Clinical Considerations on the Duration of Left Ventricular Systoles

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Tension period, isometric contraction time and ejection time were measured in 20 normal subjects and 72 patients with nonvalvular heart disease to evaluate the usefulness of the duration of left ventricular systoles as an index of the ventricular performance. Left ventricular function could not be manifested with only one of those durations. The ratio of ejection time to isometric contraction time demonstrated a parallelism to changes in left ventricular performance. Serial observations of this ratio offer a convenient detection for the changes in left ventricular performance.

IT HAS BEEN well established that a ventricular performance is influenced by such factors as venous return, arterial resistance, physical and neurohumoral regulations, and ventricular systolic phases are modified by the changes of cardiac work¹⁻⁴. In 1920, WIGGERS demonstrated in animal that the increase in venous return tended to decrease the isometric contraction phase and increase the ejection phase, while the increase in arterial resistance induced a slight prolongation in the isometric contraction phase and a shortening in the ejection phase⁵. It is further noted that tachycardia and administration of sympathomimetic amines produce an abbreviation of mechanical systole⁶. REMINGTON and associates described that length of systole showed a good correlation with stroke volume and with left ventricular work in the dog⁷. In human beings as well, GOBBATO and MEDA in 1956⁸, and WEISSLER in 1961⁹, described a linear inverse relationship between heart rate and ejection period and in addition, a direct relationship between stroke volume and ejection period. Furthermore, several investigations have found the alterations of the duration of systolic phases in vari-

ous pathological conditions^{10, 11, 12}. Thus, as a variety of determinants of the duration of systolic phases was disclosed, the clinical usefulness of the measurement of systolic time intervals has been increasingly recognized.

It is, however, permitted that the effects of hemodynamic changes on the duration of cardiac cycle in diseased heart may not be quite identical to those in normal heart. In this respect, it has not fully been revealed whether or not the duration of left ventricular systoles could reflect always accurately the ventricular function in various heart diseases. The present investigation was undertaken to provide a further detailed analysis of the duration of left ventricular systoles in nonvalvular heart disease and to assess the advantages and limitations of the measurement of systolic time interval to clinical application.

MATERIALS AND METHODS

The studies were made in 20 normal subjects, 6 hypertensive patients without heart disease, 22 with arteriosclerotic heart disease (ASHD), 13 with hypertensive heart disease (HHD), 20 with arteriosclerotic-hypertensive heart disease (ASHHD), and 11 with left ventricular failure secondary to the

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above-mentioned disease (LVF). The criteria used for diagnosis were: for hypertension, arterial blood pressure persistently higher than 150 mmHg systolic and/or 90 mmHg diastolic with out any clinical and laboratory evidence of heart disease; for HHD, hypertension and a clinical and/or laboratory evidence of cardiomegaly; for ASHD, one or more of the following, 1) clinical and laboratory evidence of old myocardial infarction, 2) typical history of angina pectoris, 3) abnormal electrocardiogram compatible with myocardial ischemia associated with clinical findings of arteriosclerosis; for ASHHD, combinations of the criteria included in both HHD and ASHD. The patients with LVF were in class III or IV of functional classification of New York Heart Association, and were subdivided into normotensive and hypertensive group. They ranged in age from 19 to 76 years and averaged 56 years. Electrocardiogram, phonocardiogram and carotid arterial pulse tracing were simultaneously recorded on a direct writing, Mingograph Cardirex 31-B at a paper speed of 100 mm/sec. with 0.01 sec. time lines. A funnel-shape pick-up was manually put on the part to feel a carotid pulsation most strongly for deriving the carotid pulse tracing. The pick-up was attached to a piezo-electric transducer by an air-filled hard rubber tube. The phonocardiogram was recorded through medium filter with a microphone placed over the precordium to define best the first and second heart sounds. The standard limb lead II was selected in electrocardiogram. All observations were made in the supine position, while breath was held at the mid-inspiration, after bed rest for approximate 10

minutes. The cases in which the initial vibrations of the heart sounds were obscure or the incisural notch of the carotid tracings could not be clearly delineated were excluded from the present study.

