Isovolumic contraction time and isovolumic contraction time index in mitral stenosis

Study on basis of polygraphic tracing (apex cardiogram, phonocardiogram, and carotid tracing)

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The left ventricular isovolumic contraction time (IVCT) is divided by the mitral closure sound (MI) into two components: the initial phase of ventricular contraction, before the atrioventricular valves close (C-MI interval), and a second pre-ejection phase, between mitral closure and aortic opening (MI-E interval). In severe pure or predominant mitral stenosis (with a diameter of the mitral orifice of 15 mm or less) there is a prolongation in the first component (C-MI interval) and a diminution in the second (MI-E interval) while the total IVCT remains practically unaltered.

The $\frac{C-MI}{MI-E}$ ratio or IVCT index is significantly increased in severe pure and predominant mitral stenosis in comparison with the normal values and those in pure mitral regurgitation.

The true isovolumic contraction time (IVCT) is the entire systolic pre-ejection period of ventricular contraction; it begins with the earliest ventricular movement and ends with the onset of ejection. This time interval comprises the initial phase of ventricular contraction (before the atrioventricular valves close) and the pressure elevation time (classic IVCT). It is susceptible to external measurement by using apex cardiogram, phonocardiogram, and carotid tracing. The IVCT can be measured from the onset of the systolic wave on the apex cardiogram (C point) to the calculated ejection point (E point) (Oreshkov, 1965). This method proved to be the optimum one for measuring IVCT on the basis of atraumatic techniques (Spodick and Kumar, 1968). Recent studies showed that the above-mentioned interval coincided exactly with the directly measured IVCT (Bush *et al.*, 1970).

The present study was undertaken to understand more clearly certain alterations in IVCT in mitral stenosis.

Subjects and methods

Studies were performed in 54 patients with mitral valve disease. Patients with aortic valve lesions or systemic hypertension were previously excluded.

There were 43 women and 11 men. Ages ranged from 16 to 53 years, with an average of 33.5 years. Thirty-six patients had pure mitral stenosis (MS), 12 had predominant mitral stenosis (MSmr), 2 had mitral stenosis and regurgitation approximately of an equal degree (MSMR), and 4 had pure severe mitral regurgitation (MR). Among the patients with MS, 34 had severe MS (diameter of mitral orifice of 2 to 15 mm), and 2 had milder MS (diameter of 20 mm). In 48 patients the diagnosis was confirmed by cardiac operation, and in 6 by right heart catheterization. Only 9 patients had received digitalis for a week before the study. There was a control group of 40 normal subjects, 20 women and 20 men. Their ages ranged from 16 to 52 years, with a mean age of 29 years.

The left ventricular apex cardiogram, the phonocardiogram, and the right external carotid tracing were recorded simultaneously with the patient lying in the left lateral decubitus position in apnoea at the end of a normal expiration. Pulse wave (linear) condenser microphones (Boucke-Brecht) for recording apex cardiograms and carotid tracings, and a crystal microphone for recording phonocardiograms were used, connected to a direct-writing multichannel recorder Hellige (Model 9400/6). The records were taken at a paper speed of 50 mm per second.

The following time intervals were measured (Fig. 1): *isovolumic contraction time (IVCT)* or *C-E interval*, from the onset of the systolic wave (C point) on the apex cardiogram to the carotid upstroke minus the delay time of the carotid pulse wave (calculated ejection point or E point)

Received 16 August 1971.

FIG. 1 Records taken from a normal subject (A) and a patient (B) with severe mitral stenosis (diameter of mitral orifice 7 mm). In the normal subject the IVCT (or $\frac{C-MI}{MI-E}$ ratio) is 0.75, while in the patient this index is 3.5. CT=carotid tracing; ACG=left ventricular apex cardiogram; PCG-MF=phonocardiogram-medium frequency; E=calculated ejection point; DT=delay time of the carotid pulse wave; C=onset of contraction; I=first heart sound; 2=second heart sound; C-I=initial phase of ventricular contraction (before the atrioventricular valves close); I-E=second pre-ejection phase (between mitral closure and aortic opening); IVC=isovolumic contraction time.

(Oreshkov, 1965); the delay time was measured from the beginning of the aortic component of the second heart sound to the carotid incisura; initial phase of ventricular contraction or C-MI interval, from the C point to the onset of the main vibrations of the mitral component of the first heart sound (MI); pressure elevation time or MI-E interval, from the onset of the main vibrations of MI to the E point. For all measurements a minimum of three consecutive cycles was used. The $\frac{C-MI}{MI-E}$ ratio or IVCT index was calculated as well.

Results

The results are shown in Fig. 2 and 3. As may be seen in Fig. 2, the IVCT was approximately the same in the normal subjects and in the patients with mitral valve disease. It was significantly shorter in both patients with

FIG. 2 Mean values of the IVCT in normal subjects (NS) and patients with mitral valve disease: pure mitral stenosis (MS), predominant mitral stenosis (MSmr), mitral stenosis and regurgitation approximately of an equal degree (MSMR), and pure severe mitral regurgitation (MR). The bars represent standard deviation of the mean values of IVCT, and its components (C-MI and MI-E intervals). In MS and MSmr there is a prolongation in the first component (C-MI interval) and a diminution in the second (MI-E interval) while the total IVCT remains practically unaltered.

