Electrophysiologic effects of papillary muscle traction in the intact heart

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ABSTRACT  In this study we used transmural multipolar electrodes, sonomicrometers implanted within the left ventricular wall, and cardiac electrical stimulation techniques to examine the effect of transient mechanical posterior papillary muscle traction on local myocardial electrophysiologic characteristics. Nine open-chest dogs were atrially paced (cycle length 400 msec) followed by insertion of timed premature extrastimuli at left ventricular epicardial pacing sites either in the vicinity of (traction zone) or remote from (nontraction zone) the site of papillary muscle traction. Electrophysiologic recordings were made before and during periods of intermittent papillary muscle traction of predetermined timing, application rate (25 cm/sec), and duration (170 msec). Papillary muscle traction was applied in late diastole just before the last beat of each atrial drive train. In seven of nine dogs application of transient papillary muscle traction resulted in significantly earlier local ventricular activation (mean activation advancement 30 ± 13 msec), altered QRS morphology of the last conducted atrial drive-train beat, and relative prolongation of ventricular functional refractory period in the traction zone. Conversely, in nontraction zones in these seven dogs, early activation did not occur and refractoriness remained unchanged as tested by a locally placed extrastimulus. In two of nine dogs traction failed to induce early activation and changes in refractoriness did not occur. Alterations in regional myocardial blood flow (assessed by radioactive microsphere technique) did not appear responsible for the observed changes, since there was no demonstrable traction-induced difference in regional blood flow between the traction and nontraction zones. Thus, in normal myocardium in situ, regional abnormal wall motion may be associated with alterations of local ventricular activation and refractoriness, factors that in the diseased heart may lead to increased susceptibility to arrhythmias.

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ALTHOUGH an association between abnormal regional myocardial mechanical function and development of potentially lethal ventricular arrhythmias has become well recognized clinically,1-5 the basis of this relationship in the intact heart is not understood. In superfused cardiac tissues, experimental studies have demonstrated that myocardial length/tension relationships influence transmembrane action potential characteristics.6-13 Additionally, in isolated perfused whole heart preparations,12,13 alterations in shortening or tension development have been shown to alter transmembrane action potential repolarization. Similarly, diastolic transmembrane potential depolarization, initiating a new action potential, has been observed during periods of transient stretch in isolated cardiac tissue.12,13 However, it is not known whether disturbances of myocardial contraction or abnormal stretch on portions of the ventricular wall alter myocardial electrophysiologic characteristics, and thereby potentially increase arrhythmia susceptibility in the diseased heart in situ.

The purpose of this study was to determine, in vivo, whether transient mechanically applied traction to the posterior papillary muscle is accompanied by potentially arrhythmogenic alterations of local ventricular electrophysiologic properties. To this end, we measured local endocardial and epicardial electrophysiologic characteristics in the vicinity of the posterior...
papillary muscle and at distant control sites in open-chest anesthetized dogs before and during periods of intermittent posterior papillary muscle traction.

**Methods**

**Animal preparation.** Nine conditioned adult mongrel dogs (20 to 30 kg) of either sex were anesthetized with α-chloralose (100 mg/kg iv) and ventilated with a Harvard volume respirator. A left thoracotomy was performed, and the heart was suspended in a pericardial cradle.

**Instrumentation**

Pacing and recording electrodes. Figure 1 illustrates the instrumentation used. Bipolar pacing electrodes (1 mm interelectrode distance) were sutured to the right atrium for atrial pacing and to the left ventricular epicardium near the base of the anterior (control area) and posterior (traction area) papillary muscles for delivery of ventricular extrastimuli. Bipolar stainless steel epicardial recording electrodes, 1 mm electrode tip separation, were sutured to reference sites on the right atrium and right ventricular free wall. Additionally, in four of nine dogs, a specially designed plaque electrode was positioned near the non-coronary cusp of the aorta to record His bundle electrograms.14

In all animals multipolar needle electrodes were positioned transmurally at the bases of both the anterior and posterior papillary muscles. Care was taken in seating the transmural electrodes to minimize tissue injury. Each transmural needle comprised 10 individual electrodes from which two bipolar pairs, each with 1 mm electrode spacing, were selected for recording electrograms closest to the subepicardium and subendocardium.