The durations of left ventricular systoles indirectly determined were the following:

(1) The electromechanical systolic time ($Q-S_2$) was measured from the onset of the QRS complex to the first high frequency vibrations of the aortic component of the second heart sound.

(2) The mechanical systolic time (S_1-S_2) was measured from the high frequency component of the first heart sound to the onset of the second heart sound.

(3) The ejection time (ET) was measured from the onset of the upstroke to the dicrotic notch of the carotid pulse tracing.

(4) The tension period (TP) was obtained by subtracting ET from $Q-S_2$.

(5) The isometric contraction time (ICT) was calculated by subtracting ET from S_1-S_2 (Fig. 1).

All measurements were made to the nearest 0.005 sec. in each of ten consecutive beats and averaged. Heart rate was derived from dividing 60 by R-R interval of electrocardiogram.

The relationship between ejection time and stroke volume was studied in 13 patients of nonfailure group (HHD; 7, ASHD; 4, ASHHD; 2) and 3 patients with LVF. Cardiac output was determined by the indicator dilution method, with the medial cubital vein used for injection of indocyanine-green 5 mg. and the femoral artery for sampling through the cubette-densitometer.

Fig. 1 Simultaneous electrocardiogram, carotid pulse tracings and phonocardiogram available for measurements of tension period, isometric contraction time and ejection time.

In order to assess how the durations of left ventricular systoles reflect the cardiac contractility, the effects of deslanoside in a single dose of 1.2 mg on them were observed in 4 with HHD, 4 with ASHHD and 10 with LVF. Hemodynamic studies by the left heart catheterization were carried out in 6 nonfailure patients and 3 failure patients before and after digitalization. Recordings of intraventricular and arterial pressure were made with Nihon-koden pressure transducer and multi-channel direct writing recorder. Mean arterial pressure was obtained electronically. Mean systolic ejection rate (MSER), left ventricular stroke work (LVSW) and left ventricular stroke power (LVSP) were derived by standard methods.

RESULTS

1) Relationship between heart rate and left ventricular systolic phases.

The respective duration of $Q-S_2$, S_1-S_2 and ET decreased with the increase of heart rate. There was no significant difference between HHD, ASHD and ASHHD, when lengths of each systolic phase were plotted against the respective heart rates. The regression equations relating heart rate to $Q-S_2$, S_1-S_2 and ET are shown in Table I and Figure 2. The slopes of regression equations relating heart rate with S_1-S_2 and heart rate with ET were the same, while the slope of the regression equation between heart rate and $Q-S_2$ was more steeper.

Fig. 2. The relationship between heart rate and $Q-S_2$ (A), S_1-S_2 (B) or ET (C).

Closed circle: arteriosclerotic heart disease, open circle: arteriosclerotic-hypertensive heart disease, triangle: hypertensive heart disease.

TABLE I REGRESSION EQUATIONS RELATING HEART RATE WITH ELECTROMECHANICAL INTERVAL ($Q-S_2$), MECHANICAL INTERVAL (S_1-S_2) OR EJECTION TIME (ET), AND RELATING STROKE INDEX (SI) WITH EJECTION TIME

$Q-S_2$:	$Y = 0.536 - 0.002X$	$SD = \pm 0.016$	$(P < 0.001)$
S_1-S_2 :	$Y = 0.445 - 0.0015X$	$SD = \pm 0.016$	$(P < 0.001)$
ET :	$Y = 0.394 - 0.0015X$	$SD = \pm 0.015$	$(P < 0.001)$
SI :	$Y = 0.150 + 0.260X$	$SD = \pm 0.025$	$(P < 0.01)$

the heart diseases.

Tension period. The values were corrected to a standard heart rate of 70/min, based on the above-mentioned regression equations, since the average heart rate of the cases applied for the present study was near to 70/min.

