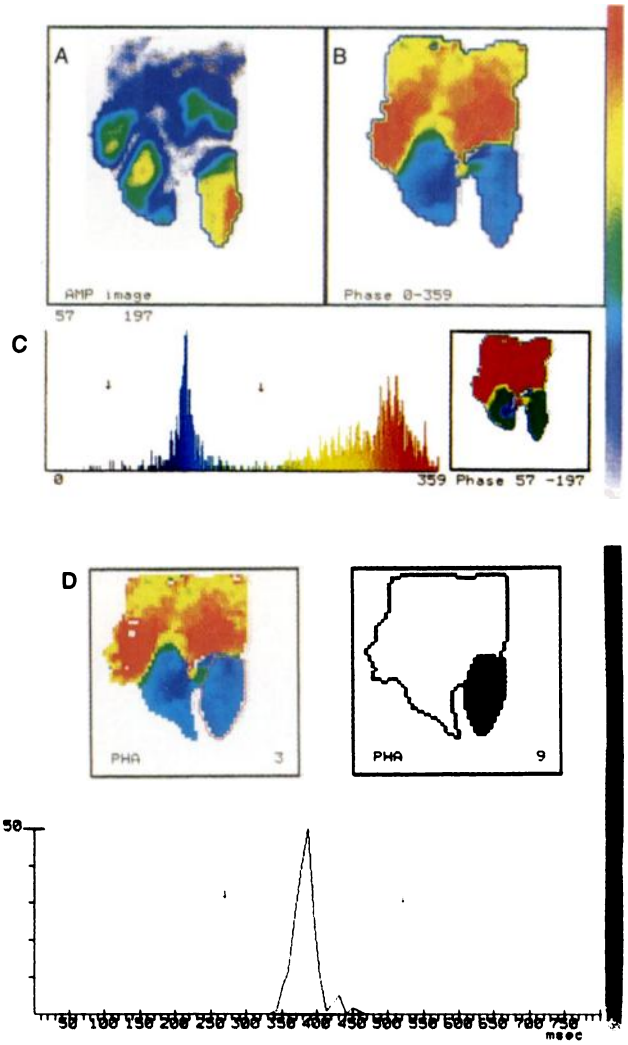


**FIGURE 2.** Quantitative phase analysis of left ventricle in a normal 57-yr-old man. The amplitude image shows normokinesis in the left ventricle (A), and the phase image shows that the phase was relatively homogeneous contraction (B and D). The phase histogram had a single spike (C and E), and in the quantitative phase analysis of the left ventricle, the standard deviation of the LV phase distribution was 6.20°.

### Phase Analysis

The mean s.d. of LV phase distribution was determined from LAO images obtained by the multigated equilibrium method. The relationship between the percentage of ejection fraction under hyperventilation stress (first-pass method) and the s.d. of LV phase distribution (multigated equilibrium method) was analyzed. Figures 2 and 3 show the amplitude image, phase image and phase histogram at rest in a normal subject and in a patient with variant angina pectoris, respectively.

There was a significantly greater s.d. phase of LV distribution in the spastic angina patients (variant angina pectoris and vasospastic angina groups) than in the control subjects (11.28 ± 1.79 and 10.02 ± 1.57°, respectively, versus 6.16 ± 1.07°, both $p < 0.0001$), and the s.d. of LV phase distribution of the variant angina pectoris patients was greater than that of the vasospastic angina patients ($p < 0.05$) (Fig. 4). Among the 36 spastic angina patients, 34 (94%) showed nonhomogeneous contraction, and the s.d. of LV phase distribution was greater than that in the control subjects (≥ 8.30°). Furthermore, a linear correlation was found between s.d. of LV phase distribution and the percentage of ejection fraction in the spastic angina patients ($r = 0.81$, $p < 0.0001$), but there was no significant relationship between the s.d. of LV phase distribution and the resting ejection fraction (Fig. 5 A, B).

