《原 著》

Peak Systolic Pressure-volume Relationships in Man: Noninvasive Determination by Equilibrium Gated Radionuclide Angiocardiography and Cuff Sphygmomanometry

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Abstract We determined the relationship between left ventricular (LV) peak systolic pressure (PSP) and end-systolic volume, non-invasively using cuff sphygmomanometry and radionuclide angiocardiography (RNA). Systolic blood pressure (SBP) measured in the arm was substituted for PSP. LV end-diastolic volume (EDV), end-systolic volume (ESV) and ejection fraction (EF) were determined by a non-geometric method of gated RNA and recorded in three different hemodynamic states: at rest (basal state), during increased SBP after angiotensin administration (initial dose, 1–2 μg/min) and during decreased SBP after nitrate (sublingual nitroglycerin, 0.3–0.6 mg, or intravenous isosorbide dinitrate, 0.5–1.0 mg/min). The reproducibility of this method, tested in six subjects, proved to be good. Fifty-five subjects were divided into four groups based on EF at rest. The EDV and ESV were increased by angiotensin, and decreased by nitrate in all groups. EF was decreased by angiotensin and increased by nitrate. In contrast, the changes in PSP/ESVI due to these drugs remained in a narrow range in all groups. The regression lines of the PSP/ESVI relationship were almost linear and were steeper in the group with higher EF. E_{max}, the slope of the lines, was 5.75 ± 3.48 mmHg/ml/m^2 in group 1 (EF$>$50%), 3.16 ± 1.83 mmHg/ml/m^2 in group 2 (EF 49–40%), 2.27 ± 0.86 mmHg/ml/m^2 in group 3 (EF 39–30%) and 0.59 ± 0.50 mmHg/ml/m^2 in group 4 (EF$<$29%). There were good correlations between E_{max} and EDVI at rest (hyperbolic function) and between E_{max} and EF at rest (exponential function). The theoretical volume at zero pressure (VoI) did not meet in a definite value and was not related to EF at rest. Thus, the left ventricular peak systolic pressure-end-systolic volume relationship can be assessed non-invasively from radionuclide angiocardiography, which can be widely used for the evaluation of ventricular contractility, even in patients with asynergic ventricular contraction for whom echocardiography is unsuitable in measuring ventricular volume.

Introduction

Cardiac performance is maintained by a balance of four major determinants: heart rate, preload, afterload and contractility. When this balance is disturbed, heart failure results. The severity of heart failure can be assessed by measuring these four parameters. Contractility is the primary parameter representing cardiac muscle function, consequently ventricular pump function; so the observation of contractility is useful and important in assessing heart failure. Of the many indices of contractility, the ejection fraction is the most commonly used, probably because it can be measured relatively easily. The ejection fraction is, however, reported to be considerably affected by changes in heart rate[1], preload[2] and afterload[3]. Recently, as a more sensitive index of contractility, the end-systolic pressure-volume

* The Second Department of Internal Medicine, Kyoto Prefectural University of Medicine,
受付：59年7月16日
最終稿受付：59年9月20日
別刷請求先：京都市上京区河原町広小路 (〒602)
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relationship has been proposed[4,5]. This is attractive because it is less affected by preload and afterload and varies directly with alterations in the myocardial contractile state[6,7]. However, it has not yet been thoroughly evaluated in human subjects, mainly because of the difficulty of measuring intraventricular pressure and ventricular volume by a non-invasive procedure.

Radionuclide left ventricular (LV) volume determination is non-invasive and independent of the LV geometry. The echocardiographic approach is also non-invasive but has geometric problems.

Purpose of this study is to evaluate the LV end-systolic pressure-volume relationships non-invasively in man with non-geometric radionuclide angiocardiography and cuff sphygmomanometry to determine volume and pressure. The validity of the method and the linearity of the peak systolic pressure (PSP) and end-systolic volume (ESV) relationship are investigated. A further study was undertaken to determine the correlation between E_{max}, the slope of the regression line of the PSP-ESV relationship, and other variable of left ventricular function, and to assess the theoretical value of end-systolic volume extrapolated to zero systolic pressure (dead volume, VoI).

Subjects

Fifty-five subjects were studied with multigated equilibrium blood pool imaging. The patients were divided into four groups on the basis of their left ventricular (LV) ejection fraction (EF) at rest. Group 1 was composed of 15 patients with an EF of more than 50%, aged 60±8 (mean±1 SD) years. Group 2 was composed of 11 patients with an EF of 40–49%, aged 65±15 years. Group 3 was composed of 13 patients with an EF of 30–39%, aged 62±12 years. Group 4 was composed of 16 patients with an EF of less than 29%, aged 63±11 years.

