Myocardial Relaxation. V. Postextrasystolic Contraction-Relaxation in the Intact Dog Heart

A. S. Blaustein, M.D., W. H. Gaasch, M.D., D. Adam, Ph.D.,
and H. J. Levine, M.D.

SUMMARY Postextrasystolic (PES) relaxation of the left ventricle was studied in 24 anesthetized dogs using the time constant (τ) of the left ventricular isovolumic pressure decline as an index of global relaxation velocity. Using programmed atrial stimulation and a control RR interval of 500 msec, the “coupling interval/compensatory pause” was varied from 400/600 msec to 250/750 msec, and left ventricular pressure-segment length data from control beats were compared with data from PES beats. Contractile state and fractional shortening increased in the PES beat, but the relaxation time constant remained unchanged (control, 35 ± 3 msec; PES at 250/750 msec, 36 ± 3 msec) (p = NS). Pretreatment with propranolol did not qualitatively influence these results. Isoproterenol and calcium were given in doses sufficient to increase the time derivative of isovolumic pressure (maximal positive dP/dt) by an amount equal to that obtained with PES potentiation (approximately 50%); isoproterenol produced a substantial decrease in the relaxation time constant (38 ± 4 to 30 ± 6 msec, p < 0.01), whereas calcium administration produced only a small decrease in the time constant (30 ± 5 to 27 ± 5 msec, p < 0.05).

Thus, in the intact dog heart, some positive inotropic interventions augment contraction and speed relaxation, but PES potentiation of contraction is not associated with a change in relaxation velocity.

POSTEXTRASYSTOLIC potentiation (PESP) of the left ventricular (LV) contractile state contributes to the increase in systolic performance that is observed in the beat after an extrasystole.5,6 In the intact circulation, the magnitude of the LV response also depends on a complex interaction between variations in filling after the extrasystole and postextrasystolic changes in aortic input impedance. Although it is known that atrial premature complexes induce PESP,4 it is not known whether atrial and ventricular extrasystoles produce the same postextrasystolic response.6 Despite incomplete understanding of the mechanisms responsible for augmented postextrasystolic contraction, PESP has been studied and used in a broad variety of experimental and clinical situations.6 In contrast, postextrasystolic relaxation has not been extensively studied.

In the intact dog heart, relaxation velocity has been shown to be increased during interventions that augment the extent and velocity of fiber shortening.6 Both pharmacologic and mechanical interventions that influence LV end-systolic load influence relaxation velocity.7,8 Because increased extent and velocity of fiber shortening and decreased end-systolic fiber length result from PESP, it might be expected that relaxation velocity would be increased in the postextrasystolic beat. However, data from isolated muscle experiments indicate that myocardial contraction and relaxation are not tightly coupled, and under some circumstances it appears that paired stimulation and other inotropic interventions (such as isoproterenol and calcium) can produce disparate effects on relaxation.9 A similar comparison of these three interventions in the intact heart has not been reported. Accordingly, we analyzed the effect of PESP on relaxation velocity in the intact heart and we examined the relative effects of PESP, isoproterenol, and calcium infusion on ventricular relaxation.

Methods

Twenty-four mongrel dogs were premedicated with morphine sulfate, 1 mg/kg, and anesthetized with sodium pentobarbital, 15 mg/kg. After a midsternotomy, the heart was suspended in a pericardial cradle, the sinus node crushed, and the right atrium paced at 120 beats/min. A high-fidelity transducer-tip catheter (Millar Mikrotip) was inserted into the apex of the left ventricle for the measurement of LV pressure. Aortic pressure was measured in the descending thoracic aorta by advancing a fluid-filled catheter retrograde from the femoral artery (Statham P23Db transducer). LV pressure at the time of aortic valve opening (aortic diastolic pressure [A,D]) was determined by matching the beginning of the upstroke of the aortic pressure tracing with the corresponding point on the LV pressure tracing. Myocardial segment length was measured with ultrasonic crystals implanted approximately 1 cm apart through epicardial stab wounds. The crystals were placed in the inner third of the LV wall perpendicular to the long axis of the ventricle. Segment lengths were measured at end-diastole and end-systole, and fractional shortening was calculated. An example of these records is shown in figure 1.

