Effects of Open Heart Surgery on End-Diastolic Pressure-Diameter Relations of the Human Left Ventricle

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SUMMARY Curves relating left ventricular end-diastolic pressure (LVEDP) to echocardiographically determined end-diastolic diameter (LVEDD) were obtained before and after ischemic arrest in 15 patients during open heart surgery. LVEDP ranged from 0–20 mm Hg during routine operation of the heart-lung machine. Ischemic arrest ranged from 0–94 minutes.

In eight patients averaging 21 ± 7 (SEM) minutes of arrest, we saw no change in LVEDP-LVEDD curves. In five patients averaging 55 ± 15 minutes of arrest, we noted temporary alterations in EDP-EDD curves (p < 0.05). The curves returned to normal within 30–60 minutes after ischemia. In two patients with an average of 66 minutes of arrest, we observed changes in the EDP-EDD curves which did not revert to normal. Available data did not allow us to distinguish between impaired ventricular relaxation and a true change in ventricular compliance as a cause of the shift in the EDP-EDD curve, but ischemia appears to be a major factor in the observed changes.

Increased LVEDP after ischemic arrest during open heart surgery may reflect a decrease in left ventricular compliance, rather than an increase in heart size. The probability of altered LVEDP-LVEDD relations appears to depend on the duration of ischemic arrest.

ALTHOUGH ELEVATED LEFT VENTRICULAR END-DIASTOLIC PRESSURES (LVEDP) are often observed after ischemic arrest during heart surgery, the relevance of changes in myocardial compliance has not been defined. Numerous observations of ischemia-induced reduction of left ventricular (LV) compliance in experimental animals and humans have recently been reviewed.1, 2 Serial measurements of compliance in the clinical setting are technically difficult, and the relationship of LVEDP to chamber volume or diameter has been examined only over a very narrow range of values during physiologic interventions in humans.

Recently, standardization of echocardiographic techniques has permitted reproducible measurements of the minor semiaxis of the left ventricle to be conveniently obtained in the open chest.3, 4 This development, combined with the wide variation in LVEDP produced by normal operation of the heart-lung machine during open heart surgery, provides a natural laboratory to study alterations in LV compliance. Accordingly, we examined the effect of myocardial ischemia during cardiac surgery on diastolic properties of the human left ventricle.

Methods

We studied the relationship between left ventricular end-diastolic diameter (LVEDD) and LV filling pressure in 38 patients during cardiac surgery. In 15 patients we obtained technically satisfactory data both before and after 0–90-minute periods of ischemic cardiac arrest, allowing us to determine the effects of ischemia on diastolic properties of the left ventricle.

Informed consent for intraoperative studies was obtained before surgery. Patients at high risk of embolization due to thrombus or valvular vegetations were excluded. Instrumentation was not introduced until each patient had been anticoagulated with 90 mg/m² BSA heparin, our standard technique for cardiac surgery. Once anticoagulation and arterial cannulation for extracorporeal circulation had been performed, a 3.5 mHz retrocardiac transducer (Aerotech Laboratories, Lewistown, Pennsylvania) 3 mm thick was inserted into the posterior pericardium. The transducer was gas-sterilized and mounted, in the operating room, in an autoclaved Silastic disk (382 Medical Grade Elastomer, Dow Corning Corp, Midland, Michigan) to stabilize motion and position of the transducer. This two-part sterilization avoided autoclave-induced damage to the echo transducer and entrapment of toxic gases by microbubbles in the Silastic.

M-mode echocardiograms (ECT Echocardiotracer, Metrix, Inc, Denver, Colorado) were photographed (Tektronix C-59 Camera, Tektronix, Inc, Beaverton, Oregon; Film Type 107, Polaroid Corp, Cambridge, Massachusetts) or recorded with an optical oscillograph (1858 CRT Visicorder, Honeywell, Inc, Denver, Colorado). Intraventricular echo clouds5 were produced by injecting 7 ml of normal saline into the
left atrium when necessary to identify the endocardial limits of the left ventricle.

