



Title	Increased Atrial Contribution to Ventricular Filling in Ischemic Heart Disease : Non-invasive Measurement by ECG-gated Radiocardiography
Author(s)	SAKURAI, Tsunetaro
Citation	Japanese Circulation Journal, 41(11), 1231-1236 https://doi.org/10.1253/jcj.41.1231
Issue Date	1977-11
Doc URL	http://hdl.handle.net/2115/30354
Type	article
File Information	igaku_1231-1236.pdf



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TSUNETARO SAKURAI, M.D.

Japanese Circulation Journal
Vol. 41, No. 11, November 1977
(Pages 1231—1236)

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Non-invasive Measurement by ECG-gated Radiocardiography

TSUNETARO SAKURAI, M.D.

ECG-GATED radiocardiography (ECG-gated RCG) is a non-invasive method to record the volume change of the heart by an averaging of precordial radioactivity synchronous with an ECG, using an on-line minicomputer! When combined with ordinary radiocardiography (RCG), from which cardiac output and other parameters are derived, the rate of the volume change of the heart (dV/dt) can be calculated. We have reported the characteristic decrease of peak diastolic dV/dt (PDdV/dt) in ischemic heart disease? In this paper, measurement of the contribution of atrial contraction to ventricular filling by ECG-gated RCG is reported and the abnormal pattern of diastolic filling in ischemic heart disease will be discussed.

MATERIAL AND METHOD

A total of 112 subjects (Table I, 37 controls without significant heart disease, 43 hypertensives with a diagnosis of essential hypertension, 27 with ischemic heart disease (IHD) including 11 with myocardial infarction, and 5 with hypertrophic cardiomyopathy (HCM) diagnosed by cardiac catheterization and/or echocardiography) were tested by routine RCG and consecutive ECG-gated RCG. Routine RCG was performed with an intravenous injection of 50–100 μ Ci of I-131 radioiodinated human serum albumin (RIHSA), and the precordial dilution curve was

recorded. The cardiac output and other parameters were calculated by the analog simulation method as reported elsewhere.³

ECG-gated RCG was performed after the routine RCG was over, when the injected RIHSA had distributed uniformly in the circulating blood. In ECG-gated RCG, precordial radioactivity was continuously recorded for 800 heart

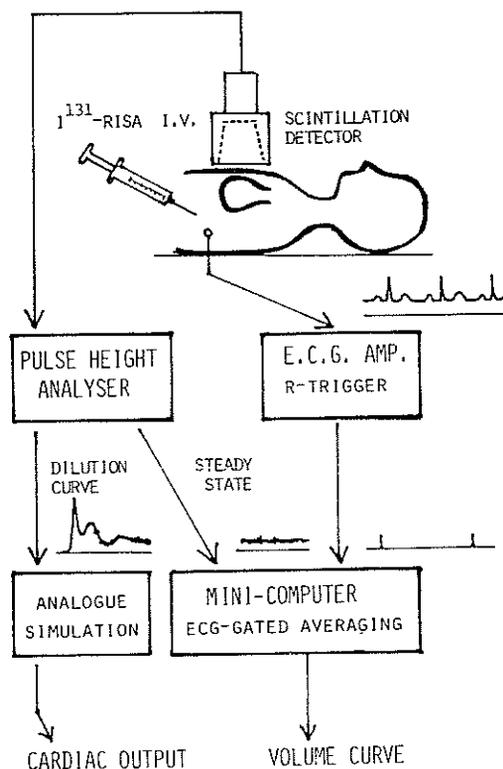


Fig. 1. The Blockdiagram of the routine RCG and the ECG gated RCG.