The time constant of left ventricular isovolumic pressure decline (τ) was determined from LV pressure
signals, which were recorded on magnetic tape (Hewlett-Packard, model 3513-4) and replayed at one-fourth speed. Using a sonic digitizer (Graf pen, Science Accessories Corporation) interfaced with a Digital PDP/8e computer, LV pressure was measured at 5-msec intervals throughout the period of isovolumic pressure decline; in the present experiments we defined this period as beginning 10 msec after maximal negative dP/dt (dP/dt min) and ending at a pressure equal to 15 mm Hg above the LV end-diastolic pressure of the ensuing diastole (which ranged from 2–5 mm Hg). We used 15 mm Hg above end-diastolic pressure to ensure that pressure measurements were not made after mitral valve opening and especially to avoid measurements just before mitral opening when pressure appears to be falling at a slightly faster rate than that predicted by a perfect exponential. These coordinates of pressure (P) and time (t) were fitted (least squares) by the equation \[ \ln P = \ln P_0 - \frac{t}{\tau}, \] and \( \tau \) was determined.\(^{6-9,11}\) The linear correlation coefficient between \( \ln P \) and \( t \) ranged from 0.96–0.99.

Experimental Protocol

The heart rate was maintained constant at 120 beats/min with an atrial pacemaker and a series of premature contractions was generated (Grass stimulator) with stimuli 2 msec in duration and an amplitude that was twice threshold. The control RR interval (RR\(_c\)) was 500 msec, and the coupling interval (RR\(_1\)) between the control beat and the premature contraction was varied from 400–250 msec in 50-msec decrements; the compensatory pause (RR\(_2\)) was varied from 600–750 msec so that the RR\(_1\) interval plus the RR\(_2\) interval was twice the RR\(_c\) interval. Both atrial and ventricular extrasystoles were studied, and only postextrasystolic beats with a normal QRS morphology were analyzed. In the case of atrial premature contractions, a slight increase in the PR interval was occasionally observed, but in no instance did this increase exceed 20 msec.

In seven dogs, we compared LV postextrasystolic pressure transients after right atrial (APC) and right ventricular (VPC) extrasystoles. The VPC was produced by stimulation of the right ventricular free wall epicardium just below the outflow tract. Because there was no difference between the APC and the VPC in the LV postextrasystolic pressure response, subsequent studies involved only APCs. In 10 dogs, postextrasystolic pressure and segment length data were analyzed before and after the administration of propranolol (0.2 mg/kg, i.v.)

After examining the effect of PESP, we studied the effects of two other positive inotropic interventions on LV relaxation. In six dogs, isoproterenol (1 mg/250 ml, i.v.) was administered at a rate sufficient to produce a 50% increase in maximal positive dP/dt (dP/dt max), and data were collected 2–3 minutes after a steady state was achieved. In six additional dogs, calcium chloride (1.5 g/100 ml) was administered by a continuous i.v. infusion at a rate sufficient to produce a 50% increase in dP/dt max. As above, these experiments were performed at a constant heart rate (atrial pacing, 120 beats/min). The effects of isoproterenol and calcium on LV relaxation were then contrasted with the effects of PESP (the RR\(_1\)/RR\(_2\) associated with a 50% increase in dP/dt max was selected).

In the APC vs VPC studies and in the baseline vs propranolol studies, the data over the entire range of RR intervals were analyzed using a three-way analysis of variance and a Duncan's test to locate the variance. In the isoproterenol and calcium studies, the intervention data were compared with control data by means of a paired \( t \) test; the magnitude of the difference between control and intervention data was compared by means of an unpaired \( t \) test. The data are reported as the mean ± SD.