Seventeen patients had had a myocardial infarction (7 in Group 3, 10 in Group 4). One patient in group 4 had dilated cardiomyopathy. No patient had congenital cardiac malformation or valvular heart disease.

In our laboratory, the lower limit of the EF in subjects older than 60 years who have no heart disease is 50%; this may be lower than that in other institutions. Almost all patients in group 1 (EF>50%) had normal cardiac function, if EF alone was used as the index of function.

Method

Radionuclide angiocardiography

Multigated equilibrium blood pool scintigrams were performed after in vivo labeling of red blood cells with 20 mCi of technetium-99m sodium pertechnetate by the method described previously[7]. Data were collected with a gamma scintillation camera (Searle Pho/gamma 4) equipped with an all purpose, parallel-hole collimator and interfaced with an on-line computer system (Elscint, Dycom-80). Scintigrams were obtained in the left anterior oblique projection with the angle of obliquity adjusted to allow the clearest separation between the ventricles. Radionuclide data and electrocardiographic signals were acquired for 6–8 min, and 24 frames per cardiac cycle were obtained.

The first scintigraphy was performed at rest and without any drugs (control conditions). Then, intravenous infusion of angiotensin (Hypertensin, Ciba) was started at 1–2 μg/min. The dose was adjusted to increase systolic blood pressure (SBP) by approximately 20–30 mmHg and to maintain it at this level during the collection of data. The second scintigraphy was performed during angiotensin infusion without any positional change between subject and camera. After the blood pressure had returned to its control level upon cessation of the infusion of angiotensin, nitroglycerin was given sublingually to 40 patients in a dose of 0.3–0.6 mg. To avoid data collection during the extreme hemodynamic instability immediately after the administration of nitroglycerin, scintigraphy was started approximately 5 minutes later. The other 15 patients were given isosorbide dinitrate intravenously at a rate of 0.5–1.0 mg/min. The third scintigram was taken during the period of reduced SBP. Blood pressures were measured by the same investigator with a cuff sphygmomanometer on the right upper arm. At least three times during each scintigraphy, the blood pressure was measured, and the mean SBP was substituted for the peak systolic LV pressure (PSP) which is analogous to the end-systolic LV pressure.

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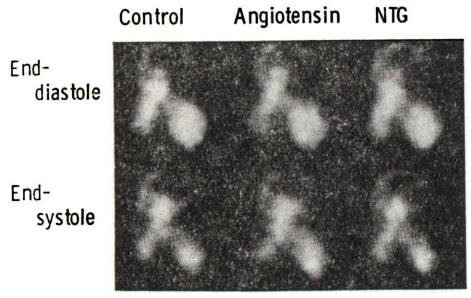

Fig. 1 End-diastolic and end-systolic ventricular images of a 53-yr-old patient with old antero-inferior myocardial infarction. In the control study (left row) systolic blood pressure (SBP) was 124 mmHg, end-diastolic volume (EDV) was 319 m*l*, end-systolic volume (ESV) was 236 m*l* and ejection fraction (EF) was 26%. With angiotensin (middle row), SBP, EDV and ESV were increased to 160 mmHg, 361 m*l* and 298 m*l*, respectively, and EF decreased to 18%. With nitroglycerin (right row), SBP, EDV and ESV were conversely reduced to 114 mmHg, 306 m*l* and 219 m*l*, respectively, but EF was increased to 28%. Peak systolic pressure (SBP) to ESV ratios remained constant (0.52, 0.53 and 0.52 mmHg/m*l* in control, angiotensin and nitroglycerin studies, respectively). E_{max} was 0.98 mmHg/m*l*/m^2 in this case.