MSMR only - mean 50 (35-65) $\pm$ 21.2 msec. The rest of the mean values of IVCT varied between 68.50 (40-90) $\pm$ 13.6 msec (in the control group) and 71.45 (40-90) $\pm$ 13.4 msec (in severe MS). At the same time, in the patients with pure or predominant MS there were considerable alterations in both components of IVCT, the initial phase of ventricular contraction (C-MI interval) and the pressure elevation time (MI-E interval). These changes were characterized by a prolongation in the C-MI interval and a diminution in the MI-E interval, and were expressed by the $\frac{C-MI}{MI-E}$ ratio or IVCT index (Fig. 3). The IVCT index was significantly augmented in severe MS, MSmr, and MSMR (Table). The mean IVCT index in severe MS (2.56 (0.82-9.0) $\pm$ 1.81) was more than four times

FIG. 3 Mean values of the IVCT index (or $\frac{C-M_1}{M_1-E}$ ratio) in units (U) in normal subjects (NS) and patients with mitral valve disease. The IVCT index is significantly increased in pure mitral stenosis (MS) and predominant mitral stenosis (MSmr) in comparison with the normal values and those in pure mitral regurgitation (MR).

the normal ($0.57 (0.29-1.50) \pm 0.39$), though no correlation was established between this index and the long diameter of the mitral orifice ($r=0.10$). The mean values of the IVCT index in milder MS and MR did not differ significantly from these in the control group.

All the three time intervals measured remained relatively constant over a wide range of heart rates. For these reasons no corrections for heart rate were employed. This is in agreement with the findings of other authors (Ježek, 1963; Harrison *et al.*, 1964; Weissler, Harris, and Schoenfeld, 1968) concerning the M_1-E interval (classic IVCT) in particular.

Discussion

The very close values of IVCT in both the normal subjects and the patients with MS and MSmr indicate that the rate of left ventricular pressure rise in these valve lesions is not essentially changed. The duration of the components of IVCT is altered only. The initial phase of ventricular contraction or $C-M_1$ interval is prolonged because of pressure elevation time or M_1-E interval. In fact, the term 'pressure elevation time' is not cor-

TABLE IVCT and IVCT index: statistical data for reliability of differences between various groups examined

Groups	IVCT		IVCT index	
	t	p	t	p
Severe MS and normal subjects	0.94	<0.50	6.32	<0.001
Milder MS and normal subjects	1.12	<0.50	1.83	<0.10
MSmr and normal subjects	0.10	>0.50	3.77	<0.001
MR and normal subjects	0.16	>0.50	1.40	<0.25
Severe MS and MR	0.15	>0.50	5.61	<0.001
MSmr and MR	0.19	>0.50	3.19	<0.01

rect, since the left ventricular pressure starts to rise from the very beginning of IVCT (Dallocchio *et al.*, 1965). The pressure curve follows the apex cardiogram ascent at an average difference of only 0.7 msec (Tavel *et al.*, 1965). Obviously, in severe MS or MSmr most of the left ventricular pressure rise during IVCT occurs before the first heart sound (before the atrioventricular valves close) while normally this interval ($C-M_1$) is usually shorter than the next one (M_1-E) (Fig. 1 and 2). It is well known that with MS mitral closure is late (Weiss and Joachim, 1911) because of the time taken by the left ventricle to exceed the raised left atrial pressure (Leatham, 1970). The delay in mitral component of the first heart sound prolongs the $C-M_1$ interval. The latter shows the actual delay of the mitral component while the diagnostic value of transformation time ($Q-M_1$ interval) is diminished by the electromechanical delay (Oreshkov, 1967, 1970). The abbreviation of the M_1-E interval is also due to the delay in M_1 : with diastolic pressure gradient across the mitral valve the delayed M_1 appears at a higher left ventricular pressure than it normally does; hence, after mitral closure a shorter time is necessary for the intraventricular pressure to exceed aortic diastolic pressure. Experimental studies (Braunwald, Sarnoff, and Stainsby, 1958; Wallace *et al.*, 1963) and clinical observations (Weissler *et al.*, 1968) in normal subjects and patients with arteriosclerotic heart disease, hypertensive cardiovascular disease, and primary myocardial disease showed that a lowering in stroke volume prolonged the M_1-E interval. Obviously, the diminished stroke volume relative to severe MS, because of the already mentioned cause, had not the same effect on the duration of M_1-E interval. Other factors, such as diminished aortic diastolic pressure or heart rate (Sokolova, 1964; Warembourg *et al.*, 1964), did not appear to be responsible for the difference in duration of M_1-E interval in the normal subjects and the patients in the present

study. The mean diastolic arterial pressure was the same in both groups, the normal subjects (74.4 (60–90) mmHg) and the patients with severe MS (74.4 (60–85) mmHg). The heart rate in the patients with MS (mean 76.6 (53–105) beats per minute) was insignificantly faster than in the control group (mean 74.2 (45–125) per minute). Abnormalities in myocardial contractility may prolong the M1–E interval (classic IVCT) (Feldman, 1960; Sokolova, 1964; Warembourg *et al.*, 1964; Weissler *et al.*, 1968). In the present study the M1–E interval in the patients with heart failure (28 persons) was also shortened (mean $27.7 (10-50) \pm 12.7$ msec) but less than in the patients without heart failure (mean $25.8 (10-50) \pm 13.1$ msec; $P < 0.50$).

The $\frac{C-M1}{M1-E}$ ratio or IVCT index was significantly increased only in the patients with MS or MSmr with a diameter of the mitral orifice of 15 mm or less. This is in agreement with the findings of Schlant (1970) that the severity of the altered haemodynamics of pure stenosis of the mitral valve increases rapidly as the valve area becomes narrowed to 1.5 cm² or less. Therefore, the IVCT index may be used in the mechanocardiographic diagnosis of severe mitral stenosis.

I am indebted to Professor D. Dimitrov, Director of the Department of Cardiac Surgery (Postgraduate Medical School, Sofia), where the patients for the present study underwent operation and catheterization.

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