Tension period was prolonged in heart disease group ($P < 0.001$). There was no significant difference between normal subjects and hypertensive patients without heart disease, and between HHD, ASHD and ASHHD respectively ($P > 0.5$). In normotensive group of heart failure, the mean value of tension period seemed to be more prolonged than in nonfailure group, but the difference, when statistically examined, is not significant ($P > 0.2$). In hypertensive group of heart failure, this period was significantly longer than in nonfailure group ($P < 0.05$).

Isometric contraction time. This was prolonged in heart disease group ($P < 0.001$). There was no difference between normal subjects and hypertensive patients without heart disease, and between HHD, ASHD and ASHHD respectively ($P > 0.5$). Moreover, it was not different between normotensive patients with LVF and heart disease group without LVF ($P > 0.5$). However, it was more prolonged in hypertensive patients with LVF than other heart disease groups ($P < 0.1$).

Ejection time. This was corrected to a standard heart rate of 70/min, based on the above-mentioned regression equations and compared

with each other. This time interval was not different between normal subjects and heart disease patients without LVF ($P > 0.5$). On the other hand, it was significantly shortened in failure patient (normotensive: $P < 0.1$, hypertensive: $P < 0.05$).

These data are summarized in Table II.

3) Relationship between stroke index and ejection time.

The cardiac output ranged from 3.1 L/min to 6.2 L/min in 13 patients of nonfailure group, and from 2.7 L/min to 3.6 L/min in 3 patients with LVF.

Before digitalization, the linear relationship was obtained between stroke index and ejection

Fig. 3. The relationship between stroke index and ejection time in nonvalvular heart diseases. Arrow lines show the post-digitalized changes in the same patients. Regression function was obtained with pre-digitalized values of stroke index and ejection time.

TABLE II COMPARISON OF TENSION PERIOD, ISOMETRIC CONTRACTION TIME AND EJECTION TIME AMONG VARIOUS HEART DISEASES

	Number of cases	Tension period	Isometric contraction time	Ejection time
Normal	20	95 ± 15	37 ± 8	290 ± 13
Hypertension	6	99 ± 23	36 ± 7	287 ± 15
HHD	13	116 ± 22	57 ± 14	287 ± 22
ASHD	22	117 ± 18	53 ± 9	282 ± 14
ASHHD	20	115 ± 22	54 ± 10	290 ± 13
CHF				
normotensive	5	128 ± 19	54 ± 5	263 ± 25
hypertensive	6	141 ± 12	65 ± 10	236 ± 25

Mean values and standard deviations are shown with msec. in order.

Tension period and ejection time are the values corrected for heart rate 70/min.

HHD: Hypertensive heart disease. ASHD: Arteriosclerotic heart disease.

ASHHD: Arteriosclerotic-hypertensive heart disease. CHF: Congestive heart failure.

time in nonfailure patients (Fig. 3). Further, in 3 patients with heart failure, there was no difference from the regression data of nonfailure group. The correlation between these two variables was highly significant ($P < 0.01$). Six patients of nonfailure group and 3 of failure group were rapidly digitalized with intravenous administration of deslanoside 1.2 mg; cardiac output increased in 3 of nonfailure group and all of failure group, and decreased in 3 of nonfailure group. However, stroke index increased in all patients, ranging from 2.8 per cent to 35.7 per cent. In 5 out of 6 of nonfailure group and all of failure patients, the changes of stroke index were distinctly larger than of ejection time, so that mean systolic ejection rate was increased after digitalization (Fig. 3).

4) The changes in the durations of left ventricular systoles after acute digitalization.

In heart disease group without LVF, the durations of tension period, isometric contraction time and ejection time were significantly shortened after digitalization. The shortening of the isometric contraction time was very marked ($P < 0.001$). However, Q-S₁ interval showed no significant change statistically ($P >$

0.5). These are shown in Figure 4.