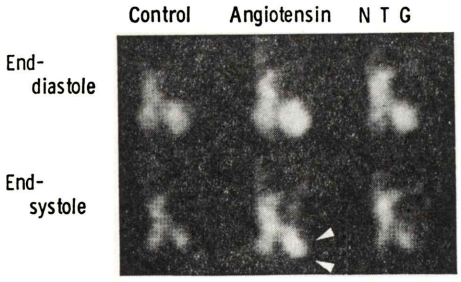

Fig. 2 End-diastolic and end-systolic ventricular images of a 66-yr-old patient with angina pectoris whose exercise electrocardiogram showed positive ST-T wave changes in left chest leads (V5–V6). In the control study (left row), SBP, EDV and ESV were 104 mmHg, 114 m*l* and 68 m*l*, respectively, and EF was 39% With angiotensin (middle row), SBP, EDV and ESV increased to 134 mmHg, 138 m*l* and 88 m*l*, respectively, but EF decreased to 36%. Note that asynergic contraction was enhanced by angiotensin in the postero-lateral region of the left ventricle (indicated by white triangles). With nitroglycerin (right row), SBP, EDV and ESV were reduced to 91 mmHg, 96 m*l* and 58 m*l*, respectively, and EF rose again to 40%. PSP/ESV remained almost constant (1.53, 1.52 and 1.56 mmHg/m*l* in control, angiotensin and nitroglycerin studies, respectively). E_{max} was 2.06 mmHg/m*l*/m^2 in this case.

ventricular volume by the non-geometric method, 10 m*l* of venous blood was drawn with a syringe at the mid-point of the scintigraphy and placed in a spherical container (approximate capacity=90 m*l*), which was then filled with water and shaken for adequate mixing. The container with venous blood was placed under the scintillation camera at the same distance at which the LV data were collected (the depth from the surface to the center of the LV cavity was constantly estimated to be 9 cm), and the radioactivity of the blood was measured.

A left ventricular time activity curve was derived from a region of interest assigned on the LV in the end-diastolic frame. The edge of the LV blood pool was determined as the isocount border of 70% of the maximum activity within the LV. The background region was defined as a postero-lateral 90° crescent, 4–5 pixels wide and separated from the edge of the LV by 4–5 pixels. End-diastolic counts (EDC) and end-systolic counts (ESC) were obtained after the background correction, and LVEF was calculated by the formula; LVEF=(EDC−ESC)/EDC.

Determination of ventricular volume

LV volumes were determined by a non-geometric technique based on the concept that the distribution of the tracer is identical to that of the blood because the tracer is uniformly mixed, and that changes in regional radioactivity are then proportional to changes in regional blood volume.

The following equation was used for the determination of LV volumes;

$$\frac{\text{EDC or ESC} \times \text{Number of frames (24)} \times \text{Blood volume (10)}}{\text{Total cardiac cycle time} \times \text{Blood activity} \times \text{DF} \times \text{AF}}$$

where total cardiac cycle time equals actual number of heart beats multipled by one cardiac cycle time, DF (decay factor) is a factor correcting for

Table 1 Reproducibility of peak-systolic pressure-volume relationship

Patient		BP (mmHg)		HR	EDVI	ESVI	SVI	EF	PSP/ESVI
		syst.	diast.	(beat/min)	(ml/m²)	(ml/m²)	(ml/m²)	(%)	(mmHg/ml/m²)
N M	C1	92	52	65	85	50	35	41	1.85
	C2	96	56	66	82	47	34	42	2.04
K F	C1	93	59	70	67	29	38	56	3.20
	C2	95	60	70	70	29	42	59	3.33
Y S	C1	95	58	55	78	41	37	50	2.28
	C2	90	55	56	83	44	39	47	2.03
T Y	C1	162	94	84	87	49	37	44	3.29
	C3	155	89	83	84	48	37	45	3.27
S Y	C1	92	58	62	56	39	17	31	1.85
	C2	91	58	65	71	51	20	29	1.79
K A	C1	130	78	69	82	47	35	43	2.77
	C2	125	76	64	85	44	42	49	2.87
SEE		3.3	3.2	2.8	3.3	5.8	3.2	3.5	0.18

Control data were compared before and after angiotensin or nitroglycerin administration. Abbreviations: BP=Blood pressure in arm; HR=Heart rate; EDVI=End-diastolic volume index; ESVI=End-systolic volume index; SVI=Stroke volume index; EF=Ejection fraction; PSP=Peak systolic pressure; C1=First control; C2=Second control after angiotensin; C3=Second control after nitroglycerin; SEE=Standard error of the estimate in two control studies.

isotope decay from the time of LV data acquisition to the time of venous blood counting, and AF is a factor correcting for the attenuation of radioactivity by the tissues from the LV to the chest surface. AF was 0.278, which was derived from comparison with volumes obtained by first-pass radionuclide angiocardiography. Using this method, we found an excellent correlation (r=0.91) between scintigraphic and angiographic LV volume measurement.

Calculations

The ratio of PSP to end-systolic volume index (ESVI, ESV normalized for body surface area) was calculated under the control conditions and after drug administration. The three points of PSP-ESVI (with PSP on the ordinate and ESVI on the abscissa) relationships were subjected to linear regression analysis, which yielded the slope, E_{max}. By solving the regression equation for PSP=0, the theoretical volume at zero presssure was calculated as the dead volume index (VoI). The relationship of E_{max} to the EF and to the end-diastolic volume index (EDVI) at rest was calculated as an exponential and a hyperbolical function, respectively.