Results

The initial experiments were designed to compare the effects of APC and VPC stimuli on LV pressure transients in the postextrasystolic beat. The results are summarized in table 1. Neither the origin of the extrasystolic (APC vs VPC) nor variations in RR\(_1)/RR\(_2\) had a significant effect on LV systolic pressure or dP/dt min in the postextrasystolic beat. Postextrasystolic dP/dt max showed a tendency to increase progressively as the RR\(_1)/RR\(_2\) declined; values associated with RR\(_1)/RR\(_2\) of 300/700 msec and 250/750 msec were significantly higher than control and 400/600 msec in both the APC and VPC studies. The slight differences between the APC and VPC postextrasystolic increase in dP/dt max was not statistically significant. There was no significant change in \( \tau \) with variations in RR\(_1)/RR\(_2\) in either the APC or the VPC studies. While \( \tau \) appeared to be slightly longer after APCs than after VPCs, this tendency was not statistically significant.

Because the postextrasystolic response to APC and VPC stimuli was similar, subsequent studies on the effects of PESP were obtained after APCs only. In this series of experiments, both LV pressure and length measurements were made, and the postextrasystolic response was studied before (baseline) and after the administration of propranolol. These data are summarized in table 2 and figures 2 and 3. In the

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**Figure 1.** A record of left ventricular (LV) and aortic pressure and myocardial segment length from a typical experiment. RR\(_c\) = control; RR\(_1\) = coupling interval; RR\(_2\) = fully compensatory pause.
Table 1. Effect of the Site of Premature Contractions on Postextrasystolic Left Ventricular Pressure Transients

<table>
<thead>
<tr>
<th>RR1/RR2</th>
<th>Control</th>
<th>400/600 msec</th>
<th>350/650 msec</th>
<th>300/700 msec</th>
<th>250/750 msec</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV pressure (mm Hg)</td>
<td>118 ± 9</td>
<td>121 ± 12</td>
<td>120 ± 11</td>
<td>121 ± 10</td>
<td>120 ± 10</td>
<td>123 ± 10</td>
</tr>
<tr>
<td>dP/dt max (mm Hg/sec)</td>
<td>1819 ± 563</td>
<td>2537 ± 570</td>
<td>2997 ± 654</td>
<td>3324 ± 860*</td>
<td>3705 ± 959*</td>
<td>1975 ± 604</td>
</tr>
<tr>
<td>dP/dt min (mm Hg/sec)</td>
<td>244 ± 536</td>
<td>2272 ± 433</td>
<td>2139 ± 369</td>
<td>2057 ± 297</td>
<td>2113 ± 216</td>
<td>2608 ± 482</td>
</tr>
<tr>
<td>r (msec)</td>
<td>33 ± 3</td>
<td>36 ± 3</td>
<td>35 ± 3</td>
<td>36 ± 4</td>
<td>36 ± 3</td>
<td>31 ± 3</td>
</tr>
</tbody>
</table>

* p < 0.05 vs control and 400/600 msec (analysis of variance).
Abbreviations: RR1/RR2 = coupling interval/compensatory pause; control RR interval = 500 msec; LV = left ventricular; r = time constant of LV isovolumic pressure decline.

