Duration of phases of left ventricular systole using indirect methods

I: Normal subjects

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The duration of the various phases of left ventricular systole has been measured in a series of 50 normal subjects using high speed simultaneous recordings of the electrocardiogram, the phonocardiogram, the carotid pulse, and the apex cardiogram.

The five phases of systole measured were: total electromechanical systole ($Q-A_2$ interval); left ventricular ejection time (LVET); pre-ejection period (PEP); isovolumetric contraction time (IVCT); total mechanical systole (TMS).

The $Q-A_2$ interval, left ventricular ejection time, and total mechanical systole were linearly and inversely related to heart rate. The pre-ejection period and the isovolumetric contraction time were not significantly related to heart rate. The validity of the method has been substantiated by comparing simultaneous records of direct and indirect pulse forms in a small series of patients. The findings are reproducible and serial daily studies at different heart rates showed no deviation from the normal range.

The results have been compared with those reported by various authors and they are in close agreement.

Methods used to assess cardiac function, which depend on measurements of intravascular pressure and flow, are not readily performed in ambulant patients. The procedures are uncomfortable, carry some risk to the patient, and are not easily repeated.

The duration of the phases of left ventricular systole, which are potentially useful as indices of left ventricular performance, can be accurately measured from externally recorded pulse waves, and such externally recorded time intervals do not appear to differ significantly from those obtained by direct measurement. Recent publications have described the various techniques which can be used (Jezek, 1963; Oreshkov, 1965; Weissler, Harris, and Schoenfeld, 1968; Spodick and Kumar, 1968a and b).

This paper describes a method of recording and measuring five important phases of left ventricular systole, viz:—

- 1) The $Q-A_2$ interval or total electromechanical systole (TEMS).

- 2) Left ventricular ejection time (LVET).
- 3) Pre-ejection period (PEP).
- 4) Isovolumetric contraction time (IVCT).
- 5) Total mechanical systole (TMS).

The results in normal subjects are presented to establish a normal range for comparison with those in various cardiac disorders. In addition, we have compared our findings with those of other workers.

Methods and subjects

Studies were performed on 50 normal healthy adults (35 men and 15 women) aged 18 to 79 years. Twenty-five were under 40 and 25 were over 40 years of age. The majority were members of the hospital staff and the remainder were surgical patients admitted for relatively minor procedures unrelated to the cardiovascular system. All subjects had a normal exercise tolerance, no cardiovascular abnormality on clinical examination, a normal electrocardiogram, and were not on any form of medication.

Simultaneous recordings were made of the electrocardiogram, the phonocardiogram, the apex cardiogram, and the right carotid arterial pulse using a multichannel oscilloscopic recorder (Cambridge Instrument Company) and a paper

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FIG. 1 Normal subject: simultaneous records of the apex cardiogram (ACG), the carotid pulse (CP), the phonocardiogram (PCG), and lead II of the electrocardiogram. $A = Q-A_2$ interval; $B =$ left ventricular ejection time (LVET); $C =$ onset of ACG upstroke (u) to onset of CP upstroke (u); $D = A_2$ to dirotic notch interval or pulse transmission time (PTT); $E =$ total mechanical systole (TMS); A minus $B =$ pre-ejection period (PEP); C minus $D =$ isovolumetric contraction time (IVCT); $S_1 =$ first heart sound; $A_2, P_2 =$ aortic and pulmonary components of the second heart sound; 2L = second left intercostal space parasternally; MF = medium frequency. Time lines are at 40 msec intervals.

speed of 100 mm/sec (Fig. 1). The recordings were performed in a similar fashion to the routine phonocardiographic practice of this Unit (Coulshed and Epstein, 1963). Usually standard lead II of the electrocardiogram was employed. A crystal microphone to record the heart sounds was placed parasternally in the second or third left interspace. Occasionally two microphones were used in order to identify the aortic and pulmonary components of the second sound. The right carotid arterial pulse and apex cardiogram were recorded using funnel-shaped pick-ups with an internal diameter of approximately 1.5 cm. The funnels were connected to identical piezoelectric crystal microphones by similar lengths of rigid plastic tubing less than 2 feet in length. There was no time lag between the two recording systems. Records were taken with the breath held in partial expiration and with the patient in a semi-left lateral position, in order to bring the apex impulse against the chest wall. Identification of the underlying zone of the ventricle was accomplished by recording a unipolar electrocardiogram lead at the site of the apex cardiogram.