Reproducibility of the method

To examine the validity of measuring pressure-volume relationships by this method, we compared control data obtained before and after the administration of angiotensin or nitroglycerin. The results of tests in six subjects are shown in Table 1. The standard error of the estimate (SEE) was 3.5% for EF, 3.3 mmHg for PSP (SBP), 5.8 ml for ESVI and 0.18 mmHg/ml/m² for PSP/ESVI. None of the parameters in the two control data were significantly different ($p<0.05$).

Results

Linearity of pressure-volume regression line

The linearity of the PSP-ESVI relationship was examined in 50 consecutive cases. The correlation coefficient (least squares fit in three points of PSP-ESVI) was more than 0.990 in 21 cases (42%) and more than 0.930 in 42 cases (84%).

Hemodynamic data and left ventricular volume

During angiotensin infusion, SBP rose by 22–30 mmHg and the heart rate (HR) decreased slightly (by 1–3 beats/min), whereas during nitrate administration, SBP decreased by 4–11 mmHg and HR increased by 2–6 beats/min (Table 2). Both EDVI

Table 2 Hemodynamic data and peak-systolic pressure-volume relationship

	Syst. BP (mmHg)	HR (beats/min)	EDVI (ml/m²)	ESVI (ml/m²)	SVI (ml/m²)	EF (%)	PSP/ESVI (mmHg/ml/m²)	VoI (ml/m²)	E_{max} (mmHg/ml/m²)
Group 1 (n=15, Age=60±8 yrs)									
C	121±22	69± 9	68.3±18.2	30.1± 9.8	38.3±10.2	56.6±6.0	4.71±2.51		
A	149±16	66± 9	76.0±24.2	38.1±15.0	37.4±10.8	49.8±7.6	4.77±2.52		
N	117±15	76±10	56.7±14.2	22.3± 5.88	34.9±10.0	60.6±6.3	5.85±2.40		
								−5.0±18.8	5.75±3.48
Group 2 (n=11, Age=65±15 yrs)									
C	142±33	71±15	76.7±17.0	43.4± 9.9	33.2± 8.1	44.0±2.8	3.39±0.85		
A	169±25	70±17	92.1±23.4	57.2±15.8	34.8± 9.3	38.0±4.6	3.33±1.19		
N	131±21	73±15	71.7±16.6	38.0± 9.33	33.7± 8.2	47.0±3.6	3.52±1.08		
								−17.3±26.2	3.16±1.83
Group 3 (n=13, Age=62±12 yrs)									
C	128±30	72± 9	78.0±19.9	50.2±13.2	29.2± 7.2	35.7±2.7	2.62±0.48		
A	158±27	70± 9	92.9±24.2	66.2±18.4	28.6± 7.7	29.0±3.6	2.52±0.53		
N	120±13	80±12	73.3±18.8	45.6±11.1	27.6± 8.5	37.5±3.8	2.67±0.55		
								−16.4±21.3	2.27±0.86
Group 4 (n=16, Age=63±11 yrs)									
C	114±22	73±11	163.0±59.1	133.2±55.8	29.8±12.9	19.9±7.7	1.05±0.56		
A	141±28	72±10	188.3±59.2	160.0±55.4	28.3±11.8	15.7±5.6	0.98±0.47		
N	103±16	79±13	141.5±48.2	112.7±44.2	28.8±10.7	21.5±6.3	1.04±0.58		
								−10.5±51.9	0.59±0.50

Abbreviation: BP=Blood pressure; HR=Heart rate; EDVI=End-diastolic volume index; ESVI=End-systolic volume index; SVI=Stroke volume index; EF=Ejection fraction; PSP=Peak systolic pressure (=systolic BP in arm); VoI=Dead volume index; C=Basal hemodynamic state (Control); A=Angiotensin administration; N=Nitroglycerin administration. Values given are mean±1 SD.