Table 2. Effects of Postextrasystolic Potentiation Before and After Propranolol

<table>
<thead>
<tr>
<th>RR1/RR2</th>
<th>Control</th>
<th>400/600 msec</th>
<th>350/650 msec</th>
<th>300/700 msec</th>
<th>250/750 msec</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV systolic pressure (mm Hg)</td>
<td>127 ± 8</td>
<td>124 ± 9</td>
<td>122 ± 8</td>
<td>121 ± 9</td>
<td>126 ± 7</td>
<td>126 ± 7</td>
</tr>
<tr>
<td>AoDP (mm Hg)</td>
<td>108 ± 8</td>
<td>98 ± 4*</td>
<td>95 ± 9*</td>
<td>95 ± 9*</td>
<td>95 ± 8*</td>
<td>115 ± 13</td>
</tr>
<tr>
<td>dP/dt max (mm Hg/sec)</td>
<td>2534 ± 777</td>
<td>3711 ± 989*</td>
<td>3806 ± 613*</td>
<td>4314 ± 888*</td>
<td>4588 ± 951*</td>
<td>1730 ± 474</td>
</tr>
<tr>
<td>dP/dt min (mm Hg/sec)</td>
<td>2455 ± 730</td>
<td>2236 ± 282</td>
<td>2168 ± 218</td>
<td>2228 ± 251</td>
<td>2179 ± 281</td>
<td>2442 ± 324</td>
</tr>
<tr>
<td>r (msec)</td>
<td>35 ± 3</td>
<td>36 ± 3</td>
<td>36 ± 4</td>
<td>36 ± 3</td>
<td>36 ± 3</td>
<td>39 ± 3</td>
</tr>
<tr>
<td>EDSL (mm)</td>
<td>10.1 ± 1.7</td>
<td>10.7 ± 1.7</td>
<td>10.6 ± 1.7</td>
<td>10.7 ± 1.7</td>
<td>10.7 ± 1.7</td>
<td>10.8 ± 1.4</td>
</tr>
<tr>
<td>ESSL (mm)</td>
<td>8.6 ± 1.6</td>
<td>8.3 ± 1.6</td>
<td>8.2 ± 1.7</td>
<td>8.1 ± 1.7*</td>
<td>8.1 ± 1.6*</td>
<td>9.4 ± 1.2</td>
</tr>
<tr>
<td>% shortening</td>
<td>16 ± 3</td>
<td>22 ± 5*</td>
<td>23 ± 6*</td>
<td>24 ± 5*</td>
<td>24 ± 5*</td>
<td>13 ± 3</td>
</tr>
</tbody>
</table>

* p < 0.05 (RR1/RR2 vs control) by analysis of variance.
Abbreviations: PESP = postextrasystolic potentiation; EDSL = end-diastolic segment length; RR1/RR2 = coupling interval/compensatory pause; ESSL = end-systolic segment length; LV = left ventricular; AoDP = aortic diastolic pressure; r = time constant of LV isovolumic pressure decline.
baseline studies, there was no change in LV systolic pressure with PESP. AoDP after an RR₁/RR₂ of 400/600 msec was significantly lower than control, but there was no further decline in AoDP with a further decrease in RR₁/RR₂. As in the APC-VPC studies, a progressive decrease in the RR₁/RR₂ was associated with a progressive increase in dP/dt max. There was no significant change in the dP/dt min or in the τ over the entire range of RR₁/RR₂. The postextrasystolic increase in end-diastolic segment length did not achieve statistical significance, but the decrease in end-systolic segment length was significant at RR₁/RR₂ of 300/700 msec and 250/750. The postextrasystolic increase in fractional shortening was statistically significant at an RR₁/RR₂ of 400/600 msec, but shortening did not progressively increase thereafter.

After propranolol, the small increase in postextrasystolic pressure did not achieve statistical significance, but AoDP declined significantly. As in the baseline studies, the initial decline in AoDP was statistically significant but was not progressive as the RR₁/RR₂ was increased. After propranolol, values for dP/dt max were significantly lower than baseline at all levels of RR₁/RR₂; dP/dt max increased progressively with a progressive decrease in RR₁/RR₂. As in the baseline studies, dP/dt min and τ were not influenced by varying the RR₁/RR₂. End-diastolic and end-systolic lengths increased after the administration of propranolol, and the tendency for end-diastolic length to increase and end-systolic length to decrease (with a decrease in RR₁/RR₂) was similar in the baseline and propranolol studies. In contrast to the baseline studies, the increase in end-diastolic dimension after the extrasystole was statistically significant in the propranolol studies. Propranolol reduced fractional shortening, but, as in the baseline state, there was a statistically significant increase in shortening with PESP.

