STUDIES OF STRAINGAUGE KINETOCARDIOGRAM RECORDING METHODS AND ITS CLINICAL IMPLICATION

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PRECORDIAL movement of ultra-low frequency (15 Hz or less) induced by cardiac mechanical events has been considered to provide valuable informations about cardiac function.^{1,2} However, several problems concerning the recording techniques remain to be solved. Some of these relate with the unsatisfactory physical characteristics of the mechanoelectric transducers^{3,4} and the inevitable interference caused by various tissues located between the heart and the transducer. The author has developed a method of recording the movement of the chest wall produced by cardiac motion by means of a strain-gauge transducer, and named it "Strain-gauge Kinetocardiogram (S-KCG)". This technique offers several advantages for evaluation of the cardiac function compared with other previously reported methods.

METHODS

a) Setup of the equipment

Fig. 1 shows the schematic representation of the setup. The endpiece was made of small, cylindrical metal rods of 6 mm in diameter and 12 mm in length, connected to the strain-gauge transducer (SB-1T, Nihon Koden, K.K.). The transducer was held by an universal type clamp, and the latter fixed firmly to the bed by a steel bar so that the endpiece could be positioned perpendicularly to the chest wall. The pressure applied by the endpiece contacted to the chest wall could be changed either by adjusting the universal clamp or by adding an appropriate weight on the transducer. The perpendicular

component of the movement of the chest wall was recorded on a 4 channel ink-writing oscillograph (WI-180, Nihon Koden, K.K.) simultaneously with the electrocardiogram (ECG), phonocardiogram (PCG) and carotid or finger tip pulse. The paper speed was 50 mm or 125 mm per second. The setup was arranged to record the outward movement of the chest wall as a positive deflection. For the recording of the phonocardiogram and the external carotid pulse tracing, a crystal microphone of contact type (TY-301, Fukuda Electro. Co.) and a photoelectric transducer (MPP-2, Nihon Koden, K.K.) were used respectively.

b) Properties of the recording system

Fig. 2 shows the relationship between the displacement applied to the tip of endpiece by means of a calibrated micrometer, and the recorded amplitude. During the constant displacement applied to the endpiece, the response recorded on oscillograph stayed constant. This means that the device had an infinite time constant.

Fig. 3 shows the frequency-response relationship, when the constant displacement (0.2 mm) of variable frequencies were applied to the endpiece. The relative amplitudes of the deflections on the oscillograph were constant at frequencies lower than 15 Hz, while at higher frequencies, relative amplitudes decreased as the frequencies increased. The time lag of response in this setup device was 0.004 seconds or less i.e. virtually negligible. The amplitude and the configuration of S-KCG changed considerably when the pressure applied to the endpiece was varied.

Fig. 4 shows the changes recorded from the same site on the precordium when the pressure applied to the chest wall was varied stepwise from 40 to 780 gram. As illustrated in this figure,

Key Words: Precordial Movement
Kinetocardiogram
A Wave
Mitral Stenosis
Isovolumetric Contraction Time

(Received for publication, October 3, 1972)

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Fig.1. Diagram of the strain gauge kinetocardiographic set-up used in recording the absolute displacement of the chest wall.

Fig.2. The relationship between various amplitude of displacement given to the endpiece by micrometer (abscissa) and its response recorded by the strain gauge kinetocardiographic pickup (ordinate).

the pressures of 390 and 780 gram seemed appropriate for the detailed analysis of the wave components. Thus, the pressure of 700 gram (2,400 gram per square centimeter) was used in

most studied cases.

c) Recording procedure.

Thirty healthy volunteers and 54 patients with various types of heart disease including 20 cases of pure mitral stenosis and 24 cases of atrial septal defect were investigated. The subjects were examined in supine position on the firm bed. Recording was done during held expiration. The pickup device was placed firmly on the desired points on the chest wall. Recording was made from various sites on the chest wall. Each site was designated by the abbreviation "K" which had a suffix consisting of two integer numbers. The first number refers to the vertical lines corresponding to the chest leads in the ECG (V_{1-6}), and the second number to the intercostal spaces. For instance, K35 indicates the point on the vertical line used to record lead V3 in the ECG but at the level of 5th intercostal space.