## DISCUSSION

Coronary artery spasm has become the focus of increasing attention. It may cause subjectively silent myocardial ischemia to produce detectable ventricular dysfunction, wall motion abnormalities and life-threatening arrhythmias (*1–2,5–6*). But the transience of coronary artery spasm makes it difficult to diagnose. The diagnosis of variant angina is usually established based on clinical history, ECG during the onset of attack, Holter ECG and finally coronary arteriography with ergonovine or acetylcholine provocation, but diagnosis is occasionally difficult. Consequently, several stressors that induce coronary spasm (cold, exercise and hyperventilation) have been used to provoke coronary artery spasm (*3,9–16*). Kaski et al. reported that the sensitivity of hyperventilation, exercise and cold stress test was 54%, 46% and 11%, respectively, for the production of electrocardiographic changes in variant angina pectoris patients (*16*). Perez Balino et al. showed that the efficiency of radionuclide ventriculography in combination with hyperventilation and cold stress was greater than that of ECG in detecting ischemia, and that the sensitivity of hyperventilation and the cold test were 74% and 89%, respectively, in patients with vasospastic angina suspected on the basis of clinical and electrocardiographic

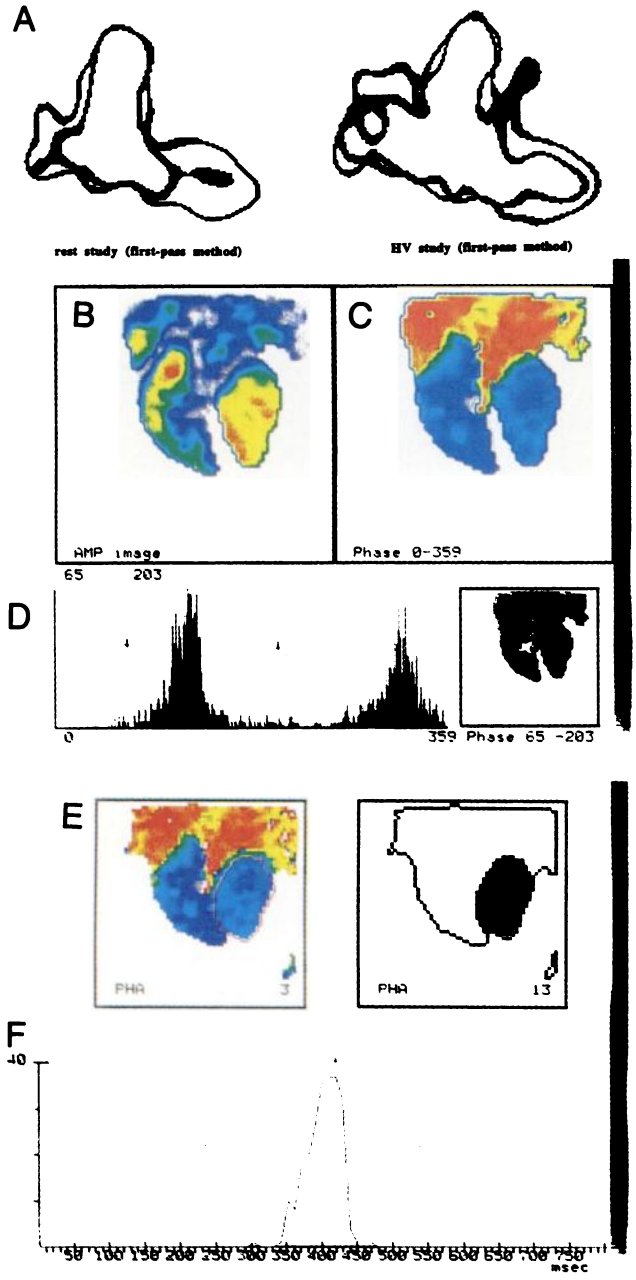


**FIGURE 3.** Quantitative phase analysis of the left ventricle in a 61-yr-old man with variant angina pectoris. Left ventricular wall motion changed from normokinesis (ejection fraction = 61.9%) to diffusion hypokinesis in response to hyperventilation stress (ejection fraction = 48.5%) by first-pass method (A). The amplitude image demonstrates hypokinesis in the apico-posterolateral regions (B), and the phase image shows nonhomogeneous contraction (C and E). Greater values of phase distribution were observed in the phase histogram (D and F). In the quantitative phase analysis of the left ventricle, the standard deviation of the left ventricular phase distribution was 10.78° (C).