A comparison of the effects of three inotropic inter-
TABLE 3. Effect of Calcium, Isoproterenol, and Postextrasystolic Potentiation

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Calcium</th>
<th>Control</th>
<th>Isoproterenol</th>
<th>Control</th>
<th>PESP</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV systolic pressure (mm Hg)</td>
<td>124 ± 21</td>
<td>128 ± 13</td>
<td>122 ± 21</td>
<td>123 ± 25</td>
<td>126 ± 8</td>
<td>122 ± 8</td>
</tr>
<tr>
<td>dP/dt max (mm Hg/sec)</td>
<td>2337 ± 471</td>
<td>3630 ± 592*</td>
<td>1830 ± 461</td>
<td>3030 ± 938*</td>
<td>2535 ± 777</td>
<td>3806 ± 613*</td>
</tr>
<tr>
<td>dP/dt min (mm Hg/sec)</td>
<td>1908 ± 987</td>
<td>2470 ± 479</td>
<td>1898 ± 584</td>
<td>2108 ± 587</td>
<td>2535 ± 370</td>
<td>2168 ± 218</td>
</tr>
<tr>
<td>r (msec)</td>
<td>30 ± 5</td>
<td>27 ± 5*</td>
<td>38 ± 4</td>
<td>30 ± 6*</td>
<td>35 ± 3</td>
<td>36 ± 3</td>
</tr>
<tr>
<td>EDSL (mm)</td>
<td>9.8 ± 1.2</td>
<td>9.7 ± 1.2</td>
<td>9.7 ± 1.3</td>
<td>9.7 ± 1.2</td>
<td>10.1 ± 1.7</td>
<td>10.6 ± 1.7</td>
</tr>
<tr>
<td>ESSL (mm)</td>
<td>7.8 ± 1.6</td>
<td>7.2 ± 1.7*</td>
<td>8.3 ± 1.6</td>
<td>7.6 ± 1.9*</td>
<td>8.6 ± 1.6</td>
<td>8.2 ± 1.7*</td>
</tr>
<tr>
<td>% shortening</td>
<td>20 ± 11</td>
<td>26 ± 11*</td>
<td>14 ± 4</td>
<td>23 ± 11†</td>
<td>16 ± 3</td>
<td>23 ± 6†</td>
</tr>
</tbody>
</table>

*p < 0.05 vs control.
†p < 0.01 vs control.

Abbreviations: PESP = postextrasystolic potentiation (RR1/RR2 = 350/650 msec); LV = left ventricular; r = time constant of LV isovolumic pressure decline; EDSL = end-diastolic segment length; ESSL = end-systolic segment length.

ventions (PESP, isoproterenol, and calcium) is shown in table 3. The three interventions produced an equivalent increase in dP/dt max (approximately 50% by experimental design). LV systolic pressure and dP/dt min were not influenced by the three inotropic interventions (all p = NS), but fractional shortening increased and end-systolic length decreased with all three interventions. Isoproterenol produced a greater decrease in r than did calcium (p < 0.05); PESP did not influence r.

Discussion

In isolated cardiac muscle, an abrupt reduction in cycle length results in an initial prolongation of the plateau phase of the action potential.12, 13 Although the mechanism responsible for this remains unclear, the degree of prolongation of the action potential and the subsequent force potentiation both increase with the prematurity of the extrastimulus. Increased calcium influx during a prolonged plateau phase of the action potential should augment calcium availability at the contractile sites; this increased calcium availability is probably responsible for the PESP of contractility. In the intact heart, the magnitude of the postextrasystolic response involves a complex interaction between the timing of the extrastimole, variations in ventricular filling after the extrastimole, and changes in aortic input impedance. Although it is known that APCs induce PESP,4 it is not known whether APCs and VPCs produce the same postextrasystolic response. Despite differences in the speed of propagation and potential differences in the morphology of the action potential, we found no differences in the LV pressure transients when potentiation was produced by APCs or VPCs. For this reason and because atrial extrasystoles may be safer and easier to produce in man,5 we used APCs to study the effect of PESP on LV contraction and relaxation.