Pressure data were recorded either with a left atrial needle or a catheter-tip micromanometer (PE-350, Millar Instruments Inc, Houston, Texas); mean left atrial pressure (LAP) and LVEDP were used, respectively. The micromanometer was introduced retrogradely across the aortic valve through a site used to vent air from the ascending aorta during surgery, or through a decompression vent (Ferguson Vent, Sherwood Medical Industries, St. Louis, Missouri) introduced into the left ventricle via the right superior pulmonary vein and mitral valve. For right ventricular pressures, a micromanometer was placed across the tricuspid valve with a pursestring suture in the lateral wall of the right atrium. Micromanometers were calibrated electrically, and calibration was confirmed before insertion with a fluid column at 0-, 7- and 13.6-cm immersion. After heparinization, we introduced pressure transducers into the operative field and cannulated the ascending aorta for cardiopulmonary bypass.

After proper placement of instruments and initial recordings, the superior and inferior vena cavae were cannulated. Cardiopulmonary bypass was then started, and cardiac surgical procedures conducted according to standard protocols. The recording equipment did not interfere with surgery. Variation of LVEDP for measurement of LV diastolic properties was allowed to occur, and additional volume manipulations with the heart-lung machine were not required. In general, LVEDP fell from the ambient level (10–15 mm Hg) to zero as total cardiopulmonary bypass was started and the left ventricle vented. Ischemic arrest was then induced by cross-clamping the ascending aorta proximal to the cannula returning blood from the heart-lung machine. Techniques used for myocardial protection during ischemia are identified in table 1. Topical hypothermia (TH) — irrigation of the pericardial cavity with saline cooled to 4°C — was most commonly used. In two patients, we used potassium cardioplegia (KCl) — intracoronary injection of 400 ml of solution* at 4°C every 30 minutes during ischemia. When surgical procedures requiring cardiac arrest were completed, the aortic cross-clamp was removed, and the heart was defibrillated. Data recording resumed when the left atrium had been closed and maneuvers requiring cardiac compression or displacement had been completed. After cardiopulmonary bypass, LVEDP increased to 20 mm Hg during transfer of volume to the patient from the reservoir of the heart-lung machine. Research instruments were removed before decannulation and reversal of heparin.

No deaths or complications occurred related to these studies. The echo transducer did not appear to impair LV performance by external compression and did not produce ventricular arrhythmias, except for a few premature contractions during initial positioning.

Position of the echo transducer generally remained stable in the posterior pericardium.

Curves reflecting diastolic properties were constructed from intraoperative recordings of LVEDD and LV filling pressure (LVEDP or mean LAP). Each patient was used as his or her own control, and correlation with absolute ventricular volumes was not included in this study. The line best fitting the observed data was drawn with a French curve. Changes in compliance were analyzed by comparing pressure-diameter curves at the beginning of cardiopulmonary bypass with data at the end of surgery, during and after weaning from the heart-lung machine.

Because techniques used to analyze LV stiffness changed considerably with the availability of strip chart recordings, the studies have been divided into two categories. Studies in category I are based on LVEDD from Polaroid photographs and separate recordings of LV filling pressures. Despite the use of event markers, the task of correlating individual beats was tedious, so LVEDD and filling pressure were averaged over 5–7 beats and plotted as a single point. Furthermore, LV filling pressure in category I was mean LAP more often than LVEDP by micromanometer. Studies were based on LAP only when it was felt to reflect LVEDP accurately, i.e., LAP was not used in patients with mitral valve disease.

In category II studies, LVEDP and LVEDD could be directly correlated from simultaneous strip chart records and plotted for individual beats, resulting in a marked increase in the number of data points. Filling pressure in category II was LVEDP by micromanometer in all studies (table 1).

An important difference between studies in these two categories is that continuous strip chart recording allowed us to follow the original endocardial echoes precisely, even when inolding of the LV wall with decreasing volume produced new echo reflections.

