Electrophysiological Effects of Acute Ventricular Dilatation in the Isolated Rabbit Heart

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We examined the effects of left ventricular dilatation on epicardial pacing threshold, conduction velocity, and effective refractory period (ERP) in the isolated, retrograde perfused rabbit heart. Left ventricular size was modified by acutely changing the volume of a fluid-filled balloon anchored within the vented left ventricle. Increases in left ventricular volume, associated with increases in left ventricular end-diastolic pressure from 0 ± 1 to 35 ± 2 mm Hg, were not associated with significant changes in pacing threshold or conduction velocity. The left ventricular ERP decreased significantly with an added volume of 1.5 ml (91.4 ± 5.5 msec) compared with starting volume (117.7 ± 3.8 msec, p < 0.01). Right ventricular ERP did not change significantly with increases in left ventricular volume. The left and right ventricular ERPs were comparable at starting volume (117.7 ± 3.8 and 117.6 ± 3.5 msec, respectively; p = NS) but were significantly different with an added volume of 1.5 ml (91.4 ± 5.5 and 112 ± 5.6 msec, p < 0.05). These changes were independent of coronary perfusion pressure and paced cycle length, suggesting that ischemia is an unlikely explanation for the observed effects. Changes in left ventricular volume decreased left ventricular ERP in a regionally heterogeneous manner, increasing the temporal dispersion of recovery over the left ventricle nearly twofold. Induced ventricular arrhythmias (ventricular tachycardia or fibrillation) were significantly more frequent at high (35%) than at low (3%) volumes during left ventricular pacing. We conclude that ventricular dilatation is associated with increased dispersion of refractoriness in this model, a finding that correlates with propensity for reentrant arrhythmias. (Circulation Research 1988;62:554-562)

A relation between ventricular arrhythmias and congestive heart failure is well established clinically. Ventricular tachycardia occurs frequently in patients with congestive failure.1-5 The incidence and grade of ventricular arrhythmia parallel the degree of myocardial dysfunction.6,7 Ventricular tachycardia appears to be a poor prognostic sign in these patients,2,8 and sudden arrhythmic death is common.5,7,8

The pathophysiology of ventricular arrhythmias in patients with congestive heart failure is poorly understood. Congestive heart failure is associated with structural, metabolic, and neurohumoral changes (e.g., myocardial fibrosis, hypokalemia due to diuretic therapy, high levels of circulating catecholamines, etc.) that may be arrhythmogenic.8-9 Another potential cause of arrhythmias in patients with congestive failure is electrophysiological changes that may be induced by ventricular dilatation. Studies in isolated muscle preparations,10,11 and in intact animal models,12-17 have shown that stretch or dilatation can affect action potential duration, refractoriness, and conduction velocity. However, these effects have not been observed consistently, and an arrhythmogenic effect of these changes has not been described.

In the present study, we examined the effects of ventricular volume on myocardial threshold, conduc-
adjustable latex balloon. The balloon was prestretched and of sufficient size that inflation with the volumes used in the study did not result in an elevation of pressure within the balloon. The balloon was placed through the mitral orifice into the left ventricle and secured with a purse-string suture through the mitral apparatus (Figure 1). The balloon was connected through a fluid-filled catheter (i.d. = 1.67 mm) to a pressure transducer (P23Db, Statham, Oxnard, California). Intraventricular pressures during isovolumetric contraction were measured through the balloon with a pressure amplifier (model 13-4615-58, Gould, Cleveland, Ohio) and recorded continuously on a strip-chart recorder (ES 1000, Gould). The zero pressure reference was set at the level of the aortic valve. The volume of the empty balloon was 0.4–0.5 ml. To compare hearts of differing size, intraventricular volume was adjusted in each experiment by first collapsing the balloon to a volume equivalent to an end-diastolic pressure of approximately zero. This volume was defined as "zero pressure" or starting volume. To dilate the ventricle from this starting volume, known volumes of fluid were then added to the balloon. The resolution of volume measurement was 0.1 ml.