The following intervals were measured directly from the tracing (Fig. 1).

- 1) The total period of electromechanical systole or the $Q-A_2$ interval. Defined as the interval from the onset of the Q wave of the electrocardiogram to the onset of the first high frequency component of the aortic component of the second sound.
- 2) The left ventricular ejection time measured from the beginning of the carotid upstroke to the nadir of the carotid incisura.
- 3) The interval from the beginning of the upstroke of the apex cardiogram to the onset of the carotid upstroke.
- 4) The delay in transmission of the central arterial pulse to the carotid artery, or the pulse transmission time, measured from the first rapid vibration of the aortic component of the second sound to the nadir of the carotid incisura.

All intervals were calculated as the mean of measurements made on at least five cardiac cycles, each read to the nearest 5 msec. Heart rate was calculated from the average RR interval of the cycles measured.

The following intervals were calculated from the above measurements.

1) **Pre-ejection period** This was calculated by subtracting the left ventricular ejection time from the total period of electromechanical systole ($Q-A_2$ interval). $PEP = (Q-A_2) - (LVET)$. This is the same as the interval from the onset of the Q wave of the electrocardiogram to the onset of the carotid upstroke corrected for the pulse transmission delay.

2) **Isovolumetric contraction time (IVCT)** The interval from the beginning of the upstroke of the apex cardiogram (ACGu) to that of the carotid pulse (CARu) corrected for the delay in transmission of the central arterial pulse to the carotid artery (PTT) (Oreshkov, 1965; Spodick and Kumar, 1968a). $IVCT = ACGu \text{ to } CARu - PTT$.

3) **Total mechanical systole (TMS)** The sum of isovolumetric contraction time (IVCT) and left ventricular ejection time (LVET). $TMS = IVCT + LVET$. This is the same as the interval from the beginning of the upstroke of the apex cardiogram to the aortic component of the second heart sound (A_2).

The results on the 50 subjects were statistically evaluated (Croxtton, 1959). Standard deviations and regression equations were calculated to relate heart rate to systolic time intervals.

In 11 patients with various cardiac disorders a Telco micromanometer was placed in the central aorta and left ventricle during a diagnostic study. Simultaneous records were made of the carotid pulse and the central aortic pressure curve in 7 patients and of the apex cardiogram and the left ventricular pressure curve in 7 patients. Three of these patients had both aortic and then left ventricular pressures recorded with the carotid pulse and the apex cardiogram, respectively. Experimental studies showed that there was no significant time lag between the Telco micromanometer and the external pulse microphone.

Results

These are shown in Table 1 and Fig. 2.

1) **Q-A₂ interval or total electromechanical systole (TEMS)** The mean Q-A₂ interval was 382 ± 23 msec with a range of 330 to 440 msec. There was a close linear relation between the Q-A₂ interval and heart rate (Fig. 2). The regression equation was $Y = +503 - 1.59 \text{ HR}$ ($r = -0.79$; $P < 0.001$).

2) **Left ventricular ejection time (LVET)** The mean LVET was 281 ± 21 msec with a range of 230 to 334 msec. There was a close linear relation between the LVET and heart rate (Fig. 2). The regression equation was $Y = +389 - 1.41 \text{ HR}$ ($r = -0.78$; $P < 0.001$).

3) **Pre-ejection period (PEP)** The mean PEP was 100 ± 13 msec with a range of 78 to 130 msec. There was no significant relation to heart rate (Fig. 2). The regression equation was $Y = +126 - 0.34 \text{ HR}$ ($r = -0.30$; $P < 0.05$).

4) **Isovolumetric contraction time (IVCT)** The mean IVCT was 70 ± 9.5 msec with a range of 51 to 90 msec. There was no significant relation to heart rate.

5) **Total mechanical systole (TMS)** The mean TMS was 351 ± 23 msec with a range of 292 to 418 msec. There was a significant linear relation to heart rate (Fig. 2). The regression equation was $Y = +464 - 1.49 \text{ HR}$; ($r = -0.73$; $P < 0.001$).