Results

Data for the 15 patients studied are summarized in table 1. Six patients had surgery for coronary artery disease, three had aortic valve replacement, three had mitral valve replacement, one underwent pulmonary valvulotomy, one had mitral commissurotomy, and one had combined aortic valve replacement and coronary artery bypass. In eight patients (group A), there was no detectable change in end-diastolic LV pressure-diameter curves compared before and after recovery from 0–67 minutes of ischemic arrest. Four patients in group A had short periods of ischemic arrest alternating with 5–15-minute periods of reperfusion. In five patients (group B), a temporary shift to the left was detected in the LV pressure-diameter curve, toward higher pressures at any given diameter. This change, occurring after 16–94 minutes of ischemic arrest, was reversible within 30–40 minutes of reperfusion. In two patients (group C), pressure-diameter curves were persistently shifted to the left, even after weaning from cardiopulmonary bypass.

*Cardioplegia solution: 1000 ml Normosol R, 50 mEq NaHCO3, 25 g albumin, 30 mEq KCl.
The longest period of continuous ischemia averaged 21 ± 7 minutes (SEM) for group A and was statistically different from the duration of continuous ischemic arrest in group B (55 ± 15 minutes, p < 0.05).

Maximal postoperative serum levels of myocardial enzymes (SGOT, LDH and CPK) are given in table 1. No statistically significant differences in enzyme levels are apparent for groups A, B and C. Values for CPK-2 (CPK-MB) are listed in parentheses as absent (−), trace (TR), or present (+). A trend toward increased CPK-2 observed in groups B and C is not readily subject to statistical analysis.

Figure 1 is a representative M-mode echocardiogram recorded during open heart surgery by the retrocardiac echo transducer (study ES-2). Because we used the retrocardiac technique, the posterior wall of the left ventricle is closest to the transducer and therefore appears at the top of the image, while the septum appears toward the bottom. Thus, the image is inverted compared with standard M-mode echocardiograms. Synchronous motion of opposing walls of the left ventricle is apparent, and measurement of EDD synchronous with the R wave of the ECG is identified.

In 15 patients we obtained satisfactory studies of the relationship between LVEDP and LVEDD at the beginning and the end of cardiopulmonary bypass, and we could examine the effect of the surgical procedure on diastolic properties of the left ventricle. In most patients, LV pressure-diameter relations appeared unchanged. Representative examples are given in figures 2 and 3.

Figure 2 shows an end-diastolic pressure-diameter curve obtained during coronary bypass (ES-2, table 1). Mean LAP is plotted on the ordinate. Each data point represents an average of five cycles recorded photographically. Comparison of curves obtained at
the beginning and the end of cardiopulmonary bypass indicates no change in LV diastolic pressure-diameter relations despite three periods of ischemic arrest (15, 15 and 18 minutes) for coronary bypass grafting (table 1).

Figure 3 shows similar data recorded during mitral valve replacement (study ES-8). In this example, end-diastolic pressure recorded by catheter-tip micromanometer is plotted on the ordinate. Again, there is no apparent change in LV diastolic properties, despite consecutive 22- and 13-minute periods of ischemic arrest.

In seven of the 15 patients studied, temporary or persistent changes in EDD-EDP relations were observed after ischemic arrest. Related observations are illustrated in figures 4-7.

Figure 4 shows pressure-diameter data during mitral valve replacement (ES-11a). Continuous strip chart recording allowed beat-to-beat correlation of EDD by echo and EDP by micromanometer. A nitroprusside drip was used when beginning cardiopulmonary bypass because of heart failure. Myocardial protection was provided by KCl cardioplegia during a 40-minute ischemic arrest. Initial recordings 10–15 minutes after reperfusion show a shift toward higher LVEDP and smaller LVEDD than the preoperative data. This shift was temporary, however, and the preoperative curve was restored after approximately 10 minutes. Pharmacologic circulatory support was not required after successful mitral valve replacement. Strip chart recordings in figure 5 show the increase in LVEDD during recovery from ischemia. An increase in LVEDD is observed despite a small increase in RVEDP and a decrease in LVEDP. LV systolic wall motion was impaired. Core temperature and arterial pressure did not change appreciably. Peak negative dP/dt was also essentially unchanged.

Figure 6 gives similar EDP-EDD data recorded

![Figure 3](image-url) **Figure 3.** Left ventricular (LV) end-diastolic diameter vs left ventricular end-diastolic pressure (LVEDP) during mitral valve replacement. Open circles represent data recorded before ischemic arrest, and solid circles represent data recorded after ischemic arrest, during weaning from cardiopulmonary bypass. There is no apparent alteration in LV pressure-diameter relations during surgery. For additional information, see text and table 1.