RESULTS

a) Tracings from normal subjects

Twenty men and ten women volunteers aged 18 to 30, served as control subjects. Recordings from the positions K34 or K35 were chosen for detailed analysis. Fig. 5 shows typical example of a normal tracing. The latter was usually com-

Fig.3. Frequency-response curve of straingauge kinetocardiographic system. The response (ordinate) was measured by applying the displacement of 0.2 mm in variable frequencies (abscissa) produced by the micrometer. The ordinate is shown as the ratio to the amplitude of response in 4 Hz. Note that responses are constant at frequencies lower than 15 Hz.

Fig.4. Effects of various pressures on the straingauge kinetocardiographic (S-KCG) tracing. The S-KCG were obtained from the same points (K34) of a 24-year-old man under the variable pressing weight of 40, 200, 390, and 780 gram.

posed of 5 main components: (1) a small outward movement (atrial wave; "a") was recorded about 0.05 second after beginning of P wave in ECG with its peak at 0.09 second after beginning of P wave. The amplitude of "a" wave was measured from the base to the peak while the total maximum deflection (TD) was measured

from the peak to the lower most point of the tracings. The amplitude of "a" wave did not exceed 10% of TD. (2) In early systole, two outward movements were recognized: the first component was named preejection (PE) and the second, ejection wave (E). PE wave began just after the onset of QRS complex and its peak

Fig.5. Typical tracings from a 23-year-old healthy man.

A: Electrocardiogram (lead II). B: Strain-gauge kymocardiogram (K34). C: Phonocardiogram (4L3). D: Carotid pulse tracings. "a": Atrial wave. PE: Preejection wave. E: Ejection wave. MOM: Midsystolic outward movement. R: Recoil wave. F: Filling wave.

TABLE 1 TEMPORAL RELATIONS BETWEEN ECG (P AND Q) AND S-KCG ("a" AND PE) AS WELL AS PE-E INTERVAL AND "a" WAVE RATIO (a/TD) IN S-KCG FROM NORMAL 30 SUBJECTS

	range		mean	S.D.
<i>Interval (second)</i>				
<i>P-onset of "a"</i>	0.040	0.090	0.05	0.03
<i>P-peak of "a"</i>	0.050	0.120	0.09	0.03
<i>Q-onset of PE</i>	0.004	0.020	0.016	0.003
<i>Q-peak of PE</i>	0.020	0.056	0.045	0.008
<i>PE-E</i>	0.016	0.070	0.055	0.0085
<i>a/TD</i>	0.0	0.102	0.055	0.026

coincided with the first component of the first heart sound (mitral valve closure sound). In most cases, PE wave was recorded over the wide area of the precordium. The maximum amplitude was usually recorded at the left parasternal or anterior regions (K34, K44), where the ventricular lateral wall is considered to be closest to the

chest wall. E wave was usually the largest positive deflection followed by a rapid descent recorded best at the apex (K45, K35, K55). The peak of the E wave coincided with the second component of the first heart sound (aortic ejection sound) and the onset of carotid pulse corrected by delay i.e. the onset of ventricular ejection. (3)

Fig.6. A 17-year-old man with second degree atrio-ventricular block of Wenckebach type. Note that all P waves in ECG (lead II) are followed by "a" wave in S-KCG (K35).

Fig.7. A 63-year-old man with arteriosclerotic heart disease and atrial fibrillation. A: ECG (II). B: S-KCG (K34). C: Finger tip pulse. D: PCG (4L3). Note that beats with a longer preceding R-R interval than 0.68 second shows low amplitude MOM on S-KCG and high upstroke on finger tip pulse, while the beats of shorter preceding R-R interval shows a high amplitude MOM and low finger tip pulse.

In mid to late systole, small and slowly ascending outward movement was observed and was named mid-systolic outward movement (MOM). However, in normal subjects, MOM was usually small and sometimes absent. (4) Following MOM, small vibration (R), probably due to recoiling movement of the aortic valve was recorded at the time of second heart sound. (5) Following MOM and R, outward movement (F) was recorded in the phase of ventricular filling. In some cases, this movement could be divided into initial rapid (rF) and later slow filling (sF) phase.

Table I summarizes the relation of each wave in S-KCG to simultaneously recorded ECG (lead

II) and "a" wave ratio (a/TD) which is expressed as a ratio of the amplitude of "a" wave to the total maximum deflection in the tracings from 30 normal subjects.

b) Tracings from patients and the modification of S-KCG by various drugs.