Bipolar recording techniques were employed because the principal measurement objective was local electromgram timing, making desirable to minimize far-field signals. Local activation was defined to have occurred at the point in time when the first rapid electromgram deflection crossed the isoelectric line.

**Dimension transducers, aortic pressure recordings, and myocardial blood flow.** Paired pulse-transit ultrasonic dimension transducers (1.5 mm diameter) were implanted 1 cm apart within the left ventricular wall in close proximity to the transmural needle electrodes to monitor local wall motion. Transducers were oriented parallel to epicardial myocardial fibers along the minor axis of the heart and consequently were approximately perpendicular to the base of the papillary muscle. Transducers were fixed in position within the ventricular wall so as to move with the myocardium during cardiac contraction, thus measuring actual lengthening and shortening of a local myocardial segment. Previous studies15, 16 have indicated that the dimension transducers can be placed with minimal tissue trauma, providing a precise method for measuring regional myocardial mechanical performance. Although the orientation of the transducers did not permit precise measurement of the magnitude of papillary muscle distortion caused by applied traction, documentation of reproducibility of traction-induced distortion throughout each experiment was possible.

Ascending aortic blood pressure was measured with a Statham P23db pressure transducer directly coupled to an intravascular catheter.

Measurement of myocardial blood flow was facilitated by placement of a small catheter in the left atrium for injection of radioactive microspheres. Aortic blood sampling during injection of microspheres was done with the aortic pressure catheter.

**Papillary muscle traction.** Posterior papillary muscle traction of reproducible timing, rate of onset, and duration was achieved by use of a custom-designed electrogram-triggered stepping motor device. Traction was applied as a ramp function with onset and offset rates of 25 cm/sec and held for 10 msec. Total duration of traction was 170 msec. Traction was applied to the posterior papillary muscle with a stainless-steel snare introduced through a purse-string suture into the left atrial appendage and carefully manipulated across the mitral valve. Sufficient tension was applied to produce visually detectable epicardial dimpling in the area at the base of the posterior papillary muscle. Placement of the snare was further confirmed by recordings from the miniature ultrasonic dimension transducers placed at the base of the posterior papillary muscle. After completion of the study protocol, the position of the snare was confirmed at necropsy, and the papillary muscle, chordae tendineae, and surrounding structures were examined. In no instance was there macroscopically evident myocardial trauma or disruption of chordal structures.

**Study protocol**

**Electrophysiologic studies.** In all studies, ventricular refractory periods and local conduction latency (see Definitions) were determined by delivery of timed ventricular extrastimuli after

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**FIGURE 1.** Schematic representation of instrumentation used to produce posterior papillary muscle traction and determine its electrophysiologic (pacing and recording electrodes) and mechanical (ultrasonic crystals) sequelae. LA = left atrium; LV = left ventricle.
FIGURE 2. Schematic representation of the study protocol used to assess ventricular refractoriness. A. Under control conditions from left to right are the seventh (S1-7) and eighth (S1-8) beats of an 8 beat atrial drive train. After the eighth beat an extrastimulus (S2) is delivered locally either in the ventricular control or traction zones to assess refractoriness. The shortest V1-8-V2 attainable is the local FRP. B. With papillary muscle traction (170 msec traction bar) applied 30 msec after S1-8, traction-induced advancement (x) of V1-8 occurs. Again, S2 is applied in either the control or traction zones to assure local refractoriness, with the shortest V1-8-V2 attainable being the FRP. S = stimulus artifact; V = ventricular activation.

the last beat of an 8 beat atrial drive train (figure 2). Fixed cycle length (400 msec) atrial pacing was used in all dogs to permit both interanimal and intra-animal data comparison.