Electrophysiological Evaluation

In the first experiment, the sinus node was intact, and the hearts were ventricularly paced at a rate faster than the sinus rate. In subsequent experiments, the sinus node was destroyed by crushing. Epicardial stimulation and recording could be accomplished at the same time through custom-designed plaque electrodes consisting of two close bipolar electrodes (electrodes 1.5 mm diameter, interelectrode distance 4 and 7 mm between pairs) embedded in acrylic. In approximately the same area in all experiments, one plaque electrode was sutured on the midanterior aspect of the left ventricle, avoiding the coronary arteries. A second electrode was sutured on either the right ventricle or in a second left ventricular site. Epicardial electrograms from both electrodes were amplified (model 13-4615-58, Gould) with filter settings of 30–1,000 cycles/sec and were simultaneously recorded on a multichannel electrostatic recorder (ES 1000, Gould) at speeds of 100 and 250 mm/sec.

Pacing was alternately performed in the right and left ventricles at each left ventricular volume with an isolated, programmable stimulator (DTU-201, Bloom Associates, Reading, Pennsylvania) using square-wave pulses of 2-msec pulse width and an amplitude twice diastolic threshold. Effective refractory periods (ERPs) were determined during continuous ventricular pacing at a fixed cycle length. After every 20th drive beat, a ventricular extrastimulus was introduced. The coupling interval (V1–V2) was progressively increased (in increments of 5 msec) from an initial coupling interval of 70 msec until capture was observed. The V1–V2 was subsequently decreased (in decrements of 1 msec) until the extrastimulus failed to capture twice in a row. This coupling interval was defined as the ERP. The standard deviation of ERP determination was ±2.7 msec with this method. Conduction time was defined as the interval between the stimulus artifact on the pacing electrode and the intrinsicoid deflection (midpoint of the most rapid transition of the electrogram) measured on the other electrode. Conduction times were measured during basic-drive pacing.

Experimental Protocol

In most hearts, ventricular pacing was initiated when the sinus node was destroyed. Immediately after instillation of each balloon volume, the pacing threshold was determined and pacing amplitude fixed at twice diastolic threshold. After 2 minutes of continuous pacing, left ventricular pressures were measured, and ERP and conduction times were determined. A coro-

**FIGURE 1.** Diagram of the preparation. See text for additional details.
nary sinus effluent sample was obtained. Pacing, at the same ventricular volume, was then initiated in the other ventricle and the protocol repeated. After completion of the experimental protocol with both left and right ventricular pacing, the balloon volume was changed, and the protocol was repeated at a different left ventricular volume (Figure 2). The order of the volume change was randomized during the study.

In several hearts in each experiment, ventricular pacing was not continuous in order to assess the effects of ventricular volume on the intrinsic ventricular rhythm. After instillation of a given volume, the preparation was allowed to stabilize 1 minute while intrinsic rhythm, rate, and left ventricular pressure were determined. Pacing was then initiated for 2 minutes before measurements were made as above.

 Coronary sinus samples were analyzed for Po2, PCO2, and pH with a Corning (model 165/2, Medfield, Massachusetts) blood gas analyzer. Lactate levels were measured with a commercial enzymatic technique with a coefficient of variation of 7%. Coronary sinus flow rates were approximated by measuring the timed collection of perfusion flow.

**Experimental Series**

**Stability of pacing threshold over time.** We determined the stability of diastolic pacing threshold over time in four hearts, paced at a cycle length of 250 msec and perfused at a pressure of 73 mm Hg. A single epicardial electrode, positioned on the midanterior aspect of the left ventricle, was used. Volumes between 0.5 and 2.0 ml were added to the left ventricular balloon, and pacing threshold was determined. Hearts were then paced at twice diastolic threshold. Diastolic pacing threshold was reetermined five times without changing ventricular volume every 2 minutes (total duration 10 minutes). For all subsequent experiments, pacing threshold was determined immediately after changing balloon volume.