In patients with LVF, tension period and isometric contraction time were shortened after digitalization. Q-S₁ interval and ejection time exhibited no consistent change and the difference before and after digitalization was not significant, when statistically examined ($P > 0.5$). However, the ratio of corrected ejection time to isometric contraction time was increased in all over cases, when signs and symptoms of heart failure disappeared with digitalization

Fig. 4. Post-digitalized changes in tension period, Q-S₁ interval, isometric contraction time and ejection time in heart disease patients without left ventricular failure. B: before, A: after.

TABLE III EFFECTS OF DIGITALIZATION ON LEFT VENTRICULAR SYSTOLIC PHASES IN CONGESTIVE HEART FAILURE

Case	Age	Sex	Diagnosis	NYHA class	Blood pressure (mmHg)	Heart rate (beats/min.)	TP (msec.)	ICT (msec.)	ET (msec.)
K. K.	68	male	ASHD	III	B 98/60	70	120	52	260
					A 110/62	68	100	29	258
K. N.	60	female	ASHD	III	B 110/70	91	150	50	210
					A 116/80	70	144	34	241
T. K.	65	male	ASHD	II	B 136/90	48	110	55	330
					A 120/84	54	100	45	330
Y. H.	72	male	ASHD	III	B 110/62	107	104	50	215
					A 118/68	94	92	35	224
M. T.	38	female	PMD	III	B 112/80	105	116	45	185
					A 106/76	80	110	29	217
S. O.	59	male	ASHHD	IV	B 190/126	73	138	50	180
					A 180/110	75	123	20	207
M. S.	42	male	HHD	IV	B 200/130	100	120	60	200
					A 190/118	82	114	60	249
S. Y.	63	female	ASHHD	IV	B 188/100	94	130	80	210
					A 180/86	71	140	75	230
Y. O.	46	male	HHD	IV	B 210/130	93	124	60	220
					A 190/120	77	107	42	237
S. K.	56	female	ASHHD	III	B 158/96	92	154	70	196
					A 140/70	75	142	50	193

Abbreviations; B: before digitalization, A: after digitalization. Tension period (TP) and ejection time (ET) are the measured values.

(Fig. 5, 6, Table III).

- 5) Relationship between the ratio of corrected ejection time to isometric contraction time (ETc/ICT) and hemodynamic functions after digitalization.

To assess whether or not the ratio of ETc/ICT can demonstrate an associated parallel change to an alteration in cardiac functions, the deviations in the ratio of ETc/ICT were correlated with the changes in MSER, LVSW and LVSP after digitalization. These results are summarized in Table IV and Figure 7.

After acute digitalization the increase in the ratio of ETc/ICT was shown in 5 out of 6 patients without heart failure and all failure patients, associated with the increase in MSER, LVSW and LVSP. In failure patients, the deviations from the predigitalized value were more marked in hemodynamic parameters than in the ratio of ETc/ICT. In contrast, in non-

Fig. 5. Post-digitalized changes in tension period, Q-S₁ interval, isometric contraction time and ejection time in failure patients; closed circle: normotensive patients, open circle: hypertensive patients. B; before, A: after.

Fig. 6. Post-digitalized change in the ratio of ejection time to isometric contraction time in heart disease patients without failure (left) and with failure (right). B: before, A: after.

failure patients, the percentile change in the ratio of ETc/ICT was larger than in hemodynamic parameters. One patients without LVF showed a decrease in the ratio of ETc/ICT as-

Fig. 7. Percentile changes in the ratio of ejection time to isometric contraction time (ETc/ICT ratio) on the abscissa and mean systolic ejection rate (MSER) (A), Left ventricular stroke work (LVSW) (B) and left ventricular stroke power (LVSP) (C) on the ordinate. Closed circle: non-failure patients. Open circle: failure patients.

sociated with a decrease in hemodynamic parameters. In general, the percentile change in the ratio of ETc/ICT showed a parallelism to the change in hemodynamic parameters.