Discussion

Modern high speed multichannel recording machines allow simultaneous recording of the

electrocardiogram, the phonocardiogram, the carotid pulse, and the apex cardiogram. With a fast recording speed of 100 mm/sec, measurements can be made within an accuracy of about 5 msec.

Braunwald, Sarnoff, and Stainsby (1958) studied left ventricular ejection time in an isolated dog heart preparation. They found that increasing the stroke volume at a fixed heart rate increased the ejection time, whereas increasing the heart rate with a fixed stroke volume shortened the ejection time. Wallace *et al.* (1963) studied in more detail the various phases of left ventricular systole in dogs. They showed that increasing the stroke volume at a fixed heart rate prolonged the ejection time, shortened isovolumetric contraction time, and had little effect on total systole. Increasing the heart rate with a constant stroke volume reduced all three phases of systole.

Our findings in normal subjects also showed that the Q-A₂ interval and left ventricular ejection time were inversely related to heart rate and agreed with the findings of other authors (Oreshkov, 1965; Weissler *et al.*, 1968; Spodick and Kumar, 1968a). However, the pre-ejection period and the isovolumetric contraction time were not related to heart rate.

The period of total electromechanical systole (Q-A₂ interval) includes the time interval between the onset of depolarization and the onset of ventricular systole; the period of left ventricular contraction preceding ejection; and also the period of left ventricular ejection. There was a linear and inverse relation to heart rate, and the regression equation was similar to that found by Weissler *et al.* (1968) in normal subjects (Fig. 2). Similar findings have been reported by Toutouzas *et al.* (1969). The inverse relation of the Q-A₂ interval to

TABLE 1 Systolic time intervals in 50 normal subjects

	Range	Mean	Standard deviation	Regression equation
Total electromechanical systole (Q-A ₂ interval)	330-440	382	± 23	$503 - 1.59 \text{ HR}$ SD = ± 14 $r = -0.79$, $P < 0.001$
Left ventricular ejection time	230-334	281	± 21	$389 - 1.41 \text{ HR}$ SD = ± 13 $r = -0.78$, $P < 0.001$
Pre-ejection period	78-130	100	± 13	$126 - 0.34 \text{ HR}$ SD = ± 12 $r = -0.30$, $P < 0.05$
Isovolumetric contraction time	51-90	70	± 9.4	—
Total mechanical systole	292-418	351	± 23	$464 - 1.49 \text{ HR}$ SD = ± 16 $r = -0.73$, $P < 0.001$

FIG. 2 (A) Relation of Q-A₂ interval to heart rate in 50 normal subjects. In this and subsequent Figures the thick line is the regression line and the fine line shows ± 2 standard deviations. (B) Relation of left ventricular ejection time (LVET) to heart rate in 50 normal subjects. (C) Relation of pre-ejection period (PEP) to heart rate in 50 normal subjects. (D) Relation of total mechanical systole (TMS) to heart rate in 50 normal subjects.

heart rate in normal subjects appears to be mainly due to shortening of left ventricular ejection time, since the pre-ejection and the isovolumetric contraction time did not vary significantly with heart rate.

The left ventricular ejection period is an important measurement of cardiac performance and occurs between the opening and closing of the aortic valve. It is measured from the onset of pressure rise to the incisural notch of the central aortic pressure pulse. Left ventricular ejection time can also be measured from the indirect carotid pulse tracing, and in 5 patients studied by Weissler, Peeler, and Roehll (1961), there was close agreement with the ejection time measured from a simultaneously recorded central aortic pressure pulse.

We have studied 7 patients to validate this

point. Simultaneous measurements of left ventricular ejection time were made using a Telco micromanometer in the central aorta and a piezocrystal pulse microphone over the right carotid artery. There was no significant difference between the direct and indirect measurements of ejection time (Table 2 and Fig. 3), and carotid pulse tracings can therefore be used for measurement of left ventricular ejection time. However, accurate measurement requires care in recording the carotid pulse wave in order to show a clear onset of the carotid upstroke and a definite incisural notch.

The left ventricular ejection time in our series was 281 ± 21 msec and was similar to the findings of Jezek (1963) who found an ejection time of 274 ± 21 msec; of Spodick and Kumar (1968a) who found a time of 292 ± 19

TABLE 2 Comparison of apex cardiogram and carotid pulse with simultaneous records of left ventricular pressure and aortic pressure using TELCO intravascular micromanometer. (All measurements are given in msec and are the mean of 5 cardiac cycles.)