**Effect of increasing left ventricular volume.** The effects of added left ventricular volumes from 0 to 1.5 ml were studied in seven hearts. Hearts were paced at a cycle length of 250 msec and perfused at a pressure of 73 mm Hg. Plaque electrodes were positioned on the midanterior aspect of the left ventricle and on the right ventricle.

**Effect of increased paced cycle length.** Seven hearts were paced at a cycle length of 450 msec with added left ventricular volumes from 0 to 1.3 ml. Conditions were otherwise as described above.

**Effect of increased perfusion pressure.** In five hearts, the pacing cycle length was 250 msec, and the perfusion pressure was raised to 88 mm Hg. Conditions were otherwise as described above.

**Regional changes in left ventricular effective refractory period.** In six hearts, the change in ERP determined with an electrode sutured on the midanterior aspect of the left ventricle was compared to the change in ERP determined with a second electrode sutured on the apical portion of the left ventricle. In an additional six hearts, the midanterior left ventricle was compared with an electrode positioned near the atrioventricular groove on the lateral left ventricle (lateral-basilar left ventricle). All hearts were paced at a cycle length of 250 msec and perfused at 73 mm Hg. The protocol was carried out by alternately pacing the two left ventricular sites and was otherwise as illustrated in Figure 2. The dispersion of repolarization was estimated as the mean (for all animals) of the absolute difference in ERP between the two compared sites at each ventricular volume.

**Statistical Analyses**

All data are presented as mean±SEM unless otherwise indicated. Comparison of ERPs, thresholds, and conduction times at various volumes for the group data was performed with the two-way analysis of variance and the Student-Newman-Keuls multiple comparison test. To characterize the relation between ERP and left ventricular volume, we performed linear regression analysis and obtained a slope and intercept for each heart. We compared mean data for slopes and intercepts under different experimental conditions using a t test. Differences in frequency of arrhythmias were tested with the Fischer exact test. A p value <0.05 was considered statistically significant.

**Results**

**Stability of Pacing Threshold and Ventricular Effective Refractory Period Over Time**

Because any change in pacing threshold could influence the apparent ERP, we examined the stability of pacing threshold over time in four hearts. Repeated determination (at 2-minute intervals, 10 minutes total) of diastolic threshold was performed at each of three added volumes (selected to approximate end-diastolic pressures of 0, 10, and 30 mm Hg, respectively). There was no consistent change in pacing threshold over time. This lack of effect was independent of added volume. Population standard deviation for repeated threshold determination averaged 7.1% (n = 10, range 0–14.5%). Similarly, ERP was stable over time at all three added volumes (standard deviation of repeated measurements = 3.1%, n = 10, range 0.7–8.4%).
Coronary flow rates (paced cycle length 250 msec, perfusion pressure 73 mm Hg) averaged 55 ± 2 ml/min (n = 35, range 36–72 ml/min). Coronary flow was consistent within individual hearts over the course of the experiment (average variation = 7%) and did not change consistently with increases in left ventricular volume.

**Effects of Left Ventricular Volume**

Increasing left ventricular volume significantly decreased left ventricular ERP but had no consistent effect on right ventricular ERP (Figure 3). The mean left ventricular ERP in seven hearts decreased from 117.7 ± 3.8 msec at zero pressure or starting volume to 91.4 ± 5.5 msec with an added volume of 1.5 ml (p<0.01) (Figure 4). Analysis of the relation of left ventricular ERP to left ventricular volume for the group data showed that the slopes were not zero (p<0.05). The mean slope of the regression lines was -18.6 ± 2.1 (Table 1). The slope of the relation of right ventricular ERP to left ventricular volume was not significantly different from zero. Although comprised of only 3–6 points, we tested individual experiments for statistical significance. In three hearts (A, C, and D), the slopes of the relation between left ventricular ERP and left ventricular volume were significantly different from zero. In none of the seven hearts was the relation between right ventricular ERP and left ventricular volume statistically different from zero. The left and right ventricular ERPs, while comparable at starting volume (117.7 ± 3.8 msec versus 117.6 ± 3.5 msec, p = NS), were significantly different at high volume (91.4 ± 5.5 msec versus 112 ± 5.6 msec, p < 0.05). An increase in left ventricular volume of 1.5 ml resulted in an increase in left ventricular end-diastolic pressure from 0 ± 1 to 35 ± 2 mm Hg.