![Figure 4](image-url) **Figure 4.** Left ventricular end-diastolic pressure (LVEDP) vs left ventricular end-diastolic diameter (LVEDD) during mitral valve replacement. Figure 4 is similar to figures 2 and 3, but includes many more data points facilitated by improved recording techniques (see text). Data represented by open squares (before ischemia) and solid triangles (at end of cardiopulmonary bypass) describe similar curves relating EDP and EDD. Solid circles represent data recorded during recovery from ischemia, within 10 minutes of defibrillation, after removal of aortic cross-clamp. These data are skewed toward smaller EDD and higher EDP, suggesting increased stiffness or decreased compliance of the left ventricle.

![Figure 5](image-url) **Figure 5.** Representative oscillograph tracings during change in diastolic left ventricular (LV) wall properties. Data illustrated correspond to end-diastolic pressure-end-diastolic diameter (EDP-EDD) curves shown in figure 4. Data in the left panel were recorded within 10 minutes of removal of the cross-clamp and reversal of ischemic arrest. Data in the right panel were recorded 10 minutes later, during weaning from cardiopulmonary bypass. LVEDP, RVEDP, heart rate and rhythm, mean arterial pressure, and core temperature are similar in both tracings. The principal dissimilarity is the increase in LV diameter during the recovery from ischemia, which is believed to reflect an increase in LV compliance. Peak negative dP/dt is approximately 460 mm Hg/sec in both panels. See text and table 1 for additional data.
during aortic valve replacement and coronary bypass grafting (ES-22A). LV function was impaired preoperatively. Cardioplegic protection of the left ventricle was used during 94 minutes of ischemia. The pressure-diameter curve again shifted temporarily to the left after ischemic arrest. In this patient the change in diastolic properties was associated with impaired systolic performance and 5-minute periods of cardiopulmonary bypass with venting of the left ventricle allowed ventricular recovery. After each of these intervals, we observed an improvement in systolic performance and a shift of the stiffness curve toward normal. Dopamine was required for inotropic support in the immediate postoperative period.

Figure 7 shows data obtained during aortic valve replacement (ES-13). Topical hypothermia without cardioplegia was used for myocardial protection, and total arrest time was 59 minutes. The LVEDP-LVEDD curve shifted to the left and did not return to normal during the study. Postoperative LV function was markedly depressed in this patient, and isoproterenol was used for myocardial support. Subendocardial ischemic injury was suggested by elevated enzymes (table 1) without electrocardiographic evidence of infarction.

Discussion

Our results indicate that alterations in end-diastolic pressure-diameter relations of the human left ventricle may be observed after ischemic arrest during open heart surgery. The curves shift to the left, toward decreased ventricular diameter and increased filling pressure, suggesting that ischemic arrest can increase stiffness and reduce compliance of the myocardium of the left ventricle. We observed decreased LV compliance in seven of 15 patients studied. This change was temporary in five of seven, and reverted to normal during the study. In general, diastolic wall properties were not altered unless continuous ischemic arrest was more than 35 minutes. However, sensitivity to ischemia varied, since only 16 minutes of ischemia temporarily reduced compliance in study ES-9. Conversely, no compliance change was noted in ES-11, despite 67 minutes of ischemia (table 1).

Since the first observations of rigor mortis, ischemia has been identified as the cause of a time-related increase in skeletal muscle stiffness, a phenomenon ascribed to progressive rigid cross-linkage of actin and myosin after destruction of ATP and other sources of high-energy phosphate bonds. In the myocardium, similar changes have been demonstrated in papillary muscle studies. In cardiac surgery, a parallel phenomenon is the “stone heart” syndrome, which results from severe subendocardial ischemic injury in hypertrophied left ventricles.

In excised, arrested dog hearts, the myocardium becomes stiffer with time, so that a given volume is associated with increasing pressures. The onset of increasing stiffness and decreasing compliance requires 1–3 hours at normothermia and can be delayed as much as 5 hours with cooling to 4°C.