Key Words:

Atrial contribution
Diastolic compliance
Ischemic heart disease
Radiocardiography
ECG-gated averaging
Computer

(Received on April 16, 1977; Accepted on July 12, 1977)

The 3rd Division, Department of Internal Medicine Faculty of Medicine, Kyoto University, Kyoto, Japan

TABLE I COMPARISON OF THE c/b RATIO AND $PDdV/dt$ IN ALL SUBJECTS EXAMINED

Group	n	Age	c/b (%)	$PDdV/dt$ (ml/100 msec·M ²)
Control	37	34 ± 12	16.1 ± 5.0	24.9 ± 5.0
Hypertensive	43	55 ± 13**	26.5 ± 8.0**	17.0 ± 5.9**
Ischemic H. D.	27	60 ± 10**	37.5 ± 7.7**	11.7 ± 2.6**
Hypertrophic Cardiomyopathy	5	38 ± 14	18.6 ± 7.5	19.6 ± 4.6
Total	112	49 ± 16	22.8 ± 9.8	19.2 ± 6.5

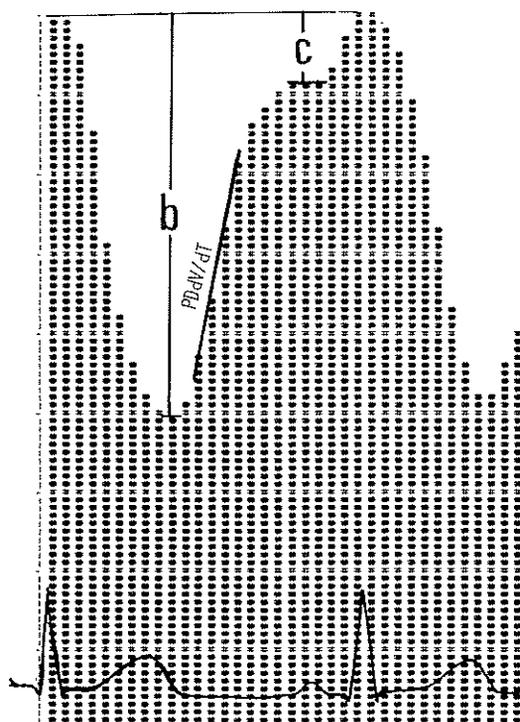
** $p < 0.001$ (mean ± s.d.)

Fig. 2. The volume curve recorded by ECG-gated RCG in a 43 year-old male of the control group. Designation of the parameter is demonstrated.

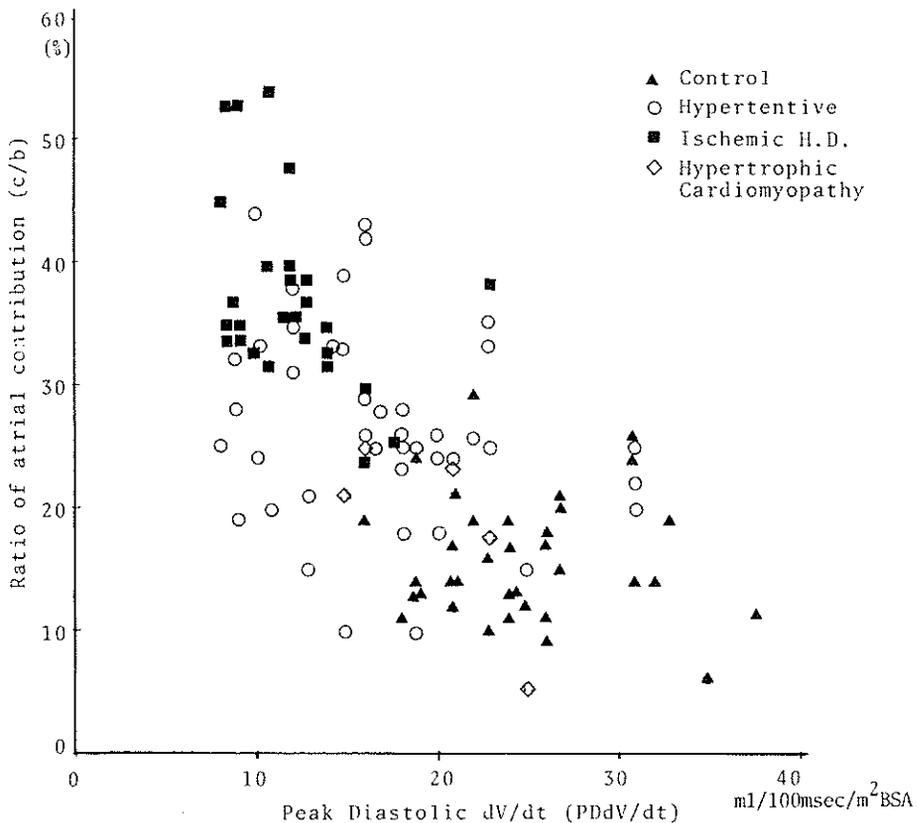
cycles by a 2-inch NaI scintillation detector with 6 x 9 x 5 collimator and every pulse from the pulse height analyser was fed into the memory of a minicomputer (YHP 4100A with 8K core) as illustrated in Fig. 1. Every second R wave in the ECG of a bipolar chest lead was used as a trigger pulse and on-line repetitive accumulation of the radioactivity synchronous with the trigger pulse