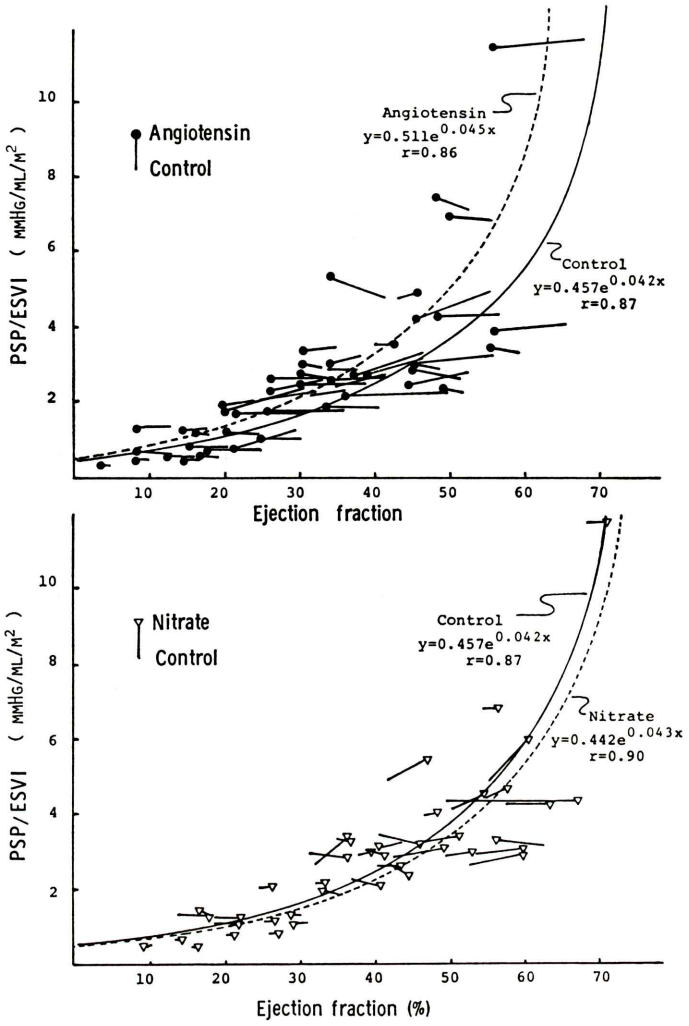

Fig. 3 Relationship between peak systolic pressure-end-systolic volume index ratio (PSP/ESVI) and ejection fraction (EF). EF is increased by angiotensin (upper graph) and decreased by nitrate (lower graph).

and ESVI were increased by angiotensin and decreased by nitrate in all groups. The changes in LV volume caused by these drugs were visible on the display system of the computer (Figs. 1, 2). EF under control conditions was 56.6±6.0% (mean± 1 SD) in Group 1, 44.2±2.8% in Group 2, 35.7± 2.7 in Group 3 and 19.9±7.7% in Group 4; and in all groups EF was increased by nitrate and decreased by angiotensin (Table 2).

End-systolic pressure-volume relations

PSP/ESVI under control conditions was 4.71± 2.51 mmHg/m*l*/m² (mean±1 SD) in Group 1, 3.39±0.85 mmHg/m*l*/m² in Group 2, 2.62±0.48 mmHg/m*l*/m² in Group 3 and 1.05±0.56 mmHg/ m*l*/m² in Group 4, and remained within a narrow range during angiotensin or nitrate administration (Table 2). The correlation between PSP/ESVI and EF was exponential ($PSP/ESVI=0.45e^{0.042EF}$, $r=0.87$, in controls; $PSP/ESVI=0.511e^{0.045EF}$, $r=0.86$, in the angiotensin treated group; $PSP/ESVI=0.422e^{0.043EF}$, $r=0.90$, in the nitrate treated group, Fig. 3). The change in EF was much less