Contraction

Increased filling during a compensatory pause might be expected to contribute to the increased stroke volume in a postextrasystolic contraction. However, our data suggest that variations in preload play a limited role in determining the magnitude of the postextrasystolic response. End-diastolic length tended to increase after an RR1/RR2 of 400/600 msec; thereafter, despite a substantial increase in diastolic filling time (progressive decrease in the RR1/RR2), there was no further increase in end-diastolic length. Similarly, fellow and associates14 measured filling volume after random extrasystoles in a canine preparation and found that even with filling time increased by as much as 76%, filling volume increased by only 6%. The shape and slope of the LV diastolic pressure-volume curve are probably the most important determinants of the amount of filling that can take place for a given filling pressure. Despite the absence of the restraining influence of a pericardium, we did not see a progressive increase in end-diastolic length in the presence of a progressive increase in filling time. A number of other factors might account for the lack of increased filling with increased filling time. The extrasystole might interrupt filling, or, in the case of late extrasystoles, a portion of the ventricular volume might be ejected. It is also possible that at low left atrial pressures, collapse of the large mediastinal pulmonary veins may limit flow during some phase of the filling period.

Our data are consonant with the accepted concept that PESP of the contractile state is unrelated to changes in end-diastolic fiber length. Nor was there a progressive change in AoDP or LV systolic pressure. Thus, we believe that the progressive increase in LV dP/dt max and the decrease in end-systolic length reflect a progressive increase in the inotropic state of the ventricle. The administration of propranolol resulted in an increase in end-diastolic and end-systolic length, a decrease in dP/dt max and a small reduction in fractional shortening, but the LV response to PESP was qualitatively similar in the baseline and propranolol studies. In the propranolol studies, the tendency for LV systolic pressure to increase with PESP, which is generally considered an abnormal response,15 was not statistically significant. Apparently, the attenuation of sympathetic tone after
propranolol did not depress the ventricle enough to result in an abnormal postextrasystolic pressure response.

**Relaxation**

In the intact heart, Karliner and associates found that factors that augment the extent and velocity of fiber shortening result in more rapid isovolumic relaxation velocity. While there has been some disagreement on this point, we have found that, in the intact animal, LV isovolumic relaxation velocity (\( \tau \)) is directly related to end-systolic load; interventions that reduce end-systolic pressure and length are associated with increased relaxation velocity. Because PESP augments LV inotropic state and results in increased fractional shortening and decreased end-systolic length, it seemed that this intervention should increase relaxation velocity. However, Kuiper et al. have suggested otherwise; they found that \( \Delta P/\Delta t \) min was not influenced by PESP. Moreover, in isolated cat papillary muscle, paired stimulation results in increased or decreased relaxation velocity, depending on whether afterloaded isotonic contractions or isometric contractions are studied. To date, there have been no other attempts to examine postextrasystolic relaxation. In our studies, the postextrasystolic beat exhibited an increased inotropic state, increased fractional shortening and decreased systolic length, but PESP did not influence isovolumic relaxation velocity.

We and others have observed load-dependent relaxation during acute hemodynamic interventions in the intact dog heart, but the mechanism by which neurohumeral, metabolic, and pharmacologic factors modulate the decay of activation are not well understood. Catecholamines promote deactivation and thus have a relaxing effect on cardiac muscle, presumably by augmenting calcium uptake by the sarcoplasmic reticulum. In the intact dog heart, both systolic load and the deactivation process probably influence relaxation velocity. Because relaxation velocity was unchanged in the postextrasystolic beat under mechanical conditions that should have enhanced relaxation, it appears that deactivation must have been slowed in the postextrasystolic beat. The explanation for slowed deactivation remains speculative, however. The increased calcium influx that occurs with a premature contraction provides an increase in intracellular calcium, which is in part responsible for the PESP of contraction. The cardiac relaxing system must then remove this calcium from the force-generating sites and, if this system is saturated, deactivation might be slowed. This concept is supported by data from isometrically contracting cat papillary muscles in which an increase in extracellular calcium or paired stimulation results in prolonged isometric relaxation time. It is difficult to compare directly these isolated muscle studies with our intact heart studies because in the studies of isometric contractions, tension declines while the muscle is at its preloaded length, whereas in the intact heart, tension declines while the muscle is at a shorter (end-systolic) length. Nonetheless, we speculate that \( \tau \) in the postextrasystolic beat reflects the sum of factors that have opposing influences on relaxation rate; that is, the effects of a smaller end-systolic length (which tends to decrease \( \tau \)) are cancelled by an increase in intracellular calcium (which tends to increase \( \tau \)); therefore, \( \tau \) remains unchanged in the postextrasystolic beat.