was performed so that the change in activities in two successive cardiac cycles was recorded. After the test was over, serial radioactivity which represents volume change of the heart was graphically displayed in 40 millisecond intervals after a three point smoothing weighed 1-2-1. Instantaneous dV/dt was calculated every 40 milliseconds as a slope of the curve by a linear least square fitting of four successive points of the curve. Stroke index derived by routine RCG was used to calculate dV/dt from the slope, and the maximum value in early diastole was denoted as peak diastolic dV/dt ($PDdV/dt$, ml/100 msec·M² BSA). The details and validity of the method have been reported previously.² The ratio of the contribution of atrial contraction to ventricular filling (c/b) was measured from the height of the presystolic peak as illustrated in Fig. 2.

In some subjects, in whom the level of mid-diastolic plateau was difficult to define, the last 200 millisecond period of the mean R-R interval was conventionally used. Subjects who had arrhythmia of any kind, or heart rate over 80/min. during the test were excluded from the study as the presystolic peak was usually difficult to recognize in these subjects. Cardiac catheterization was performed in 7 subjects, 4 with IHD and 3 with HCM, within 2 weeks of the ECG-gated RCG. LV pressure was recorded by a catheter tip micro-transducer (Millar's 7F) which was calibrated by another water filled system connected through injecting holes at the same time. In 4 subjects, 3 with IHD and 1 with HCM, who underwent left ventricular cine-angiography, the volume curve was measured by manual tracing of the film (30 frames/sec.), using the single plane arealength method. The ratio of atrial contribution corresponding to c/b in ECG-gated RCG was measured from the curve.

TABLE II COMPARISON OF THE *c/b* RATIO AND PDdV/dt IN THE SUBJECTS BETWEEN 40 AND 50 YEARS OF AGE

Group	n	Age	<i>c/b</i> (%)	PDdV/dt (ml/100 msec·M ²)
Control	13	46 ± 5	15.3 ± 4.1	24.2 ± 5.0
Hypertensive	20	51 ± 6	25.0 ± 8.7**	18.3 ± 4.9**
Ischemic H. D.	11	53 ± 5*	35.6 ± 8.0**	12.0 ± 2.9**
Hypertrophic Cardiomyopathy	3	48 ± 5	23.0 ± 2.0*	16.7 ± 3.1

p* < 0.01 *p* < 0.001 (mean ± s.d.)Fig. 3. Scattergram of *c/b* and PDdV/dt in all subjects.

RESULTS

The values of the *c/b* ratio and PDdV/dt are listed in Table I. The *c/b* ratio is definitely increased in the hypertensive and IHD groups, more markedly in the latter, as compared to the control group. PDdV/dt shows a decrease in these two groups as in our previous observation? In Table II, where the possible influence of dif-

ference in age among the groups is eliminated by selecting subjects between 40 to 59 years of age, the increase in *c/b* ratio is still definite. In Fig. 3, in which all subjects are plotted with *c/b* in the ordinate and PDdV/dt in the abscissa, *c/b* ratio showed a negative correlation with PDdV/dt, the correlation coefficient being -0.62 . Subjects with IHD are clearly separated from the controls on the two dimensional plane. In subjects who