In three hearts, left ventricular ERP was determined at starting volume, then 1.2 ml was added to the balloon, and the left ventricular ERP was repeatedly determined. The decrease in left ventricular ERP occurred within 1–2 minutes after an increase in left ventricular volume and did not change further for times up to 1 hour. Durations longer than this could not be consistently studied because of deterioration of the preparation. Repeat determination of left ventricular ERP at starting volume after prolonged dilatation was not significantly different from that determined before dilatation.

Left ventricular dilatation did not significantly affect epicardial pacing thresholds or interventricular conduction velocity of either the right or left ventricle (Figure 5). The standard error of the mean for epicardial pacing threshold was large because of a relatively large variation in pacing threshold between hearts. During each experiment, the average change in threshold for left ventricular pacing was a decrease of 1.2%. For right ventricular pacing, the average threshold change was an increase of 8%. Coronary sinus effluent PO2 averaged 85 mm Hg at a paced cycle length of 250 msec and a perfusion pressure of 73 mm Hg. No coronary sinus lactate was detected at even the highest volumes.
Effects of Paced Cycle Length and Perfusion Pressure

To assess the effects of myocardial oxygenation on the observed changes in left ventricular ERP, the sinus node was destroyed by crushing, and the paced cycle length was increased to 450 msec in seven hearts. Average coronary sinus effluent PO₂ increased to 160 mm Hg. There was a nonsignificant trend for lower coronary sinus effluent PO₂ at higher volumes consistent with the increase in myocardial oxygen consumption (Figure 6). Again, the left and right ventricular ERPs were comparable at starting volume (146.8 ± 2.7 msec versus 149.9 ± 3.4 msec, n = 7, p = NS) and significantly different (133.8 ± 4.1 msec versus 145.7 ± 3.8 msec, p < 0.05) with an added volume (1.26 ml) equivalent to a left ventricular end-diastolic pressure of 26 ± 5 mm Hg. There was a statistically significant relation between left, but not right, ventricular ERP and left ventricular volume. The slopes of the decrease in left ventricular ERP with increasing left ventricular volume were similar for paced cycle lengths of 250 and 450 msec.

In a third series of five experiments, the perfusion pressure was increased to 88 mm Hg. At a paced cycle length of 250 msec, coronary sinus effluent PO₂ averaged 159 mm Hg. Again, the slope of the relation between left ventricular ERP and left ventricular volume was negative and statistically different from zero (although the ERP at an added volume of 0.86 ml was not statistically different from starting volume, perhaps because of the small number of hearts studied). Pacing threshold and interventricular conduction velocity were not significantly changed by left ventricular dilatation at the longer paced cycle length or higher perfusion pressure. Thus, the decrease in left ventricular ERP with increasing volume appears to be relatively independent of drive cycle length and perfusion pressure.

Heterogeneity of Decreases in Left Ventricular Effective Refractory Period

Left ventricular ERP decreased nonuniformly at various left ventricular sites as left ventricular volume was increased (Table 2). The decrease in left ventricular ERP appeared to be greatest over the apical aspect of the left ventricle (16.7%) and least over the lateral-basilar aspect (9.1%). However, we did not statistically compare the percentage change for each region because of the limited numbers of hearts and because positioning of the electrodes was conceivably slightly different in each heart. We did statistically compare the difference between the ERPs at the two sites in each heart. The dispersion of recovery, estimated as the difference between the ERP at two left ventricular sites, did increase approximately twofold at added volumes of 0.8–1.0 ml (equivalent to an end-diastolic pressure of approximately 20 mm Hg) compared with starting

<table>
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<th>Hearts (n)</th>
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<tr>
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<td>RV ERP (msec)</td>
<td>LV ERP (msec)</td>
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<td>Starting volume</td>
<td>Added volume</td>
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<td>(0.4–0.6)</td>
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<tr>
<td>p*</td>
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Values shown are mean ± SEM.