In dogs on cardiopulmonary bypass, ischemic injury is associated with reduced compliance, measured with an intraventricular balloon. Myocardial injury may be delayed and minimized by topical hypothermia and increased coronary perfusion pressure. Counterpulsation and potassium cardioplegia are also effective. Other observers have not been able to detect compliance changes.
Acute ischemia in papillary muscles and intact myocardium may increase compliance under some experimental conditions, although evidence is not entirely consistent.\(^{17-22}\) However, acute ischemia has been shown to impair myocardial relaxation after systole.\(^{23-27}\)

Numerous additional variables may reduce apparent compliance.\(^{28-33}\) In animals these include elevated right ventricular end-diastolic pressure (RVEDP),\(^{34-36}\) increased arterial pressure,\(^{37-40}\) temperature reduction,\(^{41-45}\) pharmacologic or reflex influences,\(^{44-45}\) and alterations of ventricular geometry produced by surgery or atrial clamping.\(^{46, 50}\) Removal of the pericardium increases apparent LV compliance.\(^{51}\)

Clinical studies based on angiography or echocardiography suggest that compliance may be decreased by myocardial infarction,\(^{2, 52-55}\) aneurysm formation,\(^{56}\) hypertrophy,\(^{2, 57}\) and angina.\(^{58}\) Compliance may be increased by propranolol\(^{59}\) and coronary revascularization,\(^{60}\) in the presence of ischemic heart disease. Moreover, nitroprusside may increase compliance in congestive heart failure.\(^{43}\) Reduced LV compliance has been postulated to explain pulmonary edema in the absence of cardiomegaly in patients with ischemic heart disease.\(^{61}\) Dynamic properties of the diastolic left ventricle become important if compliance is measured during rapid filling.\(^{42, 64}\)

We can only speculate whether the changes in apparent stiffness of the myocardium reported here are induced by ischemia or other influences. In some studies, and in figures 4 and 5, factors such as arterial pressure, heart rate and rhythm, temperature and RVEDP are so closely matched that ischemia-induced alterations of the intrinsic properties of the myocardium appear to be the primary influence on pressure-
diameter relations. This hypothesis is supported as well by the association of reduced compliance with prolongation of ischemic arrest beyond 30 minutes.

We cannot clearly distinguish a true decrease in LV compliance after ischemic arrest from an apparent decrease due to impaired myocardial relaxation. However, peak negative dP/dt was unchanged in the three patients with LV micromanometers in place during decreased compliance (ES-11A, 21A and 22A), which suggests that an intrinsic reduction of compliance was involved, but considerably more data is needed for confirmation.

What important artifacts of the measuring technique might influence our results? Accuracy of echocardiographic dimensions of the left ventricle in the closed chest is well established, and close correlation between echocardiographic (using V = echo diameter, cubed) and angiographic ventricular volumes has been reported.65, 66 Open-chest studies from our laboratory2 and the work of others6 indicate that echo determinations of ventricular diameter and volume from the retrocardiac position are accurate. Furthermore, in the present study, accuracy of absolute dimensions is not an issue, since the patient is used as his or her own control and only changes in ventricular diameter are reported. However, movement of the echo transducer in the posterior pericardium could produce artifacts of measurement. Such movement is an even more likely source of error when the heart is displaced from the pericardial cavity (as in circumflex coronary grafting), but the heart was not displaced from the pericardium in patients in groups B and C. In fact, experience suggests that with some attention the position of the transducer is reasonably stable in the posterior pericardium, due to adherence of the Silastic to surrounding tissues and the weight of the heart itself. Additional assurance is provided by precise return to control curves after recovery from ischemia, as shown in figure 4.

A change in diastolic compliance would be expected to steepen the slope of the EDP-EDV curve and to shift the curve to the left. We think the absence of such changes in slope in figures 6 and 7 reflects a relatively small range of EDP recorded during recovery from ischemia and substantial data scatter which impedes accurate interpolation of slopes. Another issue is variation in echo patterns observed with markedly decreased LV volume, which may reflect infolding of the ventricular wall. We minimized confusion by carefully following the echo reflection originally identified as endocardium. Availability of electronic sector scanning or other methods may provide more sensitive measurements of LV volume for future studies.