DISCUSSION

Most investigations have revealed that the measurement of the durations of the left ventricular systoles would yield the valuable information as to the left ventricular performance in various heart diseases¹²⁻¹⁴ and that the indirect measurement with simultaneous recordings of electrocardiogram, phonocardiogram and carotid arterial pulse tracing could be satisfactory in deciding each duration of the cardiac cycle in man¹⁵. In the present study, therefore, the durations of tension period, isometric contraction time and ejection time in nonvalvular heart diseases were measured by the indirect method.

The duration of ejection time derived from the indirect measurement may exceed by a small interval the ventricular contraction period, since the contraction of the ventricle may cease before the incisura in carotid pulse

tracing; that is, outflow from the ventricle into the aorta may be maintained with inertia even if the pressure gradient between the ventricle and aorta is reversed at the end-systole. In addition, the isometric contraction time may be underestimated, since the slow initial phase of ventricular contraction would not be taken into account when the initial deflection of the first heart sound is taken as the onset of mechanical systole, but the errors would be so sufficiently small relative to the total duration of systole as to be negligible for comparative purpose.

Although the duration of left ventricular systole may be influenced by many factors, the heart rate has most evident influence in the supine position at rest in heart disease group without ventricular failure. The regression equations relating heart rate with $Q-S_2$, S_1-S_2 and ET in the present study were very close to those in normal subjects derived by WEISSLER and associates¹⁶. In heart disease group, it could not be clarified how the difference in sex influenced on the duration of ventricular systoles, although WEISSLER and associates noted that the average of ejection time in normal female

TABLE IV HEMODYNAMIC DATA BEFORE AND AFTER ACUTE DIGITALIZATION

Cage	Age	Sex	Diagnosis		mBP	HR	ICT	ETm	ETc/ICT	CI	SI	MSER	LVEDP	LVSU	LVSP
M. I.	30	M	JH	B	86.7	57	32	290	8.5	3.03	51.6	177.9	6.0	56.6	194.1
				A	90.0	55	30	304	9.4	3.07	55.7	183.2	5.0	67.9	233.2
Z. T.	20	F	JH	B	93.0	77	54	270	5.2	4.44	52.5	194.4	7.0	65.0	240.7
				A	95.0	81	22	248	12.0	5.57	68.4	275.8	8.0	81.0	326.6
H. U.	19	M	JH	B	110.0	108	36	210	7.1	3.97	36.8	175.2	5.0	55.2	262.9
				A	108.0	87	40	230	6.4	3.10	35.6	154.8	8.5	50.8	220.1
K. N.	46	M	ASHHD	B	125.0	59	56	318	5.3	3.31	56.0	176.1	6.0	95.0	298.7
				A	128.3	58	35	317	8.5	3.65	63.2	198.7	5.0	111.0	350.2
J. N.	70	M	ASHD	B	100.5	67	43	263	6.1	3.31	49.4	187.8	15.5	60.0	228.1
				A	102.0	62	32	265	8.0	3.18	51.3	193.6	8.0	68.8	259.6
M. M.	41	M	HHD	B	109.0	68	60	270	4.5	3.37	49.6	183.7	10.0	70.1	259.2
				A	110.0	57	45	275	5.7	2.99	52.5	190.9	6.0	77.9	283.3
Heart failure group															
S. K.	56	F	ASHHD	B	115.0	92	70	196	3.2	2.68	29.1	148.5	25.0	30.0	153.1
				A	96.0	75	50	193	4.0	2.96	39.5	204.7	10.0	48.5	251.3
T. K.	65	M	ASHD	B	105.0	48	55	330	5.6	2.25	46.9	142.1	15.0	58.1	176.1
				A	102.0	56	45	330	6.5	2.76	49.3	149.4	10.0	64.8	190.3
Y. H.	72	M	ASHD	B	78.0	108	50	215	5.5	2.38	22.0	102.3	23.0	17.6	81.9
				A	86.0	95	35	223	7.2	2.76	29.1	126.0	12.0	30.6	132.7

Abbreviations; B: before digitalization, A: after digitalization, M: male, F: female, mBP: mean blood pressure, HR: heart rate, ICT: isometric contraction time, ETm: measured ejection time, ETc/ICT: the ratio of corrected ejection time to isometric contraction time CI: cardiac index (L/min/M²), SI: stroke index (ml/beat/M²), MSER: mean systolic ejection rate (ml/M²/sec.), LVEDP: left ventricular end-diastolic pressure (mmHg), LVSU: Left ventricular stroke work (Gm. M/beat/M²), LVSP: left ventricular stroke power (Gm. M/beat/M²/sec.), JH: juvenile hypertension.

subjects was longer by 0.02 seconds than male.