findings (*3*). In our study, hyperventilation caused a decrease of 5% or more in ejection fraction in 23 of the 36 spastic angina patients, and the sensitivity was thus 64%. The ejection fraction at rest in these patients tended to be lower than that in the control subjects. These results are consistent with those reported by Perez Balino et al. (*3*).

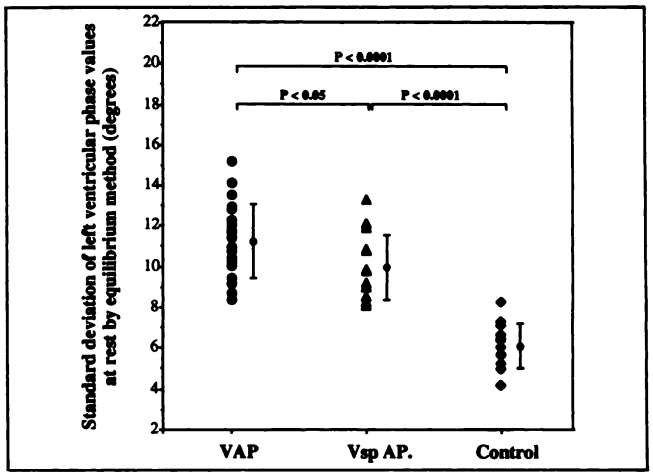


**FIGURE 4.** Standard deviation of left ventricular phase distribution in normal control subjects, in variant angina pectoris and in vasospastic angina groups.

## Asynchronous Left Ventricular Contraction by Phase Analysis

Phase analysis quantifies the temporal sequence of regional wall motion by employing a pixel-by-pixel Fourier transformation of the ventricular time-activity curve and can be used to evaluate ventricular function in radionuclide ventriculography. One of the advantages of this method is its high sensitivity in the detection of the asynchronous ventricular contraction that occurs in bundle branch block (*19–21*), the WPW syndrome (*21–22*) and coronary artery disease (*23–25*).

This study revealed that the mean s.d. of LV phase distribution of the spastic angina patients was markedly greater than that of the controls. Thirty-four of the 36 patients with vasospasm (94%) showed abnormal s.d. of LV phase distribution values even at rest, and the severity of asynchronous contraction in the variant angina pectoris group was greater than that in the vasospastic angina group. These results indicate that asynchronous ventricular contraction without significant hypocontraction occurs at rest and is severe in patients with variant angina pectoris.

The findings indicating asynchronous, resting ventricular contraction without significant hypocontraction might be explained by several mechanisms. Clinically and electrocardiographically "silent" resting ischemia, which is sufficient to produce detectable ventricular dysfunction, might be present in these patients (*5*). In fact, coronary spasm produces wall motion abnormalities such as early systolic bulging and early diastolic contraction (*6*). Alternatively, myocardial stunning might have occurred. Brief periods of myocardial ischemia that do not cause myocardial necrosis may be generated by vasospasm of the coronary arteries, and this ischemia may produce myocardial stunning (*1,6,26–27*). The available evidence regarding myocardial stunning in variant angina is described in four papers (*27–30*). Finally, diastolic ventricular dysfunction, which frequently occurs in myocardial ischemia, might