Typical tracings recorded in patients with various cardiac disorders show: second degree atrio-ventricular block (Fig. 6), atrial fibrillation (Fig. 7), ventricular (Fig. 8) and atrial (Fig. 9) premature beats, intermittent right bundle branch block (Fig. 10), mitral valvular disease (Fig. 11, 12) and myocardial infarction (Fig. 13).

The effect of amyl nitrite was studied in 30

TABLE II RESULTS OF MEASUREMENTS FROM 20 CASES OF PURE MITRAL STENOSIS

Patient, age, & sex	ECG interval (sec)		S-KCG "a" wave			Intracardiac pressure (mmHg)					
			ratio	duration		PCW		RV		RA	
	P-Q	R-R	(a/TD)	P-a _O	P-a _m	mean	a	syst.	end-diast.	mean	a
M. S. 17 M	0.170	1.120	0.24	0.050	0.140	13.0	15.0	30.0	6.3	6.0	7.5
K. T. 32 F	0.128	0.784	0.26	0.096	0.120	26.2	32.5	47.5	11.2	8.0	11.5
T. M. 37 M	0.168	0.664	0.25	0.080	0.128	17.5	28.0	42.0	5.0	4.0	7.5
K. M. 28 F	0.104	1.080	0.24	0.096	0.168	27.5	31.2	40.0	3.8	2.5	4.5
T. K. 42 F	0.170	0.920	0.26	0.030	0.130	16.0	21.0	42.5	4.0	3.5	7.5
K. T. 34 F	0.180	0.740	0.50	0.120	0.180	25.1	30.6	41.3	3.0	6.5	10.0
I. M. 17 M	0.160	0.920	0.22	0.020	0.120	25.3	31.5	65.0	8.9	4.3	8.0
N. R. 39 F	0.180	1.000	0.28	0.090	0.170	20.0	25.0	40.0	7.5	4.5	7.0
K. N. 44 F	0.180	1.000	0.40	0.060	0.170	17.6	28.0	38.5	0.0	0.5	4.5
M. T. 32 F	0.190	1.140	0.35	0.080	0.190	20.0	27.0	30.0	5.0	6.5	9.0
M. F. 45 F	0.180	1.200	0.20	0.060	0.160	12.0	16.0	25.0	2.5	2.0	5.0
Y. K. 26 F	0.200	1.040	0.26	0.060	0.120	27.5	26.9	61.5	8.8	5.5	9.0
K. A. 32 F	0.180	1.160	0.17	0.050	0.100	16.5	25.8	27.5	0.0	5.0	6.8
F. K. 30 M	0.190	1.060	0.27	0.060	0.120	16.9	26.5	41.3	3.8	3.0	4.0
O. S. 38 F	0.170	1.100	0.13	0.140	0.180	17.5	25.0	50.0	-2.5	4.0	8.0
H. T. 37 F	0.160	0.730	0.33	0.024	0.112	22.5	36.5	50.0	2.0	3.0	9.0
K. Y. 47 M	0.180	0.120	0.23	0.120	0.180	21.0	31.0	47.0	4.0	2.5	5.0
H. S. 26 M	0.160	0.840	0.33	0.070	0.160	33.6	41.3	50.0	3.8	5.5	7.5
N. K. 32 F	0.170	1.040	0.23	0.060	0.120	20.0	29.3	36.3	3.3	2.5	6.0
H. K. 33 F	0.200	1.100	0.13	0.060	0.160	11.1	16.0	31.3	5.0	3.0	6.0

Abbreviations; ECG: electrocardiogram lead II, S-KCG: strain-gauge-kinetocardiogram, a/TD: a ratio of "a" wave to total deflection (TD), P-a_O: interval from onset of P in ECG to onset of "a" wave, P-a_m: interval from onset of P in ECG to peak of "a" wave, PCW: pulmonary capillary wedge, RV: right ventricle, RA: right atrium.

TABLE III TEMPORAL RELATION BETWEEN P WAVE IN ECG AND "a" WAVE IN S-KCG AND "a" WAVE RATIO (a/TD) IN 20 CASES OF MITRAL STENOSIS

	range	mean	S.D.
Interval (second)			
P-onset of "a"	0.030	0.140	0.07
P-peak of "a"	0.140	0.190	0.14*
a/TD	0.13	0.50	0.261*

* $P < 0.01$, significant difference from the mean obtained from normal subjects.

normal volunteers and in eight patients. A typical response is shown in Fig. 14. A β -receptor stimulant (trimetoquinol hydrochloride) was injected intramuscularly in 3 normal volunteers and a typical effect is shown in Fig. 15.