Administration of inotropic or vasoactive agents during recovery from cardiopulmonary bypass could alter observed compliance.34, 44-48 Nitroprusside, particularly, has been shown to increase apparent compliance in patients with heart failure in the closed chest, with an intact pericardium.67 Review of our intraoperative records reveals only one study in which significant pharmacologic influences might have occurred. The patient in study ES-11A, who had severe mitral regurgitation, received 44 mg/min nitroprusside during anesthesia before mitral valve replacement, but drug administration was not required after the valve replacement was completed. Since the observed EDD-EDP curves at the beginning and the end of cardiopulmonary bypass are superimposable (figure 4), the effects of nitroprusside apparently were not significant with the pericardium open.

We inserted catheter-tip micromanometers in the right ventricle to determine whether increases in RVEDP during bypass might produce apparent reduction of left ventricular compliance through compression of the interventricular septum.34-36 Such influences appeared to be minimal for the range of RVEDP (2-12 mm Hg) occurring in these patients. The coupling of RV-LV compliance, like the influence of nitroprusside, may be minimized in surgical patients by the open pericardium.

It has been suggested that the decreases in compliance reported above might reflect reduced temperature of the LV myocardium as a predominant factor. While the patients in this study did not have myocardial thermistors in place, subsequent intraoperative and laboratory studies indicate that myocardial-oesophageal temperature gradients are negligible 2-3 minutes after removal of the aortic cross-clamp and resumption of coronary perfusion. Interpretations of possible influences of temperature on compliance in the beating heart may therefore be based, within small limits of error, on core (oesophageal) temperature recorded at surgery.

Possible influences of temperature on the present results are minimal in four of seven patients in groups B and C in whom decreased compliance persisted at 37°C (studies ES-9, 22A, 13, and 21A). Indeed, all of the data indicating reduced compliance in study ES-22A were recorded during an extended period at 37°C necessitated by difficulty in discontinuing cardiopulmonary bypass. The significance of decreased core temperatures in the remaining three patients is unclear but probably minimal, since no data were recorded at core temperatures of less than 34°C. Additional evidence against a primary role for temperature reduction includes the absence of compliance changes during the initiation of cardiopulmonary bypass, when it was often possible to record data to core temperatures of 28°C. In summary, it is not possible to exclude decreased temperature completely from a role in the compliance changes observed, in which hypothermia might contribute to increased myocardial stiffness or delay recovery from other influences (as in myocardial edema after reflow). Most evidence suggests, however, that hypothermia is a secondary influence under our experimental conditions.

It has also been suggested that reduction in LV compliance could reflect increased wall thickness associated with a hyperemic response after ischemia.68 The resolution of wall thickness by our echo technique was not adequate to analyze this possibility. Similarly, the possible influence of hyperosmolarity of
the cardioplegia solution cannot be resolved by our study. However, cardioplegia was not employed in most patients in groups B and C.

No increase in compliance was seen in the six patients with coronary artery disease who underwent revascularization, although reduced compliance has been reported in coronary artery disease.9 This could reflect reduced myocardial work and oxygen consumption during anesthesia and surgery.

An important possible source of reduction in apparent ventricular compliance peculiar to surgical patients is the introduction of rigid prostheses into the base of the heart during valve replacement. Clamping segments of the myocardium alters global compliance,49,50 and so might the rigid sewing ring of a valve prosthesis. This may explain the persistence of the observed reduction of compliance in study ES-21A, since enzyme evidence and clinical performance did not suggest significant cardiac injury. The specific technique (topical hypothermia or cardioplegia) of myocardial protection could also affect compliance,13,14 but the results do not suggest such a relationship (table 1).