Baseline (control) ventricular refractory period measurements (see Definitions) were obtained by delivery of single epicardial extrastimuli in the vicinity of either the anterior papillary muscle (nontraction zone) or the posterior papillary muscle (traction zone) during alternate test sequences. The extrastimulus coupling interval (i.e., the interval between the last local ventricular electrogram and the ventricular extrastimulus) was initially reduced by 10 msec decrements and then by 5 msec decrements as ventricular refractoriness was approached.

PAPILLARY MUSCLE TRACTION STUDIES. After baseline electrophysiologic measurements were obtained, posterior papillary muscle traction was applied during late diastole of the seventh beat of an 8 beat atrial drive train (see Instrumentation above; figure 2). Specifically, traction was applied beginning 30 msec after the atrial stimulus artifact of the eighth beat for a duration of 170 msec. Traction resulted in a discrete localized wall motion disturbance detected by the regional ultrasonic dimension transducers (figure 3). Since papillary muscle traction was applied only in late diastole of the seventh atrially paced beat, potential adverse hemodynamic alterations (e.g., mitral regurgitation) caused by the papillary muscle traction was minimized, yet the effect of wall motion disturbances on local ventricular refractoriness of the eighth (last) atrial drive-train beat could be assessed by ventricular extrastimulus testing.

PROMISE ELECTRICAL ACTIVATION STUDIES. In a subgroup of dogs we attempted to ascertain whether local electrophysiology properties were altered in a different manner by premature electrically induced compared with premature mechanically induced activation. In four dogs, early local ventricular activation of the eighth drive-train beat, similar to that produced by papillary muscle traction, was achieved by insertion of an epicardial extrastimulus of fixed coupling interval applied near the posteri- or papillary muscle. Refractoriness of extrastimulus-induced early local activation was determined with a second epicardial extrastimulus (coupling interval reduced initially by 10 msec decrements, then by 5 msec decrements as ventricular refractoriness was approached).

Regional myocardial blood flow. In six dogs, an estimate of the effect of papillary muscle traction on regional myocardial blood flow was assessed by the radioactive microsphere technique.17 Radiolabeled (Co, Nb Cr, I, or Sn) microspheres (15 μm in diameter, approximately 2 × 10⁶ microspheres/injection) were injected into the left atrium with simultaneous reference blood sampling performed from the ascending aorta. Differently labeled microspheres were injected either under baseline conditions or during repetitive late diastolic papillary muscle traction. Myocardial blood flow was estimated in subepicardial, midwall, and subendocardial zones for both the posterior (traction zone) and anterior (control zone) papillary muscles and their surrounding left ventricular wall. Additionally, blood flow in sections of the right and left kidney was measured.

During measurement of regional myocardial blood flow, papillary muscle traction was applied during late diastole of every paced beat, since myocardial blood flow cannot be determined on a beat-to-beat basis with this technique. Consequently, this subsection of the study provided a “worst” case analysis of the influence of papillary muscle traction on blood flow.

Definitions. (1) Local ventricular functional refractory period (FRP) was measured at a right ventricular epicardial site, and at left ventricular subepicardial and subendocardial sites in both the nontraction (control) and traction zones. FRP was defined as the shortest coupling interval between local ventricular activation of the last (eighth) drive-train beat (either atrial propagated-control, traction altered, or premature electrically activated; figure 2) and the local ventricular electrogram of the extrastimulus-induced ventricular premature beat. FRP measurements were used because the eighth drive-train beat was variably initiated by either atrial impulse propagation or local premature mechanical or electrical stimulation. Measurements of ventricu-
lar effective refractory period were not used because a local ventricular extrastimulus was generally not present on the last drive-train beat.

(2) Local latency of premature beats was determined during extrastimulus testing and was defined as the interval between the extrastimulus artifact and the local bipolar electrogram measured on the subepicardial needle electrode pair. To evaluate the effects of papillary muscle traction on conduction of premature beats, both maximum latency achieved and latency at comparable local coupling intervals were assessed.