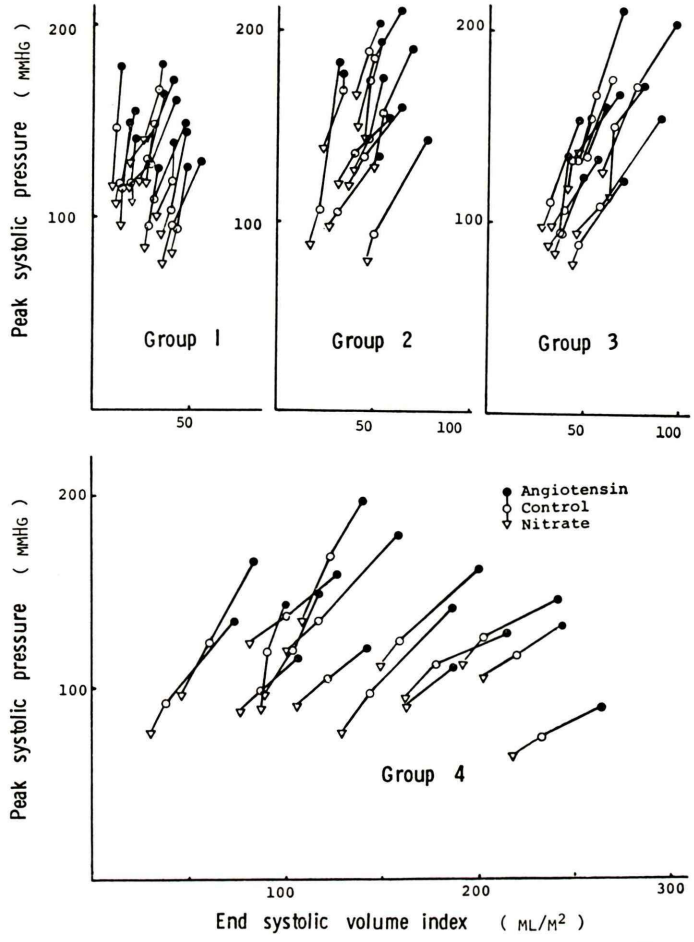

Fig. 4 Peak systolic pressure-volume relationship in the four groups. The regression lines of the group with a high EF is at the left of the graph, and their slope is steeper.

than in PSP/ESVI caused by alterations in the afterload (Fig. 3). The regression lines of the PSP-ESVI were almost linear. The lines in the groups with higher EF were steeper and situated more to the left in the PSP-ESVI graph (Fig. 4). The slope of the regression line, E_{max}, was 5.75 ± 3.48 mmHg/ml/m^2 (mean$\pm$1 SD) in Group 1, 3.16 ± 1.83 mmHg/ml/m^2 in Group 2, 2.27 ± 0.86 mmHg/ml/m^2 in Group 3 and 0.95 ± 0.50 mmHg/ml/m^2 in Group 4 (Table 2). E_{max} in the patients with old myocardial infarction was 1.25 ± 0.56 mmHg/ml/m^2.

There were good correlations between E_{max} and EDVI at rest ($E_{max}=110.6/(EDVI-24.9)$) and between E_{max} and EF at rest ($E_{max}=0.38e^{0.045EF}$, $r=0.801$) (Fig. 5). The relationship of PSP/ESVI in the basal hemodynamic state (controls) to E_{max} was also good (PSP/ESVI$=0.651\ E_{max}+0.958$, $r=0.841$, $p<0.001$).

Dead volume

The intercept of the PSP-ESVI line on the volume axis (VoI) did not coincide with one point and fell into a positive or negative value. The VoI was -5.0 ± 18.8 ml/m^2 (mean$\pm$1 SD) in Group 1, -17.3 ± 26.2 ml/m^2 in Group 2, -16.4 ± 21.3 ml/m^2 in Group 3 and -10.5 ± 51.9 ml/m^2 in

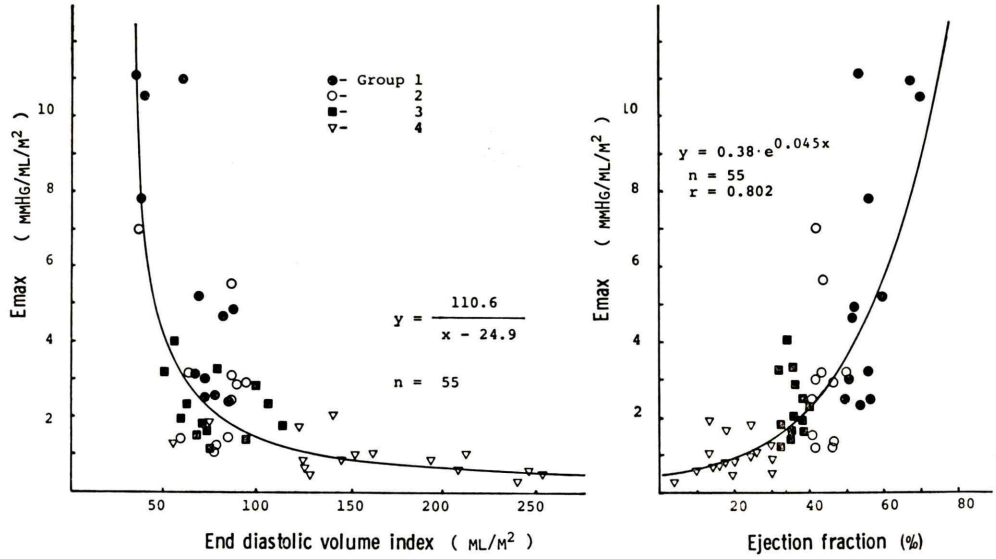

Fig. 5 Relationships between E_{max} and end-diastolic volume index (left graph) and between E_{max} and ejection fraction (right graph).