The change of each wave component of S-KCG produced by the underlying disease and the tested drugs will be described below.

"a" wave

The "a" wave, which was always recorded in

TABLE IV CORRELATION COEFFICIENT BETWEEN $a/$ TD AND INTRACARDIAC PRESSURES IN 20 CASES OF MITRAL STENOSIS

PCW "a" pressure	0.491*	$P < 0.05$
PCW mean pressure	0.408	
RV systolic pressure	0.280	
RV end-diastolic pressure	0.066	
RA "a" pressure	0.296	
RA mean pressure	0.219	

Abbreviations are same as in Table II.

Fig.8. Tracings from a 24-year-old woman with ventricular premature beats.

A: ECG (II). B: S-KCG (K34). C: Finger tip pulse. D: PCG (4L3).

2nd and 5th beats are sinus origin with normal PE-E interval (0.050 second). 3rd and 6th beats are premature beats of ventricular origin. In the premature beats, no upstroke on finger tip pulse tracings and no second heart sounds were recognized. The S-KCG showed abnormal patterns with prolongation of PE-E interval (0.082 second). In post-extrasystolic beats (1st and 4th beats), shortening of PE-E interval (0.043 second) with increased amplitude of PE was observed associated with sharp rise in the finger tip tracing.

normal subjects, disappeared in atrial fibrillation (Fig. 7), while in the atrioventricular block (Fig. 6), it was well defined after each P wave in the ECG. Large "a" waves were recorded in the patients with myocardial infarction (Fig. 13), congestive heart failure of various etiologies and aortic valvular stenosis. After amyl nitrite inhalation, the "a" wave increased in amplitude in all 30 normal subjects as typically shown in Fig. 14B. In all patients with atrial septal defect and in all patients with mitral stenosis and sinus

rhythm, large "a" waves were recorded (Fig. 11). In mitral stenosis, the magnitude of $a/$ TD was compared with intracardiac pressures (Table II). The mean value of $a/$ TD in mitral stenosis (0.261) was significantly greater than that in normal subjects (0.055) (Table III). In addition; there was a significant positive correlation between the $a/$ TD value and the amplitude of the "a" wave in the pulmonary capillary wedge pressure tracing, but no significant correlation between the $a/$ TD and the right ventricular

Fig.9. Tracings of a 49-year-old man with ischemic heart disease and atrial premature beat.

A: ECG (II). B: S-KCG (K34). C: Finger tip pulse. D: PCG (4L2).

ECG shows variable R-R interval with similar QRS complexes. According to the change of preceding R-R interval, S-KCG changed its configuration accompanied by the change in finger tip pulse and PCG.

Fig.10. Tracings from a 58-year-old man with arteriosclerotic heart disease and intermittent right bundle branch block.

1st, 4th and 5th beats show normal conduction while 2nd, 3rd and 6th beats show right bundle branch block (RBBB) (A). Beats in RBBB show splitting of first heart sound in PCG (B) and prolonged PE-E interval (0.082 second) in S-KCG tracings (C), while beats of normal conduction show the non-split first heart sound in PCG and short PE-E interval (0.054 second) in S-KCG. However, no changes were observed in the finger tip pulse tracing (D). * Artefact.

end-diastolic pressure, or the "a" wave in the right atrial pressures tracing (Table IV).

PE and E wave

The β -stimulatory drug increased the PE as well as E waves (Fig. 15). This result suggests that the PE and E waves increase when the

contractile force of the heart increases. An increase in PE wave in the post-extra-systolic beat (Fig. 8) also lends support to this hypothesis. The PE and E waves were recognized even patients with prosthetic mitral valve.

The PE-E interval was measured between the

Fig.11. Tracing from a 32-year-old man of tight mitral stenosis with high mean pulmonary capillary wedge pressure (30 mmHg). Note large "a" wave ($a/TD = 0.20$) and slow rising F wave.