Data acquisition. Epicardial and transmural electrogram recordings and tracings from surface electrocardiogram lead II were monitored continuously with a multichannel recorder; high-speed recordings (200 mm/sec) were obtained on heat-sensitive paper as needed (Hewlett Packard 7404A). Bandpass filter settings for bipolar electrogram signals were 5 to 1000 Hz (Hewlett Packard Bioelectric 8811A amplifiers). Electrophysiologic measurements (see Definitions) were obtained by manually digitizing the recorded electrograms (Houston HiPad Digitizer) with an interactive computer program.

Right atrial pacing and ventricular extrastimulus testing were performed with a custom-designed stimulator with two independent optically isolated outputs. The electrical stimulus was a constant-current monophasic square wave with a pulse width of 2 msec and an amplitude of twice late diastolic pacing threshold.

The paired miniature ultrasonic dimension transducers used piezoelectric crystals with a response frequency of 5 MHz, thereby resulting in a minimum dimension resolution of 0.07 mm. The sampling rate of the sonomicrometer system was 1 kHz, and the analog output was electronically filtered at 100 Hz, providing sufficient frequency response for cardiac dimension signals. Analog measurements of ventricular wall segment dimension, aortic pressure, and surface electrocardiogram lead II were visually displayed on a monitoring screen. Wall segment dynamic dimensions and aortic pressure recordings were recorded on magnetic tape (Hewlett Packard 3968A) for later analysis.

Segments of myocardium and kidney in which blood flow determinations were made were fixed with formalin, sectioned, and weighed. Radioactivity levels in the tissue sections along with reference aortic blood samples were measured with a Packard Auto Gamma Scintillation Spectrometer.

Statistical analysis. The statistical significance of traction-induced changes in electrophysiologic measurements was analyzed by Student's t test for paired observations. All data are presented as mean ± SD, and statistical significance was set at p values less than or equal to .05.

Results

Effect of papillary muscle traction on QRS morphology and ventricular activation. In seven of nine dogs, the QRS morphology of the eighth conducted atrial drive-train beat was altered by papillary muscle traction applied during late diastole of the preceding beat (figure 4). In each instance, the change in QRS morphology of the electrocardiogram was associated with alteration of local intraventricular activation caused by early activation in the vicinity of the traction zone (posterior papillary muscle) and is exemplified by findings from dog 7 (figure 4).

Advancement of local electrical activity in association with papillary muscle traction was further characterized in those dogs in whom recordings were obtained from electrodes implanted in the vicinity of the His bundle. In each case, atrioventricular conduction time was not measurably affected by traction, whereas the interval between activation of the His bundle and local ventricular activation was shortened. The latter observation was most compatible with premature ventricular excitation as a result of the imposed abnormal myocardial stretch. In dogs exhibiting traction-induced advancement of local ventricular activation in the vicinity of the posterior papillary muscle, excitation was advanced by 18 to 54 msec (mean 30 ± 13 msec; p < .01 vs baseline).

In two dogs the QRS morphology was unaltered by posterior papillary muscle traction and no alteration of local intraventricular electrogram sequence was detected.

Effect of papillary muscle traction on local FRP. The effect of papillary muscle traction on local ventricular

| TABLE 1 |
| Local functional refractory periods (cycle length 400 msec) (mean ± SD) |
| Extrastimulus in traction zone | Control zone | RV |
| Traction zone | Control zone | RV |
| Control | | | |
| Epicardial | 219 ± 27 | 270 ± 25 | 286 ± 26 |
| Endocardial | 235 ± 34 | 272 ± 26 | 286 ± 39 |
| Traction | | | |
| Epicardial | 219 ± 30 | 251 ± 35 | 257 ± 37 |
| Endocardial | 232 ± 28 | 252 ± 35 | 293 ± 39 |
| p value | | | |
| Epicardial | NS | .015 | NS (.002) |
| Endocardial | NS | .01 | .045 |