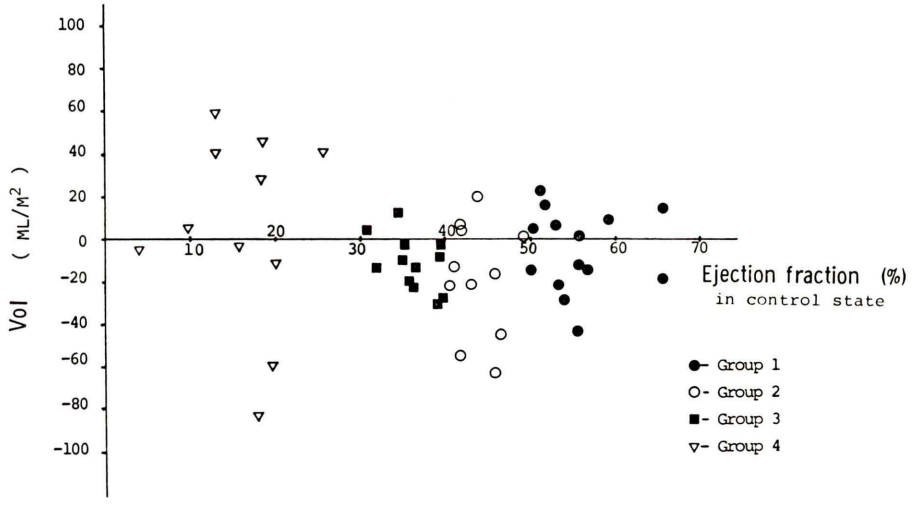

Fig. 6 Plot of the intercept of the regression line on the volume axis (VoI) against ejection fraction (EF) in the control state. No significant correlation was found between VoI and EF in any group.

Group 4 (Table 2). There was no significant relationship between VoI and EF in the basal hemodynamic state (Fig. 6).

Discussion

On the basis of animal studies, several investigators[8-10] have proposed that the LV end-systolic pressure-volume relationship is useful in evaluating LV contractility. In 1977 Grossman et al[11] investigated this relationship in man and concluded that the results in man were consistent with animal studies performed under a variety of experimental

conditions and supported the potential usefulness of the end-systolic pressure-volume relationship as an index of myocardial contractivility in man. More recently (1981), Meheml et al.[12] studied this relationship with monoplane left ventriculography and pharmacologic intervention and concluded that the end-systolic pressure-volume relation was linear in the physiologic range of the human left ventricle, that the use of peak systolic pressure instead of end-systolic pressure gave equally good results, and that the intercept of this relationship on the abscissa was unable to separate impaired from normal left ventricular function.

Although the importance of the end-systolic pressure-volume relationship is definite, it is rarely used clinically, because the measurement of LV pressure and volume requires invasive techniques. Therefore we tried non-invasive methods of determining the end-systolic pressure-volume relationship using LV volume obtained by gated radionuclide angiocardiography and blood pressure in the arm by cuff sphygmomanometry. The results seemed to be valid and suggest that this non-invasive method can be substituted for the invasive method.

The measurement of LV volume should be useful in the assessment of cardiac function. Single and biplane contrast ventriculography have undergone intensive mathematical analysis by many investigators who have shown good correlations between actual and calculated volumes using a prolate ellipsoid model. However, one difficulty of such a model is the validity of the assumptions about LV geometry during contraction, and additional concerns have included pathologically induced geometric changes, such as aneurysms and wall motion disturbances[13], that do not conform to the prolate ellipsoid model. Furthermore, contrast ventriculography is an invasive procedure.

The echocardiographic determination of LV volumes is not invasive, but relies heavily on assumptions that relate the long and short axes, and is particularly susceptible to geometric problems[14,15]. However, despite the uncertainty of geometric principles in the determination of ventricular volumes, echocardiography has been used widely for the determination of LV volumes and then for the evaluation of the end-systolic pressure-volume relationship[16,17].