TABLE III DATA OBTAINED BY CARDIAC CATHETERIZATION AND ANGIO-CARDIOGRAPHY. THE *c/b* VALUES AND PDdV/dt BY ECG-GATED RCG WERE ALSO LISTED

Subjects	Age	Sex	LV pressure			Angiography			ECG-gated RCG	
			<i>sys/ed</i>	<i>m.d.</i>	<i>a.k.</i>	LVEDV	E.F.	<i>c/b</i>	<i>c/b</i>	PDdV/dt
			mmHg	mmHg	mmHg	ml	%	%	%	ml/100 msec·M ²
<i>Ischemic H. D.</i>										
T.T.	60	M MI	95/ 5	3	3	110	65	33	37	9
K.I.	56	M MI	149/16	6	12	165	48	46	43	9
F.H.	59	F MI	113/ 7	5	2	130	63	37	38	23
S.O.	52	M AP	156/ 8	6	2	104	79	*	34	9
<i>HCM</i>										
J.K.	42	M	102/ 7	4	4	140	71	22	23	14
K.F.	27	M	108/10	5	9	120	80	*	18	23
T.E.	19	F	101/14	7	9	81	68	*	6	25

sys/ed=systolic/end-diastolic, *m.d.*=mid-diastolic, *a.k.*=atrial kick, *E.F.*=ejection fraction
 *=cine-angiography was not done (LVEDV & E.F. measured by biplane angiography, 6 films/sec)
 MI=myocardial infarction, AP=angina pectoris, HCM=hypertrophic cardiomyopathy.

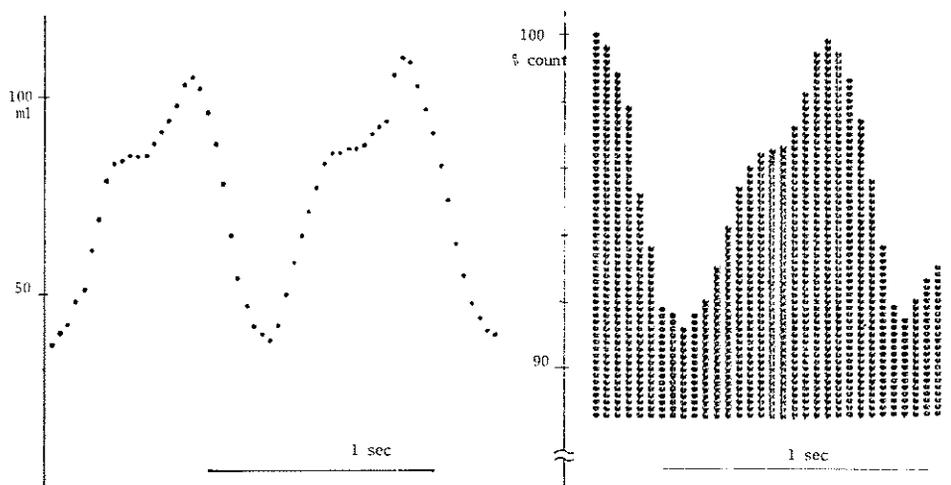


Fig. 4. Volume curve of the heart recorded by ECG-gated RCG (right) and that of left ventricle by cine-angiography. Case T. T., a 60 year-old male with myocardial infarction, in the Table III.

underwent cardiac catheterization, mid-diastolic pressure prior to atrial contraction and the height of the atrial kick were also measured from the pressure tracings. These data are presented in Table III with the *c/b* values measured by cine-angiography and ECG-gated RCG. In all 4 subjects in whom the volume curve was measured by the two methods, the two curves corresponded well in the pattern of the curve as well as the

values of *c/b* (Table III). In Fig. 4, an example of the two curves in good agreement was presented.

DISCUSSION

As the ECG-gated RCG is a record of radioactivity mainly of the ventricles, the presystolic peak in the record is reasonably attributed to the volume change by atrial contraction. As shown

in Fig. 4 and table 3, coincidence of the curves as well as the values of c/b measured by ECG-gated RCG and cine-angiography also support the validity of quantitative analysis by ECG-gated RCG. The ratio of atrial contribution to ventricular filling in man has been measured by the analysis of cine-angiography. The c/b ratio in normal subjects by ECG-gated RCG is very close to that reported by Hammermeister et al⁴ who also referred to its increase in IHD.