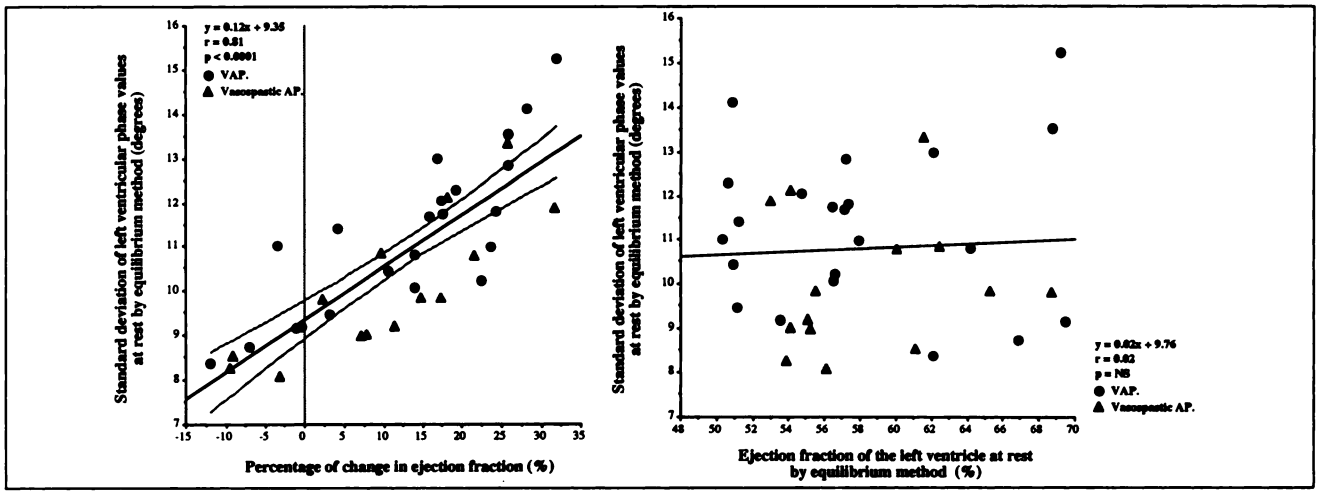


**FIGURE 5.** Relationship between the percentage of change in ejection fraction and the s.d. of LV phase distribution in the spastic angina patients (A) and that between resting ejection fraction and the s.d. of LV phase distribution (B) in the spastic angina patients.

also be present (*31*). Diastolic stunning which continues for a few days without significant hypokinesis might be especially important. This phenomenon has been reported after percutaneous transluminal coronary angioplasty (*32–33*) and after acute myocardial infarction with reperfusion (*34*), so the occurrence of diastolic stunning in variant angina pectoris might not be so unusual.

### Relationship between Standard Deviation of Left Ventricular Phase Distribution and Percent Ejection Fraction

The relationship between s.d. of LV phase distribution and ejection fraction has been investigated in coronary artery disease, but there have been no reports on the relationship between s.d. of LV phase distribution at rest and the percentage of ejection fraction changes caused by hyperventilation stress. Schwaiger et al. found a linear inverse correlation between ejection fraction changes and the s.d. of LV phase distribution at rest and during exercise stress in coronary artery disease patients (*24*).

In our study, a linear correlation was seen between the percentage of ejection fraction changes caused by hyperventilation stress and the s.d. of LV phase distribution at rest in patients with spastic angina, but no significant relationship was observed between resting ejection fraction and s.d. of LV phase distribution. This indicates that the asynchrony is severe in patients with coronary vasospasm induced by hyperventilation stress. In contrast, measurement of the resting ejection fraction is insufficient to evaluate ventricular function in spastic angina, as in the case of coronary artery disease.

The ejection fraction and s.d. of LV phase distribution reflect different aspects of left ventricular function, since the ejection fraction corresponds to the global ventricular function, whereas the s.d. of LV phase distribution corresponds to regional synchronicity (*18*). Therefore, the s.d. of LV phase distribution might be a more sensitive parameter in the detection of regional myocardial dysfunction in spastic angina pectoris.