At times, LV function is depressed and LVEDP is elevated at the end of cardiopulmonary bypass. These alterations are often reversible, so that LVEDP is close to normal before the patient leaves the operating room. The increased LVEDP could represent compensation for decreased contractility with increased fiber and sarcomere length.10 Alternatively, a reduction in compliance could require elevated LVEDP in order to achieve normal sarcomere length. The increase in LVEDP observed under such circumstances is not necessarily associated with a true increase in heart size. Parallel observations have been reported previously in closed-chest patients with ischemic heart disease.61

The clinical importance of the observed alterations in LVEDP-LVEDD relations depends ultimately on the frequency of related impairment of LV performance. Two of the 15 patients (ES-13 and ES-22A) in this study required inotropic agents and compliance was reduced in both. Compliance changes during ischemic arrest should be investigated, since detection of such changes might lead to better methods for estimating the safe time period for ischemic arrest in individual patients.

We conclude:

1) EDD-EDP curves can be constructed from data derived during open heart surgery to analyze intrinsic stiffness and compliance of the human left ventricle.

2) After periods of ischemic ventricular arrest longer than 30 minutes, the EDD-EDP curve tends to shift upward and to the left; this abnormality is reversible in most cases.

3) The observed shift in the EDD-EDP relation might be explained either by a true decrease in myocardial compliance (increased stiffness) or by an ischemia-related impairment of diastolic relaxation.

4) Increases in LVEDP at the end of open heart surgery might reflect an alteration in ventricular pressure-volume relations rather than an increase in heart size.

5) These observations may provide a method to define the safe period of ischemic cardiac arrest.

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CONSIDERABLE EFFORT has been expended in studies in both animals and humans to determine whether the transverse or minor axis of the left ventricle initially increases during the preejection phase of systole (isovolumic contraction). Although only a limited number of true normal subjects has been studied, there is now general agreement that the transverse diameter of the endocardial cavity in humans decreases during the preejection phase while the epicardial transverse diameter increases because of muscle thickening. These results imply that the shape of the normal human ventricular cavity does not become more spherical during the preejection phase of systole.

The present study was performed to analyze in greater detail the temporal sequence of endocardial wall motion for individual segments and to determine whether a characteristic pattern exists for the sequence of onset of mechanical contraction of the normal human left ventricle.

SUMMARY The sequence for the onset of segmental contraction of the left ventricle was studied in 25 normal patients by analyzing sequential frames obtained at 16.7-msec intervals of right anterior oblique (RAO) ventriculograms by two independent methods. In the first method, we compared the times of onset of contraction of the hemidiameters associated with each of 54 segments with the time of onset of contraction of the average of all the hemidiameters for the ventricular contour. In the second method we used a radial coordinate system and determined relative phase relationships by plotting the motion of each of 54 segments against the average motion of all segments.

The resulting pattern showed that, on the average, the midregion of the inferior wall began to contract 25 msec before the apex and the midregion of the anterior wall began contraction 18 msec before the apex. In 12 of 25 patients the interior and anterior walls both began to contract before the apex. In only one of 25 patients did the apex begin to contract first. This sequence of contraction corresponds to the reported sequence of electrical activation for normal human left ventricles.

Methods

The 25 normal patients used in this study had 1) no valvular or congenital lesions, 2) no significant coronary artery lesions (less than 30% diameter reduction), 3) normal end-diastolic volumes and pressures, 4) normal ejection fractions, and 5) normal ECG or nonspecific S-T-T wave changes. Twenty-one of the 25 patients had no evidence of coronary artery lesions by selective coronary arteriography. Four patients each had a single lesion in a single coronary artery — patients 14, 16 and 20 in the right coronary artery and patient 7 in the left anterior descending — none of which reduced the luminal diameter more than 20–30%.

Before coronary arteriography we obtained left ventricular angiograms in the 30° RAO projection by injecting a 76% solution of meglumine diatrizoate at the rate of 15 ml/sec for 3 seconds. Using a 9-inch image intensifier, we recorded single-plane ventriculographic images at 60 frames/sec (16.7-msec intervals) on cine film, video disc and video tape with a simultaneous ECG tracing. We analyzed only normally conducted beats which did not follow an extra systole.

We analyzed the motion of multiple segments of the endocardial border of the left ventricular silhouette using two independently derived parameters, time of onset of contraction (TOC) and quadratic measure of asynchrony (QMA). To decrease the possibility that the results might be biased by the method of analysis, we obtained the parameters by using different methods for tracing the ventricular chamber contours.
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