RV = right ventricle.
FIGURE 4. Analog tracings of electrophysiologic recordings obtained during refractory period measurements with posterior papillary muscle traction applied just before the eighth beat of an 8 beat atrial drive train followed by premature stimulation of the left ventricle at the control site. Beginning from left-to-right, the beats represented include the seventh (S1-E7) and eighth (S1-E8) atrially paced drive-train beats followed by the premature ventricular stimulated beat (V2) and first recovery beat. Note the traction-induced distortion of the surface electrocardiogram QRS of the eighth drive-train beat, and the change in activation sequence of the ventricles as recorded from the intracardiac transmural and epicardial recording electrodes. Earliest ventricular activation of the eighth drive-train beat occurs in the endocardium at the site of traction with activation occurring nearly simultaneously with the His bundle electrogram. The time from the atrially placed stimulus artifact to each local ventricular electrogram activation is indicated in milliseconds. Recordings from top to bottom are: surface electrocardiogram, lead II; subepicardial (Epi) and subendocardial (Endo) electrogram recordings from transmural electrodes in the traction and control zones, respectively; right ventricular (RV) epicardial electrogram; and His bundle electrode recording (HBE). S = stimulus artifact (S1 = atrial pace; S2 = ventricular premature); A = atrial electrogram; V = ventricular electrogram; H = His bundle electrogram.

FRP in the seven dogs exhibiting traction-induced changes in QRS morphology is summarized in table 1 and figures 5 and 6. Findings are presented for studies in which ventricular extrastimuli were delivered within the left ventricular traction zone (i.e., in the vicinity of the posterior papillary muscle), as well as for those in which extrastimuli were delivered at the left ventricular control (nontraction) zone.

Extrastimulus in the traction zone. When extrastimuli were delivered within the traction zone, initiation of traction did not alter local FRP in the traction zone from baseline values (table 1, figures 5 and 6). On the other hand, local FRPs of the control (nontraction) left ventricular and right ventricular sites were significantly shortened (p < .01).

Extrastimulus in the nontraction zone. When extrastimuli were delivered within the nontraction left ventricular site (i.e., in the vicinity of the anterior papillary muscle), FRP of the traction zone tended to prolong when traction was applied (endocardial p = .045, epicardial p = .062, NS). FRPs of left and right ventricular control zones were unchanged by introduction of the transient mechanical disturbance (table 1, figures 5 and 6).

In the two dogs in which traction failed to induce detectable changes in QRS morphology or intraven-
FIGURE 5. Local subendocardial FRP values at (ordinate, msec) cycle length 400 msec under both baseline conditions and with papillary muscle traction applied as indicated at the bottom of the panels. A, Measurements obtained with the ventricular extrastimulus placed in the traction zone. B, Measurements obtained with the extrastimulus in the nontraction (control) zone. Means and SDs are depicted by the vertical bars.

FIGURE 6. Local subepicardial FRP measurements (ordinate, msec) in the left ventricular control and traction zones and the right ventricular epicardium. Data are presented in the same fashion as the figure 4, with the addition of the right ventricular (RV) recording site.
tricular activation sequence, findings differed from those obtained in the seven dogs reported above. In one case, traction induced an approximate 10 msec prolongation of FRP independent of extrastimulus site; in the remaining dog, FRPs at all sites were unaffected by application of traction.

**Effect of premature early local ventricular activation by extrastimulus technique on local FRP.** In four dogs the QRS morphology of the eighth atrial drive-train beat was altered by insertion of a local epicardial extrastimulus so that local ventricular activation was advanced by 25 ± 9 msec in the traction zone simulating that produced by papillary muscle traction. Figure 7 illustrates a reduction in local FRP at all sites tested (p < .005).