### Limitations

The production of errors may occur in the calculation of the phase value in the region of the ventricle affected by cardiac angulation, and the LV and left atrium (LA) overlap. We therefore introduced the modified LAO position (15° caudal tilt) to minimize the overlap of the LV, LA and the effect on cardiac angulation. In normal subjects, it is reported that there is little variation of the LV phase value within the left ventricle, even in the region of LV and LA overlap (*19,21*); however, the presence of LV dilation or mitral regurgitation might cause an error of phase value in this region. The effect of spasm of the right coronary artery might be underestimated or overlooked in this LAO projection.

## CONCLUSION

This study revealed that asynchronous LV contraction without significant hypokinesis is present at rest in patients with spastic angina pectoris, and that the severity of this asynchronous contraction is well correlated with the percentage of ejection fraction change induced by hyperventilation. The sensitivity of s.d. of LV phase distribution at rest (94%) is superior to that of percentage of ejection fraction change induced by hyperventilation stress (64%). Accordingly, the analysis of the s.d. of LV phase distribution at rest is expected to provide useful information regarding LV function in spastic angina pectoris.

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## REFERENCES


1. Maseri A, Severi S, De Nes M, et al. Variant angina: one aspect of a continuous spectrum of vasospastic myocardial ischemia: pathogenetic mechanisms, estimated incidence and clinical and coronary arteriographic finding in 138 patients. *Am J Cardiol* 1978;42:1019–1035.
2. Heupler FA. Syndrome of symptomatic coronary arterial spasm with nearly normal coronary arteriograms. *Am J Cardiol* 1980;45:873–881.
3. Perez Balino N, Liprandi AS, Masoli O, et al. Usefulness of radionuclide ventriculography in assessment of coronary artery spasm. *Am J Cardiol* 1987;59:552–558.
4. Kugiyama K, Yasue H, Okumura K, et al. Coronary artery spasm demonstrated by quantitative analysis of thallium-201 single-photon emission computed tomography. *Am J Cardiol* 1987;60:1009–1014.
5. Crea F, Abela GS, Pepine CJ. Multivessel coronary spasm without electrocardiographic changes. *Am Heart J* 1985;110:1064–1067.
6. Aksnes G, Kirkeboen KA, Christensen G, Ilebekk A. Characteristics and development of myocardial stunning in the pig. *Am J Physiol* 1992;263: H544–H551.
7. Pfisterer ME, Slutsky RA, Schuler G, et al. Profiles of radionuclide left ventricular ejection fraction changes induced by supine bicycle exercise in normal and patients with coronary heart disease. *Cathet Cardiovasc Diagn* 1979;5:305–317.
8. Berger HJ, Reduto LA, Johnston DE, et al. Global and regional left ventricular response to bicycle exercise in coronary artery disease. *Am J Med* 1979;66:13–21.
9. Masuoka T. A new diagnostic application with nuclear cardiology in patients with ischemic heart disease (in Japanese). Doctoral thesis, the University of Tsukuba, Japan 1989.
10. Imamura T, Nagatomo Y, Nakagawa S, et al. Hyperventilation thallium-201 myocardial imaging for the diagnosis of vasospastic angina. *Clin Nucl Med* 1987;12:729–734.
11. Falstie-Jensen N, Engby B, Rasmussen K, Bagger JP, Thuesen L, Tagehoj-Jensen E. Vasospastic angina assessed by hyperventilation thallium-201 myocardial scintigraphy. *Acta Med Scand* 1987;222:133–136.
12. Mitani I, Nishimura T, Uehara T, et al. Hyperventilation stress thallium-201 scintigraphy: clinical application to a patient of variant angina [Abstract in English]. *Kakuigaku* 1988;25:71–75.