The response of the left ventricle to systemically administered calcium was similar to that of PESP except that the former produced a small decrease in \( \tau \), while the latter did not. In contrast, isoproterenol clearly enhanced relaxation velocity. Catecholamines enhance myocardial relaxation rate by influencing the rate of cyclic-AMP-dependent processes in the sarcoplasmic reticulum, but the extent to which calcium and PESP influence deactivation remains unknown. In the intact heart it is difficult to separate the relative contributions of load-dependent and deactivation-dependent mechanisms. Nevertheless, it is clear that some positive inotropic interventions produce a more rapid LV relaxation, while others do not.

**References**

Angiographic Anatomy of the Normal Heart Through Axial Angiography

RICARDO CEBALLOS, M.D., BENIGNO SOTO, M.D., AND LIONEL M. BARGERON, JR., M.D.

SUMMARY We sectioned a series of hearts in a manner similar to that seen on angiographic axial views. A correlation with normal angiograms to identify the anatomic components of the four cardiac chambers showed that the components of the normal cardiac anatomy can be identified accurately through axial angiography in a manner not shown previously.

KNOWLEDGE of the normal appearance and relationships of the different cardiac segments is necessary for diagnosing malformations. The angiographic anatomy of the normal heart has been described as seen by standard frontal and lateral views.1,2 With the recent appearance of axial angiography3 these descriptions are not accurate or complete enough. In this report, we analyze the anatomy of the normal heart using axial angiography and compare these images with actual specimens.

Material and Methods

We studied a series of normal hearts from patients without heart disease during life or cardiac lesions at postmortem examination. The hearts were cut following the orientation of the radiographic axial views. Photographs were taken of the cut surfaces simulating the appearance shown on the projections of axial angiography.

The angiographic material was obtained from the Pediatric Cardiology Division of the University of Alabama in Birmingham and consisted of normal angiograms performed during investigation for possible cardiac malformations. Therefore, the anatomic specimens and angiograms shown in this paper do not correspond to the same patient.

Radiographic pictures are volume images, while sections of fixed hearts represent planes, or tomograms. Therefore, the anatomic pictures do not at times give the full set of findings as seen by x-rays, and a series of them, at different levels, may be necessary.4,5 The projections in axial angiography are: long-axis view (60° left anterior oblique plus 30° craniocaudal angulation), elongated right anterior oblique (30° right anterior oblique plus 30° craniocaudal angulation) and “four-chamber” view (45° left anterior oblique view plus 30° craniocaudal angulation).6 Different anatomic specimens were sectioned at these angles and each of the four chambers in each view was photographed.

Results

Angiographic Anatomy of the Cardiac Chambers

Right Atrium

Long-axis view. The left border is formed by a straight contour representing the atrial septum in its most anterior portion. The upper septal contour is formed by the superior atrial free wall. The anterolateral wall between superior and inferior venae cavae is seen on the right side. The anterior wall and the atrial appendage are not seen in this view because they overlie the atrial chamber. The annulus of the tricuspid valve is located inferiorly and to the left, somewhat overlying the entrance of the inferior vena cava (fig. 1A). At the beginning of diastole a rapid flow through the annulus is easily seen (fig. 1B). An exact similarity is seen in the anatomic picture (fig. 2).

Elongated right anterior oblique view. The right atrium is shown as a globe-shaped structure. The superior and inferior venae cavae are in continuity with the posterior border seen on the right. The inferior contour is formed by the space between the in-
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