**Effect of papillary muscle traction on latency.** The application of transient papillary muscle traction did not alter latency of premature extrastimuli of equivalent coupling intervals (V1-S2) delivered in the traction zone (figure 8). However, the maximum latency achievable before failure of ventricular capture by the extrastimulus was reduced in the presence of traction (control 53 ± 5 msec, traction 31 ± 7 msec; p < .02).

**Effect of premature early local ventricular activation by extrastimulus technique on latency.** In the subset of dogs in which premature local ventricular activation was achieved by electrical stimulation, the latency of subsequent premature extrastimuli applied to the test zone was unaltered if comparable coupling intervals (V1-S2) are examined (figure 9). However, maximum latency achievable before failure of ventricular capture increased in all four dogs by premature electrical activa-

**FIGURE 7.** Shortening of local subendocardial and subepicardial FRP values (ordinate, msec) observed in four dogs under baseline conditions and after premature electrical activation (PEA) of the ventricle by the extrastimulus technique applied in the traction zone as indicated at the bottom of the panel (see Methods). Data are presented as in figure 5.

**FIGURE 8.** Latency measurements (local ventricular stimulus artifact [S2] to local ventricular activation [V2], msec) from the traction zone are plotted on the ordinate with local coupling interval of ventricular extrastimulus (local V1 from eighth atrially paced beat to S2 from local premature ventricular stimulation) on the abscissa for two experiments. In each panel, at comparable V1-S2 intervals, latency is unchanged (control vs traction), but during periods of traction, latency at the shortest coupling intervals before loss of ventricular capture fails to lengthen when compared with the control experiments.

**FIGURE 9.** Latency measurements (local ventricular stimulus artifact [S2] to local ventricular activation [V2]) at the traction zone are plotted as in figure 7 for two experiments. In each panel, at comparable V1-S2 intervals, latency is unchanged (control vs premature electrical activation), but during premature electrical activation latency at the shortest coupling intervals lengthens before failure of ventricular capture as compared with the control experiments.
Myocardial blood flow (mean ± SD; ml/min/g)

<table>
<thead>
<tr>
<th>Traction zone</th>
<th>Control zone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td></td>
</tr>
<tr>
<td>Epicardial</td>
<td>1.16±0.50</td>
</tr>
<tr>
<td>Midwall</td>
<td>1.22±0.40</td>
</tr>
<tr>
<td>Endocardial</td>
<td>1.27±0.55</td>
</tr>
<tr>
<td>Transmural</td>
<td>1.21±0.46</td>
</tr>
<tr>
<td>Renal</td>
<td>6.12±1.09</td>
</tr>
<tr>
<td>Traction</td>
<td></td>
</tr>
<tr>
<td>Epicardial</td>
<td>0.59±0.34</td>
</tr>
<tr>
<td>Midwall</td>
<td>0.58±0.30</td>
</tr>
<tr>
<td>Endocardial</td>
<td>0.60±0.31</td>
</tr>
<tr>
<td>Transmural</td>
<td>0.59±0.37</td>
</tr>
<tr>
<td>Renal</td>
<td>2.69±1.45</td>
</tr>
</tbody>
</table>

...tion of the ventricle (control 55 ± 15 msec; premature electrical activation 66 ± 21 msec; p = .07, NS).

**Myocardial blood flow.** By the radioactive technique in six dogs, myocardial blood flow was estimated in the subepicardium, midwall, and subendocardium for both the traction zone (posterior papillary muscle) and the control zone (anterior papillary muscle) and their surrounding left ventricular wall. With the “worst” case situation with traction applied to every cardiac cycle, there was a decrease in blood flow to both the traction and control zones with a concomitant fall in renal blood flow as shown in table 2. However, there was no evidence of a local reduction of myocardial blood flow in the traction zone caused by traction.