Fig.12. Tracing of a 45-year-old woman with mitral insufficiency. A: ECG (II). B: PCG (3L4). C: S-KCG (K34). D: Finger tip pulse. S-KCG shows a large "a" wave, prominent late systolic outward movement (OM) and rapid filling wave. Note that 3rd heart sound was coincided with the end of rF wave.

peaks of PE and E waves. This interval was shortened in the post-extrasystolic beats than in normal beats (Fig.8). The β -stimulant also induced a

shortening of PE-E intervals (Fig.15). This suggests that PE-E interval shortens when the contractile force increases. The PE-E interval was prolonged

Fig.13. Tracings from a 72-year-old man with recent myocardial infarction.

A: ECG (II). B: S-KCG (K34). C: Finger tip pulse.

Note the large "a" wave ($a/TD = 0.40$), exaggerated MOM and prolonged PE-E interval (0.073 second).

Fig.14. Effects of amyl nitrite inhalation on the precordial movement of a 16-year-old healthy woman. From top to bottom, ECG (II), S-KCG (K34), finger tip pulse and PCG (5L3). A before, B: 20 seconds and C: 60 seconds after inhalation. Note the exaggerated "a" wave after the procedure.

with ventricular premature beats (Fig. 8) and the beats with the bundle branch block pattern (Fig. 10). In a patient with myocardial infarction, the PE-E interval was also prolonged (Fig. 13).

Midsystolic outward movement, MOM

The midsystolic outward movement (MOM) was often observed in patients with cardiac failure, ischemic or hypertensive heart disease and in some patients with valvular disease. In patient with myocardial infarction, marked MOM was usually associated with prominent "a" wave and prolonged PE-E interval. Typical recording is shown in Fig. 13. The MOM was abnormal in

atrial and ventricular premature beats as well as in beats with short preceding R-R interval in atrial fibrillation (Fig. 7, 8, 9). In mitral incompetence, a huge outward movement appeared in mid to late systole (Fig. 12)

DISCUSSION

a) Recording methods.

The heart sound, murmurs and other auscultatory phenomena represent vibrations within the audible range. However, cardiac motion produces also vibrations with much lower frequencies than 20 Hz and these vibrations are

Fig.15. Effects of beta-stimulatory drug on S-KCG (K34) of an 18-year-old healthy man.

Before (A) and 3 minutes after (B) the injection of trimetoquinol hydrochloride 0.1 mg (INOLIN®). In B, the first heart sound was extremely exaggerated and PE and E increased in amplitude while PE-E interval was shortened (from 0.060 to 0.036 second).

transmitted to the surface of the chest wall. These precordial low frequency vibrations may provide certain informations about the cardiac mechanical events, which cannot be obtained by other methods such as auscultation or phonocardiography.

There are two different categories of methods used to record the low frequency precordial movements. One is the measurement of the displacement relative to the ground or to some fixed point on the bed (absolute displacement)^{5,8} The S-KCG used in the present study belongs to this category. The other type is the measurement of the differences between two points on the precordium (relative displacement)^{9,11} The recording of the relative movement with such as apexcardiogram (ACG) reported by *Benchimol*¹⁰ does not always show a true precordial movement.¹² For example, the outward movement of one point might be recorded as an inward movement if the surrounding area shows a much greater outward movement. It appears therefore that the absolute displacement as measured in this study is more suitable for the analysis of precordial movements. In order to measure the absolute displacement, various kinds of transducer have been employed.⁵⁻⁹

The recording system used by the author

showed a linear relation between given displacement and the response (Fig. 2). The response to the variable frequencies was flat in the range of subaudible ultra-low frequencies (Fig. 3). The infinite time constant of the system enables to pick up very slow movements i.e. MOM wave with high fidelity.

The bed-side experience shows that even when one cannot see the precordial pulsation on the surface of the chest wall, one can feel the pulsation on the finger tips by applying considerable pressure to the chest wall. This means that the energy of the pulsatile movement of the heart may be reduced by the soft tissues intervening between the heart and the chest surface.

In order to avoid such interference of the soft tissues, considerable pressure was applied to the pick up device placed on the chest wall using this procedure, the movement of the chest wall produced by cardiac motion could be more faithfully transmitted to the pick up device as shown in Fig. 4. Some investigators used the photoelectric transducer to measure the absolute displacement without pressure.^{8,9} These methods will be suitable for the recording "surface" movement of the chest wall modified by cushion effects of the soft tissues, but may not be helpful

in registering the cardiac motion through the movement of the precordium.