The difference of the regression slope between $Q-S_2$ and ET implies that tension period varies inversely with heart rate, as LANG¹⁷ and WEISSLER¹⁸ also have demonstrated. In contrast, FRANK and KINLAW¹⁹ noted no such relationship between heart rate and tension period. This may be due to a small number of cases applied for the study.

The correction of the systolic time intervals to a standard heart rate by the use of the regression equation facilitates the comparison of each duration of left ventricular systoles in patients with different heart rate.

BLUMBERGER¹³, HOLLDACK²⁰ and SAMBHI²¹ reported the prolongation of tension period and isometric contraction time in hypertensive patients, and in addition, JEZEK¹² and MATSUO²² described its prolongation in arteriosclerotic heart disease as well. These observations were consistent with the findings in the present study. Furthermore, the present observation may offer a detailed information with respect to the relationship between the myocardial contractile state and the duration of left ventricular systoles. COTTEN and BAY demonstrated that a considerable portion of the left ventricular contractile force developed during the isometric contraction phase and the changes in the duration of isometric pressure gradient were directly related to those in the strength of the ventricular contraction²³. REEVES and associates described that the duration of the isometric contraction of the cardiac cycle was in part a function of the rate of contraction and, consequently, to allow the period of isometric contraction to remain essentially constant, even though the blood pressure in the aorta may change remarkably²⁷. Therefore, in hypertension without myocardial involvement, the isometric contraction period is not prolonged as shown in the present study because of the increase in pressure rising rate accommodated to the elevated arterial resistance. This ability of the myocardium appears to be still held in hypertensive and arteriosclerotic heart disease, because a significant difference in tension period or isometric contraction period between these two groups is absent. The prolongation of isometric contraction time in heart diseases implies that

the reduced ability of the myocardium to change its contractility may act in a new level of adjustable range. These may suggest clinically the existence of "Homeometric Autoregulation" that SARNOFF²⁵ described. On the other hand, it is lost in failure state.

$Q-S_1$ interval derived by subtracting isometric contraction time from tension period showed no significant difference between normal subjects and patients with heart disease. This indicates that the prolongation of tension period in heart diseases is due largely to the prolongation of isometric contraction time. In contrast, $Q-S_1$ interval in failure patients was markedly prolonged and the average value was 0.074 seconds in normotensive and 0.076 seconds in hypertensive group respectively. Consequently, the height of systemic blood pressure seems not to play an important role in producing the prolongation of $Q-S_1$ interval even in heart failure. However, the prolongation of $Q-S_1$ interval was observed in some cases of hypertensive heart disease and arteriosclerotic heart disease. The principal factor contributing to the prolongation of $Q-S_1$ interval is perhaps a myocardial involvement induced by a persistent hypertension. It is conceivable from this that a prolongation of $Q-S_1$ interval suggests a latency of the myocardial damage. In addition, although a prolongation of $Q-S_1$ interval in heart failure may be attributable to the longer time required from the onset of ventricular depolarization to the initiation of mechanical contraction in the presence of elevated end-diastolic pressure, it showed no significant change even after a failure state was eliminated with acute digitalization. The prolongation of $Q-S_1$ interval would be due not to the delayed closure of mitral valve in the presence of elevated left atrial pressure, but to a disturbance in electromechanical coupling, which might exist already until falling into heart failure.