**Discussion**

Findings in this study indicate that mechanically induced late diastolic posterior papillary muscle traction alters myocardial electrophysiologic characteristics in the intact canine heart. Three principal electrophysiologic changes were observed. First, at the site of traction, local premature ventricular activation was induced by mechanical distortion. Second, despite shortening of the cardiac cycle length by mechanically induced premature ventricular activation, refractoriness in the traction zone remained unchanged or prolonged. Third, although at comparable V1-S2 coupling intervals the latency of locally placed premature stimuli was unaltered, maximum achievable latency was reduced by traction. The last two findings were in contradistinction to the shortening of refractoriness and prolongation of latency seen with premature ventricular activation by the extrastimulus technique.

**Relationship of mechanical distortion to electrophysiologic changes.** Local premature ventricular activation induced by transient papillary muscle traction coincided with maximum mechanical distortion (documented by regional ultrasonic crystals, figure 3) and occurred before the beginning of muscle release. Presumably, mechanically induced premature ventricular activation in this preparation was the result of stretch or distortion of either the papillary muscle itself or local attached Purkinje fibers. Studies in isolated cardiac tissues have shown myocardial stretch to be associated with reversible depolarization of diastolic transmembrane potential. In some instances transmembrane potential depolarization caused by myocardial stretch has been associated with both transient afterpotentials and propagated action potentials.

Premature ventricular activation at the site of traction resulted in earlier local electrical activity at this site compared with both left ventricular control and right ventricular sites. Hence, in the traction zone, the interval between the last local ventricular activation propagated from the seventh atrial drive-train beat and initiation of the subsequent ventricular activation due to mechanical distortion was less than the basic drive-train cycle length. Under these conditions ventricular myocardial refractoriness would be expected to be reduced, an expectation confirmed by premature electrical activation of the traction zone in four dogs by placement of a timed extrastimulus in the traction zone simulating the premature local ventricular activation produced by mechanical traction. Local FRP after premature electrical activation of the traction zone was reduced by 29 ± 8 msec. Conversely, traction zone FRP after mechanically induced local premature ventricular activation was either unchanged or prolonged. Thus mechanically induced papillary muscle traction resulted in a relative prolongation of local ventricular refractoriness.

Prolongation of refractoriness by papillary muscle traction parallels previous findings from this laboratory, in which myocardial mechanical dysfunction associated with transient aortic occlusion resulted in prolongation of ventricular refractoriness. The basis for prolongation of refractoriness is uncertain but may be related to initiation of afterdepolarizations after release of “stretched” myocardial tissue, a finding that has been observed in isolated cat papillary muscle.

Latency of premature extrastimuli at comparable coupling intervals was unaffected by mechanically or electrically induced early ventricular activation. On the other hand, mechanically induced prolongation of local refractoriness resulted in diminished latency of premature extrastimuli placed in the traction zone. In contrast, premature electrical activation shortened local refractoriness and prolonged maximum latency.

Myocardial blood flow was reduced globally when
traction was placed during every cardiac cycle. This global reduction in blood flow, as further evidenced by the fall in renal blood flow, was probably related to mitral regurgitation caused by repetitive traction. However, there was no significant traction-induced difference between blood flow in the left ventricular traction and control zones. Furthermore, the intermittent papillary muscle distortion employed during the portion of the study in which electrophysiologic measurements were obtained likely resulted in a smaller hemodynamic disturbance. Thus it is reasonable to conclude that the local electrophysiologic changes observed in the area of traction were probably not a consequence of compromised blood supply.

Conclusions. Given the transient nature of the mechanical stimulus used in these studies, and the rapidity of the electrical changes noted, either a direct mechanical effect (e.g., mechanically induced membrane changes, changes in local geometry) or a local reflex neural mechanism may play a role in effecting electrophysiologic changes. Nonetheless, findings in this study support the concept of “contraction-excitation feedback” in the intact normal canine ventricle in situ. Potentially, abnormal ventricular wall motion in disease states may both induce premature ventricular ectopic activity and exacerbate differences in myocardial excitability leading to increased risk for the development of cardiac arrhythmias.

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