The LV volume can be determined by radionuclide scintigraphy in three ways. The geometric method utilizes the area-length geometric formula in contrast ventriculography[18-21], but a small error in the measurement of area or length causes a substantial alteration in the calculated ventricular volume, and accurate detection of the ventricular edge is often indistinct because of limited imaging quality. The second approach is a non-geometric method with radionuclide tracer dilution curves[22-25], but it has not been used widely because it is not suitable for serial measurements and has inherent errors and problems in clinical application[26]. The third approach is another non-geometric method using equilibrium gated blood pool scintigrams. The concept of this technique is based on the fact that the count rate from the left ventricle is convertible to volumes with the use of an analogous relationship to the radioactivity measured from a known volume of peripheral blood[27-29]. This scintigraphic volume determination is independent of the geometry of the LV cavity and is easily repeatable both at rest and during all investigations. With our method, which belongs to the third approach to obtaining LV volumes, there was an excellent correlation between scintigraphic and cineangiographic results with a correlation coefficient of 0.91, which is lower, however, than the 0.98 of Slutsky et al.[27], the 0.985 of Dehmer et al.[28] and the 0.95 of Links et al.[29]

Because the time of end-systole is often obscure, some investigators[12,16] use the dicrotic notch pressure in the artery for end-systolic pressure, and some[11,30] use peak systolic pressure in the ventricle or aorta. To obtain the end-systolic pressure-volume relationship as proposed originally by Suga and Sagawa[31], the pressure data should be recorded exactly at end-systole or at least simultaneously with the ventricular volume. However, the precise measurement of ventricular pressure requires invasive techniques. Therefore, in our study the pressure in the arm was measured by the sphygmomanometer with the assumption that the systolic areterial pressure is almost equal to the peak ventricular pressure. Matsuzaki et al.[32] reported a close correlation ($r=0.99$) between peak systolic pressure in the arm obtained by sphygmomanometry and the LV peak systolic pressure

recorded during cardiac catheterization.

Linearity in the end-systolic pressure-volume relationship is a point of discussion. Suga and Sagawa showed that in the canine left ventricle, the relationship can be represented by a straight line at pressures between 50 mmHg and 150 mmHg[6,31]. In the human left ventricle, a linear relationship has been reported in physiologic pressure ranges[12,16,17]. On the other hand, Fujiyama et al.[33] reported the superiority of quadratic regression analysis in canine LV end-systolic pressure-diameter relationship. Our study showed that in eight (16%) of our 50 consecutive cases the correlation coefficient was less than 0.930 by linear regression analysis. This lower correlation may be due partly to the insensitivity of this non-invasive technique in measuring LV pressure or volume and may not indicate the inadequacy of linear regression analysis.

The measurement of E_{max} needs at least two pressure-volume data points from each patient under a constant inotropic background, but it is not easy to change afterload or to obtain multiple pressure-volume data points. Since the dead volume appears to be small and tends to meet in a definite value, attempts have been made to obviate alterations of afterload and to substitute the ratio of pressure to volume in one data point E_{max}[30,34,35]. Although in our study E_{max} correlated well with PSP/ESVI in the basal state, the dead volume (VoI) showed a wide variation, and Mehmel et al. also reported diverse dead volumes ranging from positive to below zoro, so, as Sagawa[36] warned, it may be important to acquire at least two pressure-volume data points from each patient.

In conclusion, the determination of ventricular volume by radionuclide is less affected by the geometry of the ventricle and can be used even in the presence of asynergy. The PSP-ESV relationship can be assessed non-invasively by radionuclide angiocardiography and cuff sphygmomanometry with methodological validity and linearity. The PSP/ESVI ratio was less affected by the alteration of afterload than was EDVI, ESVI or EF. E_{max}, the slope of the regression line, was correlated well with EDVI and EF. VoI, the dead volume, did not appear to be a sensitive index of ventricular function. The non-invasive evaluation of the PSP-ESV relationship by radionuclide is useful in clinical practice.

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要　旨

平衡時心プールシンチグラフィーによる収縮期末圧と容積関係の非侵襲的測定

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収縮期末の圧 (PSP)・容積 (ESV) 関係は収縮性を反映する新しい指標として注目されている．ESV を平衡時心プールシンチグラフィーで求め，PSP を上腕での収縮期血圧で代用して，この関係を非侵襲的に測定する方法を検討した．

アンギオテンシンによる昇圧で EDV, ESV は増大，駆出率 (EF) は低下し，亜硝酸剤による降圧で EDV, ESD は減少，EF は増大したが，PSP/ESV の変動は僅少であった．PSP/ESV 関係はほぼ直線に回帰可能で，この傾斜 E_{max} は心機能低下群で低値をとった．

PSP/ESV, E_{max} などの指標は前・後負荷に影響の少ない指標であり，これらの測定に，左室形態の影響の少ない RI による本法は非侵襲的心機能評価法として有用と考えられた．

Key words: Radionuclide angiocardiography, Peak systolic pressure, End-systolic volume, Pressure-volume relation, E_{max}.