*Probability that the slope is not significantly different from zero.

**LV, left ventricle; RV, right ventricle; NS, nonsignificant.**

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**TABLE 1. Comparison of Effective Refractory Period at Various Left Ventricular Volumes With Mean Slope and Y Intercepts for the Linear Regression Equation: Effective Refractory Period = b (Added Volume) + a, for Both Left and Right Ventricles Under Three Experimental Conditions**

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**FIGURE 5. Effect of left ventricular volume on epicardial pacing threshold (Panel A) and conduction time (Panel B) for the left and right ventricles at a pacing cycle length of 250 msec and a perfusion pressure of 73 mm Hg. The mean ± SEM is shown (n = 7). Volume is plotted as in Figure 4. The values at any given added volume were not significantly different from that at starting volume (SV), and the slopes were not significantly different from zero.**
volume. This increase was statistically significant ($p<0.05$) for both comparisons.

**Arrhythmias Observed**

No consistent changes in the intrinsic ventricular rate were seen with acute changes in left ventricular volume. Occasionally, increased ventricular ectopy was observed with a change in ventricular volume, especially when larger volumes were added, but no sustained ventricular arrhythmias occurred spontaneously. In contrast, ventricular fibrillation or tachycardia (Figure 7) was frequently induced during the determination of ventricular ERP with the introduction of a single ventricular extrastimulus during continuous ventricular pacing. This most commonly occurred with coupling intervals ($V_1-V_2$) close to the refractory period. Ventricular arrhythmias were induced more frequently when pacing the left ventricle at high (added volume ≥ 1.0 ml) volumes (35% of 37 trials) than when pacing the left ventricle at added volumes ≤ 0.5 ml (3% of 37 trials, $p<0.01$) or when pacing the right ventricle at high volumes (6% of 18 trials, $p<0.05$). Ventricular fibrillation was always persistent and required defibrillation. Ventricular tachycardia was frequently sustained but could be terminated by short bursts of epicardial overdrive pacing.

**Discussion**

We have demonstrated a progressive decrease in left ventricular ERP as a function of increasing left ventricular volume in an isolated rabbit heart model. These changes in refractoriness are unaccompanied by changes in conduction velocity or pacing threshold. Lerman et al recently observed that, in the cross-circulated, servo-controlled canine heart, increasing the left ventricular diastolic volume from 10 to 30 ml was associated with a 7% decrease in left ventricular ERP (from 205 to 191 msec). Conduction velocity was not measured, and arrhythmogenesis was not assessed. We have confirmed their observations in a denervated, isolated heart model in which we observed a progressive decrease in left ventricular ERP as ventricular volume was increased within the range of left ventricular end-diastolic pressures that may occur clinically with congestive heart failure. This effect appears to be due to an intrinsic myocardial phenomena and is not dependent on circulating catecholamines or central nervous system reflexes.
Lab has extensively studied the effects of mechanical stress on the electrophysiology of isolated muscle preparations. Changes in resting potential, action potential amplitude, and duration can be observed with changes in muscle length and tension. The nature of this contraction-excitation relation depends on the type and timing of the mechanical perturbation. Available evidence suggests that high muscle tension (e.g., isovolumetric contraction) is associated with a shortening of the action potential duration in isolated muscle preparations or in the intact frog heart. Our results demonstrate similar phenomena in the isolated, intact mammalian heart and suggest a potentially arrhythmogenic role for the changes that may occur.

At low ventricular volume, measured epicardial ERPs were similar for the right and left ventricles and within the left ventricle. This similarity between ERPs has also been observed at two endocardial sites in clinical studies. With increasing left ventricular volume, we observed an increase in the difference of refractoriness between the left and right ventricles and between different regions of the left ventricle. Since heterogeneity of myocardial recovery favors the development of reentry arrhythmias, cardiac enlargement is potentially arrhythmogenic. This proarrhythmic effect is not just theoretical. Arrhythmias were observed with a significantly higher frequency at high ventricular volumes. The observation that these arrhythmias were induced and terminated by epicardial pacing is considered evidence for a reentrant mechanism, although triggered automaticity cannot be excluded.