The impulse cardiography by *Mounsey*⁶ was also designed to record the absolute displacement. However, the pressure applied to the chest wall was small (100–200 gram), and therefore not sufficient to exclude the cushion effects of the intervening tissues. In the author's experience, the use of weights less than 200 gram resulted in greatly reduced amplitude of the precordial movement probably due to elasticity of the soft tissues. In the range between 400 gram and 800 gram, the amplitude of deflection was large and stable (Fig. 4). The use of weight exceeding 800 gram (about 3,000 gram per square centimeter) was not practical because it often made the patient uncomfortable. Therefore, 700 gram of pressing weight was usually applied in this study. *Eddleman* and *Harrison*⁵ recorded the absolute movement of the precordium applying about 900 gram to the chest wall. They used a piezoelectric transducer connected to the endpiece by a tubing. However, the latter may produce some distortion in the record. S-KCG tracing were highly reproducible from day to day perhaps because the endpiece was firmly applied perpendicularly to the precordium with considerable pressure in supine position.

b) Possible relation to the hemodynamic events.

"a" wave

It has been generally accepted that prominent atrial waves in the precordial movement tracings are present in congestive heart failure, ischemic heart disease and cardiomyopathies.^{1,3,14} On the other hand, small or absent "a" wave has been reported in mitral stenosis in the apexcardiogram and impulse cardiogram. *Benchimol*¹³ and *Mounsey*⁶ reported that mitral stenosis tends to diminish the size of "a" wave due to the increased resistance to the blood flow through the mitral valve. My observations, however, show large and not small "a" waves in patients with mitral stenosis (Fig. 11, Table II, III). Significant correlation was found between a ratio of "a" wave to the total deflection (a/TD) and "a" wave pressure in pulmonary capillary wedge (Table IV), while there was no correlation between the value of a/TD and the RA or RV pressures (Table IV). Hence, it is possible that increased left atrial contraction and pressure in mitral stenosis contributes to the development of "a" wave in S-KCG. The author usually observed also large "a" waves in atrial septal defect and in normal

subjects after amyl nitrite inhalation, where the increased blood flow through mitral valve is expected. Thus, "a" wave in S-KCG is assumed to be formed not only by the increased blood flow induced by the atrial contraction in the late ventricular diastole but also by the direct outward movement of the whole ventricular mass protruded by the strong atrial contraction. Therefore the amplitude of "a" wave on S-KCG could be an indicator of both pressure and volume overload in the left atrium.

PE wave

PE wave may be formed by the expansion of the left ventricular lateral wall and corresponds to the initial systolic expansion by *Rushmer*¹⁵ during the initial phase of ventricular systole. *McDonald* reached the same conclusion by studying the motion of the epicardial markers on the left ventricle in men by cineventriculography.¹⁶ This initial systolic expansion has been attributed to the descent of the mitral valve ring caused by the contraction of the papillary muscle.¹⁷ The increase in PE wave after injection of β -stimulant (Fig. 15) and after premature ventricular contraction (Fig. 8), where the contractile force of papillary muscle would be enhanced, may support this hypothesis. However, PE wave was recorded even in patients with mitral valve prosthesis. This suggests that the PE wave produced not only by the contraction of papillary muscle but also by the contraction of other parts of the ventricle. *Tafer*¹⁸ mentioned a notch on the E wave in the apexcardiogram just before the ejection. This notch coincided with the first component of the first heart sound. However, *Bancroft*¹² pointed out that the employed microphone had a sharp peak at the range of approximate 20 Hz in the frequency-response curve. Thus, it is possible that this notch is derived from some component of the heart sound exaggerated by the transducer, and that a notch on the E wave reported by Tafer may not be the direct reflexion of cardiac movement but an artifact caused by the transducer. S-KCG did not show such a peak in the frequency-response curve (Fig. 3). Thus it is reasonable to assume that the PE wave in S-KCG is a reliable indicator of the time of initial systolic expansion or closure of the mitral valve.