GOBBAT and MEDA⁸ described that a rise of the mean and pulse pressure had no significant influence on the duration of isotonic systole. BRAUNWALD³ demonstrated in heart-lung preparation that the substantial change in mean aortic pressure had little influence on the duration of ejection. BLUMBERGER¹³ also found little change in compensated hypertensive patients.

Furthermore, it was shown by JEZEK¹² that an acute myocardial infarction exhibited a shortening of the ejection period, while the sequence of events of cardiac contraction was practically normalized in all patients during healing of myocardial necrosis. The findings in ejection time in the present study are consistent with these descriptions. However, in severe hypertensive and arteriosclerotic heart disease, the abbreviation of ejection time was observed with an associated decrease in cardiac output. The duration of ejection was indicated to be directly proportional to stroke output, as shown by WEISSLER and associates. It is, therefore, reasonably considered that an abridged ejection time indicates a decreased stroke output. On the other hand, most diseased hearts without failure may maintain to some extent normal values of ejection time, even though they are not ejecting the same blood volume as normal hearts in the presence of decreased systolic ejection rate. From this view-point, the latency of reduced cardiac performance, if any, could not be detected only by the measurement of ejection time.

In regard to the evaluation of the ventricular performance by the measurement of systolic time intervals, it is reasonable that isometric contraction time as well as ejection time should be taken into account. It was, therefore, examined in the present study whether or not a ratio of ET/ICT showed a parallel change to the alteration in hemodynamic functions. This ratio exhibited a tendency to be smaller in patients with heart disease than in normal subjects, because of the prolongation of isometric contraction time even in the absence of the shortening of ejection time. In nonfailure patients, it may not be parallel to such hemodynamic parameters as MSER or LVSW, which is maintained within normal range until a severe myocardial involvement develops later. After digitalization, however, the percentile increase in this ratio was followed by the percentile increase in MSER, LVSW and LVSP. In failure patients, the ejection time showed no consistent change and the isometric contraction time in some cases showed little change, after digitalization. The ratio of ETc/ICT was increased in all-over cases. One instance of non-

failure patients showed a decrease in the ratio of ETc/ICT associated with a decline in MSER and LVSW. This gives an impression that the administration of digitalis to nonfailing heart might suppress the ventricular performance. However, the previous investigations demonstrated by the use of the strain gauge arch that digitalis produced invariably the increase of myocardial contractility^{26,27}. In addition, it was revealed that changes in systemic output or stroke work did not necessarily reflect the actual changes occurring in contractile force after the administration of digitalis, since the systemic output and stroke work were the resultant of several variables including ventricular filling pressure, contractile force, heart rate and blood pressure²⁶. Especially, the volume of venous return appears to play an important role to determine the systemic output and stroke work, and also the duration of left ventricular systoles.

Although the direction and degree of the post-digitalized changes in the durations of systolic phases in failure patients may be variously influenced by the amount of augmented venous return, the changes of the ratio of ETc/ICT were accompanied by the associated parallel changes in MSER and LVSW. From this point of view, serial observations of the ratio of ETc/ICT offer the valuable informations for the evaluation of left ventricular function.

SUMMARY

Tension period, isometric contraction time and ejection time were measured in 20 normal subjects and 72 various nonvalvular heart disease patients with simultaneous recordings of electrocardiogram, phonocardiogram and carotid arterial pulse. Tension period and isometric contraction time were prolonged in the patients with heart disease. However, ejection time was within normal limits in heart disease patients without failure. In failure patients, ejection time was significantly shortened, accompanied by the marked prolongation of tension period. The myocardial involvement is a principal factor to modify the duration of the left ventricular systoles. In heart failure, the elevation of arterial resistance appears to play an important role to prolong the isometric contraction

time and to shorten the ejection time.

It was evaluated by augmenting the myocardial contractility with acute digitalization how the duration of left ventricular systoles might reflect the left ventricular performance. The ratio of ejection time to isometric contraction time showed a parallel change to the alteration in mean systolic ejection rate and left ventricular stroke work after digitalization. The serial observations of this ratio facilitates an early detection of the changes in left ventricular function.

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