In the subjects with IHD, the definite decrease of $PDdV/dt$ and the increase of c/b ratio make the record of ECG-gated RCG easily distinguishable from that of normal subjects. As in the example in Fig. 2, one can expect a gentle slope in early diastole followed by a increased presystolic peak. In subjects with IHD who underwent cardiac catheterization, mid-diastolic pressure prior to atrial contraction was not elevated though they had decreased $PDdV/dt$ (Table III). The atrial kick was not prominent in these subjects except in one, while the c/b ratio was high. These findings suggest that a relatively large change of volume occurs with a small rise of pressure during atrial contraction and that the pressure-volume relationship, on which the ventricle works, does not shift upward. So, it seems to be reasonable to attribute the cause of the decrease of $PDdV/dt$ in IHD not to the increased passive compliance, which is unlikely in mid-diastole of the ventricle, but to the abnormality of the relaxation process itself.

Recently, abnormalities of the left ventricular relaxation in IHD has been reported by cine-angiographic analysis of the isovolumetric relaxation period. Gibson et al⁵ analysed segmental movements of LV wall and reported that abnormal inward movement occurred in the area supplied by diseased coronary arteries. He suggested that such a "delayed relaxation" or a change in cavity shape before the mitral opening would interfere with the rapid filling of the ventricle without any alterations in the passive elastic stiffness of the myocardium.

The results of this study is in agreement with his observation in the point that these changes are probably caused by the primary abnormalities of ischemic heart and that passive stiffness of the myocardium is not necessarily affected. In subjects with HCM, in contrast with IHD, normal $PDdV/dt$, normal c/b and increased pressure during atrial contraction (atrial kick) suggest that diastolic filling occurs normally and less volume is added by atrial contraction. Thus the

change in HCM can be expressed as a pressure overload to the atrium, while it is rather a volume overload in IHD.

In subjects with essential hypertension, the values of the c/b ratio and $PDdV/dt$ distributed in wide ranges as plotted in Fig. 3. These variations seem to be mainly due to the difference in the severity of the disease among the subjects selected. Wikstrand et al⁶ found, in his study of 35 hypertensive subjects of 50 year of age, that there was no one with both signs of left ventricular hypertrophy on orthogonal electrocardiography and either an increased 'a' wave ratio ($> 15\%$) or an abnormal atrial sound. He suggested the presence of two different forms of cardiac involvement as the result of hypertension, and the signs of decreased distensibility of the left ventricle during atrial contraction would disappear when left ventricular hypertrophy became evident. These changes might explain the wide variation of the c/b ratio and $PDdV/dt$ observed in the hypertensive subjects in this study, and further studies are necessary to clarify this point. It can be said that the diastolic abnormalities, which are closely related to the ischemic changes of the myocardium, should be analysed by serial determinations of the pressure-volume relationship throughout diastole, so that relaxation in early diastole and the passive elasticity during atrial contraction can be properly evaluated.

SUMMARY

1. A total of 112 subjects, including 37 controls, 43 hypertensives, 27 IHD and 5 HCM, were tested by routine RCG and ECG gated RCG.
2. The atrial contribution to ventricular filling (c/b ratio) was measured by ECG-gated RCG and a characteristic increase was found in IHD (37.5 ± 7.7 , mean \pm s.d.) and Hypertensives (26.5 ± 8.0) compared to controls (16.1 ± 5.0).
3. The increase in c/b ratio and the decrease in $PDdV/dt$ in IHD seemed to be caused by primary abnormalities in the relaxation of the ventricle.
4. ECG-gated RCG, being a non-invasive method, serves useful informations in estimating ischemic heart disease especially by a quantitative analysis of the diastolic filling.

Acknowledgement

The author is deeply indebted to Dr. Chuichi Kawai, Professor of Internal Medicine, and to Dr. Akina Hirakawa, Director of the Biomedical Information

Science, Kyoto University Hospital, for their encouragements and constructive advice throughout this study.

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