Patient No. and diagnosis	Onset apex cardiogram to onset of LV pressure (TELCO)	Simultaneous		Simultaneous	
		LVET* aorta (TELCO)	LVET carotid pulse	Onset aortic pressure to onset of carotid pulse	A ₂ to carotid pulse dicrotic notch
1) Cardiomyopathy	17	229	227	29	25
2) LV aneurysm and VSD	22	212	213	29	29
3) MR	17	210	209	28	30
4) MR	19	—	—	—	—
5) Cardiomyopathy	31	—	—	—	—
6) Congestive cardiomyopathy	15	—	—	—	—
7) Congestive cardiomyopathy	19	—	—	—	—
8) MR and MS	—	215	217	15	17
9) Mild MR	—	282	286	25	25
10) Constrictive pericarditis	—	246	246	18	17
11) LV aneurysm	—	221	220	16	16
Average	20	231	231	23	23

LVET = left ventricular ejection time.

msec: and of Sawayama *et al.* (1969) who found a time of 274 ± 21 msec. There was a linear and inverse relation to heart rate (Fig. 2), and the regression equation was also similar to that found by Spodick and Kumar (1968a) and Weissler *et al.* (1968).

Weissler *et al.* (1961) studied the relation between left ventricular ejection time, heart rate, and stroke volume in normal resting subjects. They found that the ejection time decreased as heart rate increased and increased as stroke volume was augmented, both relations being linear. In a clinical study, Harley, Starmer, and Greenfield (1969) also showed that the duration of ejection was directly and linearly related to stroke volume and inversely related to heart rate. These findings are consistent with the experimental work carried out in dogs by Braunwald *et al.* (1958) and by Wallace *et al.* (1963). It is therefore clear that heart rate and stroke volume each have an independent influence on the duration of left ventricular ejection.

The pre-ejection period is the time interval between the onset of depolarization and the onset of left ventricular ejection. It includes the time taken for the spread of the electrical stimulus through the myocardium; the period of left ventricular contraction before mitral valve closure; and the period between mitral valve closure and the onset of ejection. It is equal to the difference between the Q-A₂ interval and the left ventricular ejection time. In this series of 50 normal subjects, the pre-ejection period showed a poor correlation with

heart rate ($r = -0.30$). The regression equation was similar to that calculated by Weissler *et al.* (1968) in their series of normal subjects (Fig. 2).

The preceding measurements have used the electrocardiogram, the phonocardiogram, and the carotid pulse wave. For indirect measurement of the isovolumetric contraction time of the left ventricle a simultaneous apex cardiogram is also recorded. Spodick and Kumar (1968b) have studied the use of the simultaneous apex cardiogram and carotid pulse wave to measure the isovolumetric contraction time of the left ventricle and considered three definitions: firstly, the time interval from mitral valve closure to the onset of left ventricular ejection; secondly, the time interval from the onset of left ventricular pressure rise, which precedes mitral valve closure, to the onset of ejection (Wiggers, 1921); and thirdly, the time interval from the onset of left ventricular contraction or movement, which precedes the rise of pressure, to the onset of ejection. This third definition is that used by Spodick and Kumar (1968b). It has been described previously by Oreshkov (1965) and we have used it in this study. It entails the use of the apex cardiogram, which is the only simple clinical method available for detecting the earliest movement of the left ventricle.

At the onset of left ventricular systole the ventricular wall begins to move before the pressure in the left ventricle starts to rise. This early motion of the left ventricular wall is first detected by the apex cardiogram. Tafur,

FIG. 3 Simultaneous tracings of the central aortic pressure pulse recorded with an intra-cardiac micromanometer, and the indirect carotid pulse recorded with a piezocrystal transducer. The top tracing shows an intra-aortic phonocardiogram (PCG) and the third tracing shows an external PCG recorded at the second left intercostal space parasternally (2L). Left ventricular ejection time measured from both the aortic and carotid pulse waves was 230 msec. B = onset of the aortic pressure pulse; C = onset of the indirect carotid pulse; D = onset of the aortic component of the second heart sound (A₂); E = the nadir of the dirotic notch (DN) of the indirect carotid pulse. The intervals B to C and D to E both equal 30 msec.

findings differ from those of Willems, De Geest, and Kesteloot (1971) in dogs, and of Bush *et al.* (1970) in man, who failed to show any delay between the onset of the upstroke of the apex cardiogram and the onset of the rise of left ventricular pressure.