Increases in left ventricular volume resulted in decreases in refractoriness that were different over various aspects of the left ventricular epicardium. This heterogeneity of recovery may represent unequal changes in left ventricular wall stretch due to the exertion of unequal pressure by the intracavitary balloon. This view is supported by the observation that there appears to be a trend toward shortening of right ventricular refractoriness at high left ventricular volumes because right ventricular wall tension would probably increase as a reflection of marked enlargement in the intracavitary balloon. At first glance, this may seem artifactual. However, it is likely that many of the processes resulting in clinical congestive heart failure are also nonhomogeneous, and our model may be appropriate in this regard. Another explanation for the observed heterogeneity is that different regions of the left ventricle were stretched to different degrees by the same distending pressure. Sanders et al have demonstrated differences in the length-tension relation in isolated canine ventricular muscle when compared with false tendon. Alternatively, the physiological response of individual cells to the same degree of stretch may be variable and may result in heterogeneity of repolarization. Similar electrophysiological heterogeneity has been reported in an animal model of heart failure and when isolated ventricular muscle is stretched beyond its optimal length.

Our observed results do not appear to be related to passive mechanical factors (e.g., electrode-myocardial contact) since the measured thresholds were unaffected by modification of ventricular volume. Epicardial pacing can produce asynchronous ventricular contraction. It might be argued that the observed changes in repolarization with ventricular dilatation are secondary to increased asynchrony caused by enlargement of the epicardial pacing.
ventricle. This seems unlikely since any significant increase in asynchrony of contraction would have been accompanied by an increase in conduction time, which was not observed. Moreover, refractoriness as we assessed it is a property of the tissue under the electrode. Subsequent spread of contraction should have little influence on local refractoriness.

Global ischemia is probably not the cause of the changes observed. We observed no statistically significant decrease in coronary sinus effluent Po2 with increasing ventricular volume, and coronary sinus lactate was undetectable. Additionally, comparable decreases in left ventricular ERP were observed at higher perfusion pressure and longer paced cycle lengths even though myocardial oxygen consumption is decreased at the longer drive cycle length and myocardial oxygen supply is increased at the higher perfusion pressure. Opie has summarized the evidence that myocardial oxygenation is adequate in this model at levels of coronary sinus effluent Po2 above 150 mm Hg. Finally, ischemia has been shown, in other preparations, to increase refractoriness and slow conduction velocity and which we did not observe. Thus, it is reasonable to conclude that these changes were not a consequence of compromised global myocardial oxygenation in this model. Regional ischemia due to the mechanical effects of the intracavitary balloon also appears to be an unlikely mechanism because of the absence of changes in conduction velocity.

Although our model explores the electrophysiological effects of acute changes in ventricular size, it may have clinical relevance. First, several clinical situations can produce acute changes in global or regional wall tension. For example, ischemia or mitral valve prolapse can produce acute dyskinesia and increase localized myocardial tension. Acute ventricular septal rupture or papillary muscle dysfunction may similarly cause an acute change in chamber size. Second, the changes we have described in our acute model may be operative for chronic ventricular dilatation. We observed that the changes in refractoriness were stable for periods at least up to 1 hour. In our model, epicardial pacing does not activate the ventricle over the normal conduction pathway. However, epicardial pacing probably reflects accurately the clinical situation in which a premature ventricular beat may induce a ventricular arrhythmia.

In humans, the role that increased volume per se may play in the development of ventricular arrhythmias has not been adequately evaluated. Spontaneous and inducible ventricular arrhythmias have been correlated with ventricular function in some but not all reports. Clinical studies assessing the effect of decreased ventricular size with, for example, angiotension converting enzyme inhibition have on occasion shown an antiarrhythmic effect, but interpretation of these observations is complicated by the multiple biological effects of the agents used. Nevertheless, our data suggest the possibility that changes in ventricular refractoriness associated with ventricular dilatation can produce important clinical arrhythmogenic effects.

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