PE-E interval and isovolumetric contraction time

The measurement of isovolumetric contraction time (IVCT) has been reported to be a useful indicator of myocardial contractility.¹⁹ Usually

the IVCT has been obtained by measuring the interval between the mitral component of the first heart sound in the PCG or the onset of E wave of the apexcardiogram and the onset of the carotid upstroke corrected by the pulse delay (the interval between the aortic component of the second heart sound and the dicrotic notch on the carotid pulse tracings)²⁰ However, this method is often difficult to measure the IVCT. Mitral closure sound in the PCG is sometimes indistinguishable from other components of the sound. The beginning of E wave in apexcardiogram does not always correspond to the time of mitral closure.¹⁸ It may be also difficult to identify the onset of upstroke and the dicrotic notch in the carotid pulse tracings because they are sometimes obscured. Moreover, even if the measured interval is corrected by the pulse delay, this measurement is based upon an assumption that the interval between the beginning of the ventricular ejection and carotid upstroke in early systole is precisely the same as the interval between the aortic valve closure sound in PCG and dicrotic notch of carotid pulse tracing. In S-KCG, however, IVCT could be directly measured by the PE-E interval assuming that PE and E wave correspond to the time of mitral valve closure and the beginning of ventricular ejection, respectively. Prolongation of PE-E interval is shown in the myocardial infarction (Fig. 13), intermittent bundle branch block (Fig. 10) and ventricular premature beat (Fig. 8), where ventricular contractile force may be decreased.^{1-2,3} Shortening of PE-E interval was shown in the postextrasystolic beat (Fig. 8) and after administration of β -stimulatory drug (Fig. 15), where ventricular contractile force may be increased.^{2,4,25}

MOM wave

Sustained outward movement during systole was described in various diseases of the heart.^{26,27} These abnormal movements and the MOM recorded in S-KCG would be of same origin and may be produced by the following mechanisms: (1) As shown by *Prinzmetal*²⁸ and others,²⁹ the ballooning of ischemic or necrotic ventricular wall may be induced by high pressures generated by the residual intact musculature. Possible changes induced by this mechanism was illustrated on S-KCG of myocardial infarction (Fig. 13) and ischemic heart disease (Fig. 7). (2) The second mechanism is the altered sequences of ventricular excitation.^{21,23,30} The observed abnormal MOM in the ventricular

premature beat (Fig. 8) supports this hypothesis. (3) As shown in atrial fibrillation (Fig. 7) and atrial premature beat (Fig. 9), the configuration of S-KCG changed from beat to beat. Therefore, it is possible that alterations in diastolic filling period changes the pattern of the MOM of succeeding ventricular condition. (4) In patient with mitral incompetence (Fig. 12), abnormal outward movement was recorded in systole. This phenomenon is probably due to outward shift of the ventricular mass caused by expansion of the left atrium produced by a large quantity of regurgitant blood.³¹ (5) Lastly, the extracardiac factor may not be negligible since, for instance, pulsation of the thoracic aorta may transmit to the precordium in some conditions. Further analysis of the relation between the hemodynamic data and the change in S-KCG tracings remains to be carried out. It was already pointed out that, in the evaluation of the cardiac function, not only hemodynamic data but also the consideration of the synchrony of the ventricular movement is important.³² In this area of investigation, the measurement of absolute precordial movement by S-KCG will be of great value.

SUMMARY

- 1) A new technique of kinetocardiogram (S-KCG) by using a strain-gauge was introduced to measure the absolute precordial movement.
- 2) The device had a constant frequency-response curve within the range lower than 15 Hz, linear relationship between the given displacement and the recorded amplitude and infinite time constant.
- 3) The influences of intervening soft tissue on the record could be avoided by the pressure (700 gram) on the chest wall applied through the pickup device.
- 4) The recorded tracings were consisted of 5 main components: atrial wave ("a"), preejection wave and ejection wave (PE and E), midsystolic outward movement (MOM), recoil movement at the time of second heart sound (R), rapid and slow filling wave (rF and sF). A magnitude of "a" wave was expressed as "a" wave ratio to the total maximum deflection (a/TD).
- 5) Various changes in the configuration of S-KCG in altered hemodynamics which were induced by amyl nitrite inhalation, β -stimulant injection, arrhythmias and various con-

genital and acquired heart diseases were demonstrated.

- 6) Absolute amplitude of "a" wave as well as a/TD increased under the conditions of both pressure and volume overload in the left atrium. In mitral stenosis, there was significant positive correlation between a/TD and the pulmonary capillary wedge pressure.
- 7) The direct measurement of isovolumetric contraction time could be possible by measuring the PE-E interval.
- 8) Several possible mechanism and genesis of MOM wave were discussed.
- 9) It was concluded that the S-KCG is useful in the evaluation of the synchrony of the contraction.

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