This definition of isovolumetric contraction time includes the total period of left ventricular contraction before ejection, i.e. the period of rising left ventricular intramural tension and the period of rising left ventricular intracavitary pressure up to the onset of aortic valve opening. It includes the preisometric contraction phase of Wiggers (1921).

The period of isovolumetric contraction ends with the onset of ejection. This is identified by recording the onset of the carotid pulse

FIG. 4 Simultaneous tracings of the apex cardiogram (ACG) (lower tracing) and of the left ventricular pressure pulse recorded with an intracardiac micromanometer (upper tracing) from a patient with idiopathic cardiomyopathy. The top tracing shows an intracardiac phonocardiogram recorded from the left ventricle and the second tracing an external phonocardiogram. A = the atrial displacement impulse of the apex cardiogram; a = the left atrial pressure pulse recorded from the left ventricle; 1 = onset of the QRS complex of the electrocardiogram; 2 = onset of the apex cardiogram; 3 = onset of the left ventricular pressure pulse. The apex cardiogram clearly precedes the left ventricular pressure pulse by the time interval between 2 and 3, which is 15 msec. S₁, S₂, S₃, S₄ are the first, second, third, and fourth heart sounds.

Cohen, and Levine (1964) first noted this difference in 2 patients with simultaneous records of the apex cardiogram and left ventricular pressure. The upstroke of the apex cardiogram preceded the onset of the rise in left ventricular pressure by 18 milliseconds. Inoue *et al.* (1970) found experimentally in dogs that the onset of the apex cardiogram preceded the rise of left ventricular pressure by 17 msec. In the 7 patients we have studied with simultaneous records of the apex cardiogram and left ventricular pressure, the upstroke of the apex cardiogram preceded the rise of left ventricular pressure by an average of 20 msec (Fig. 4 and Table 2). This agrees with the findings of Tafur *et al.* (1964) and confirms their observations. However, our

wave simultaneously with the apex cardiogram and subtracting the time taken for the pulse wave to travel from the central aorta to the carotid artery (Fig. 1). This time lag, the pulse transmission time, appears to be the same at the onset as at the end of ejection (Harrison *et al.*, 1964), and equal to the time interval between the aortic second sound and the nadir of the carotid incisura. We have been unable to find any data to substantiate this point and therefore we studied 7 patients using simultaneous recordings of central aortic pressure and the indirect carotid pulse. The delay between the onset of the aortic pressure pulse and the right carotid pulse wave was on average 23 msec (range 19–29), (Table 2 and Fig. 3). The interval between the aortic second sound and the nadir of the carotid incisura in these patients was also on average 23 (range 16–30) msec. These two measurements were therefore in close agreement, thus validating the use of the A_2 – incisura interval as an accurate measure of the pulse transmission time from the central aorta to the carotid artery.

The isovolumetric contraction time in the 50 normal subjects studied was 70 ± 9.5 msec (range 51–90). This agrees with the findings of Oreshkov (1965) who found it to be 67 ± 15 msec (range 40–90) and Spodick and Kumar (1968b) who found it to be 70.9 ± 15.8 msec (range 40 to 90). There was no significant relation between the isovolumetric contraction time and heart rate, and our findings confirm those of Oreshkov (1965) who also found no relation to heart rate in a study of 40 normal subjects. This differs from the experimental studies of Wallace *et al.* (1963) who found that the isovolumetric contraction time in dogs was significantly shortened by increasing the heart rate.

Serial daily studies of the phases of left ventricular systole in normal subjects, before and after exercise, have shown no deviation of the measurements from the normal range with heart rates varying from 50 to 120 beats a minute. Observations in the same subject are reproducible from day to day.

The methods described in this paper provide a simple non-traumatic technique for measuring cardiac performance. Measurements can be repeated as often as required without any disturbance or risk to the patient. The normal measurements and their relation to heart rate provide a basis for comparison with the findings in cardiac disease.

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