13. Ardissino D, Barberis P, De Servi S, et al. Usefulness of the hyperventilation test in stable exertional angina pectoris in selecting medical therapy. *Am J Cardiol* 1990;15:417–421.
14. Girotti LA, Crosatto JR, Messuti H, et al. The hyperventilation test as a method for developing successful therapy in Prinzmetal's angina. *Am J Cardiol* 1982;49:834–841.
15. Ardissino D, De Servi S, Barberis P, et al. Significance of hyperventilation-induced ST-segment depression in patients with coronary artery disease. *J Am Coll Cardiol* 1989;13:804–810.
16. Kaski JC, Crea F, Meran D, et al. Local coronary supersensitivity to diverse vasoconstrictive stimuli in patients with variant angina. *Circulation* 1986;74:1255–1265.
17. Adam WE, Tarkowska A, Bitter F, et al. Equilibrium (gated) radionuclide ventriculography. *Cardiovasc Radiol* 1979;2:161–173.
18. Bonaduce D, Morgano G, Petretta M, et al. Phase analysis of radionuclide angiography in acute myocardial infarction. *Eur J Nucl Med* 1990;16:161–165.
19. Takeda T, Toyama H, Iida K, et al. A study of ventricular contraction sequence in complete right bundle branch block by phase analysis. *Ann Nucl Med* 1991;5:19–27.
20. Frais MA, Botvinick EH, Shosa DW, et al. Phase image characterization of ventricular contraction in left and right bundle branch block. *Am J Cardiol* 1982;50:95–105.
21. Underwood SR, Walton S, Laming PJ, et al. Patterns of ventricular contraction in patients with conduction abnormality studied by radionuclide angiocardiography. *Br Heart J* 1984;51:568–574.
22. Botvinick EH, Frais MA, Shosa DW, et al. An accurate means of detecting and characterizing abnormal patterns of ventricular activation by phase image analysis. *Am J Cardiol* 1982;50:289–298.
23. Underwood SR, Walton S, Laming PJ, Ell PJ, Emanuel RW, Swanton RH. Quantitative phase analysis in the assessment of coronary artery disease. *Br Heart J* 1989;61:14–22.
24. Schwaiger M, Ratib O, Henze E, Schelbert HR. Limitations of quantitative phase analysis of radionuclide angiograms for detecting coronary artery disease in patients with impaired left ventricular function. *Am Heart J* 1984;108:942–949.
25. Ratib O, Henze E, Schon H, Schelbert HR. Phase analysis of radionuclide ventriculograms for the detection of coronary artery disease. *Am Heart J* 1982;104:1–12.
26. Bolli R. Myocardial "stunning" in man. *Circulation* 1992;86:1671–1691.
27. Mathias P, Kerin NZ, Blevins RD, Cascade P, Rubenfire M. Coronary vasospasm as a cause of stunned myocardium. *Am Heart J* 1987;113:383–385.
28. Takatsu F, Suzuki A, Nagaya T. Variant angina pectoris with prolonged electrical and mechanical stunning. *Am J Cardiol* 1986;58:647–649.
29. Fournier C, Boujon B, Hebert JL, Zamani K, Grimon G, Blondeau M. Stunned myocardium following coronary spasm. *Am Heart J* 1991;121:593–595.
30. Dote K, Sato H, Tateishi H, Uchida T, Ishihara M. Myocardial stunning due to simultaneous multivessel coronary spasms: a review of five cases. *J Cardiol* 1991;21:203–214.
31. Bonow RO, Bacharach SL, Green MV, et al. Impaired left ventricular diastolic filling in patients with coronary artery disease: assessment with radionuclide angiography. *Circulation* 1981;64:315–323.
32. Serruys PW, Wijns W, Brand MVD, et al. Left ventricular performance, regional blood flow, wall motion and lactate metabolism during transluminal angioplasty. *Circulation* 1984;70:25–36.
33. Wijns W, Serruys PW, Slager CJ, et al. Effect of coronary occlusion during percutaneous transluminal angioplasty in humans on left ventricular chamber stiffness and regional diastolic pressure-radius relations. *J Am Coll Cardiol* 1986;7:455–463.
34. Williamson BD, Lim MJ, Buda AJ. Transient left ventricular filling abnormalities (diastolic stunning) after acute myocardial infraction. *Am J Cardiol* 1990;66:897–903.