Intracardiac Electrography in Endocardial Cushion Defects

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SUMMARY Conduction of the sino-atrial impulse from the high right atrium to the ventricles was studied by intracardiac electrography in 21 unoperated patients, age 3 months to 11 years, with endocardial cushion defects (ECD). The high right atrium-to-low right atrium conduction time was prolonged in 15 of 18 subjects (mean 57 ± 20 msec). The low right atrium-to-His bundle conduction time (LRA-H) was normal in 16 of 17 subjects (mean 82 ± 30 msec), prolonged in one. The His-to-ventricle conduction time (H-V) was normal in 16 of 17 subjects (mean 37 ± 8 msec), equivocally short in one.

Nine patients with ECD, age 3 to 21 years, were studied postoperatively. One had an acquired complete atrioventricular block in the His bundle. Two had prolonged LRA-H and two prolonged H-V. The surface ECG failed to identify accurately either prolonged atrioventricular conduction or the site of prolongation.

ENDOCARDIAL CUSHION DEFECTS (ECD) include abnormalities in both the anatomy and the function of the specialized conduction system. These abnormalities account for the characteristic electrocardiographic QRS pattern as well as a predisposition to partial or complete atrioventricular block in the natural course or after surgical repair of the defect. Intracardiac electrography (IE) has proved to be useful in the investigation and management of conduction disturbances in adults and in children with other types of cardiac defects. Knowledge of the typical as well as the individual IE in patients with ECD may be important for proper interpretation of postoperative IE findings and findings in patients with advanced atrioventricular (A-V) block. Three groups of patients with ostium primum atrial septal defect (PASD) have been previously reported.

The purpose of the present study was to evaluate conduction of the sinus node impulse through the atria, A-V node, bundle of His, and bundle branches to the ventricles, using IE in a large group of patients with pre and postoperative ECD in order to determine the values in children with a variety of forms of ECD and the effects of surgery on them.

Subjects and Methods

Thirty subjects with ECD confirmed by selective angiocardiography had IE performed during diagnostic heart catheterizations, after giving informed consent. Twenty-one patients were studied before intracardiac repair and nine were studied postoperatively. Four of the nine postoperative patients were studied early after surgery, i.e., 12 weeks to six months, and five were studied one and one-half to 14 years after the operation. In two of the postoperative patients permanent and transient complete A-V block was the main indication for the catheterization study.

Among the unoperated patients, seven had PASD while the remaining 14 had angiographically demonstrated interventricular communications and were classified as the complete form of ECD or atrioventricularis communis (AVC). One of these had additional valvar and another infundibular pulmonic stenosis, two had patent ductus arteriosus and one had cor triatriatum and coarctation of the aorta. Seven of the postoperative patients had PASD and two AVC, one of whom has associated tetralogy of Fallot.

The age of the unoperated patients ranged between 3 months and 11 years (mean 4 years) and the age of the postoperative patients ranged from 3 to 21 years (mean 10 years). The pertinent clinical data of the unoperated patients are given in table 1.

Catheterizations were performed by the percutaneous sheath technique via the right or left femoral vein as previously reported. Digitalis was withheld for 24 hours prior to the study. We have found no significant effect from therapeutic doses of digitalis on conduction intervals when withheld for 24 hours prior to study so no attempt has been made to distinguish between digitalized and nondigitalized patients. Premedication consisted of meperidine 1 mg/lb, chlorpromazine 0.25 mg/lb, and promethazine 0.25 mg/lb given thirty minutes before starting the catheterization.

In each patient a bipolar catheter with 10 mm interelectrode distances, a tripolar catheter with 1 mm and 10 mm interelectrode distances, or a quadripolar catheter with 5 mm interelectrode distances was advanced to the right ventricle to slow withdrawal with clockwise rotation until a potential was recorded occurring between the atrial and ventricular depolarizations. Withdrawal was continued until the catheter tip was completely within the right atrium. One to three surface leads of the ECG were recorded simultaneously with the IE. In 19 studies, high right atrial potentials were recorded after the catheter tip was moved to the region near the entrance of the superior vena cava (fig. 1). In four studies, a second quadripolar catheter was introduced percutaneously and positioned in the high right atrium for recording and atrial pacing, while the first catheter was left in position for recording the His bundle and low right atrial potentials. All records were obtained through an Electronics for Medicine junction box on a multichannel photographic recorder at paper speeds of 100 and 200 mm/sec. The filter setting for the surface ECG was 0.1 to 200 Hz and for the IE recordings 40 to 500 Hz.
The conduction intervals searched for and measured when possible were the following: high right atrium to low right atrium (HRA-LRA), low right atrium to His bundle (LRA-H), representing the A-V nodal conduction time; and His bundle to ventricle (H-V), representing the His-Purkinje conduction time. The P-R interval was measured also and the heart rate calculated from the cycle length at the time of interval measurements. The high right atrial activation was only accepted as being near the sinus node if it was at or before the P wave. The intervals were measured from

**Table 1. Pertinent Clinical Data and Findings by Intracardiac Electrography (IE) in 21 Unoperated Patients with Endocardial Cushion Defect**

<table>
<thead>
<tr>
<th>Age/Sex</th>
<th>Diagnosis</th>
<th>Resting heart rate</th>
<th>QRS A</th>
<th>P-R during IE (msec)</th>
<th>HRA-LRA (msec)</th>
<th>LRA-H (msec)</th>
<th>H-V (msec)</th>
<th>PR by standard ECG (sec)</th>
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</thead>
<tbody>
<tr>
<td>2yr/F</td>
<td>PASD</td>
<td>118</td>
<td>-30</td>
<td>175</td>
<td>50</td>
<td>95</td>
<td>42</td>
<td>0.18</td>
</tr>
<tr>
<td>3yr/F</td>
<td>PASD</td>
<td>110</td>
<td>-60</td>
<td>155</td>
<td>58</td>
<td>56</td>
<td>31</td>
<td>0.15</td>
</tr>
<tr>
<td>3yr/F</td>
<td>PASD</td>
<td>104</td>
<td>-60</td>
<td>133</td>
<td>50</td>
<td>-</td>
<td>14</td>
<td>0.16</td>
</tr>
<tr>
<td>5yr/M</td>
<td>PASD</td>
<td>111</td>
<td>-90</td>
<td>167</td>
<td>50</td>
<td>-</td>
<td>-150</td>
<td>0.16</td>
</tr>
<tr>
<td>5yr/F</td>
<td>PASD</td>
<td>120</td>
<td>-60</td>
<td>165</td>
<td>60</td>
<td>80</td>
<td>40</td>
<td>0.12</td>
</tr>
<tr>
<td>7yr/M</td>
<td>PASD</td>
<td>105</td>
<td>-90</td>
<td>125</td>
<td>-100</td>
<td>-</td>
<td>-150</td>
<td>0.12</td>
</tr>
<tr>
<td>13yr/M</td>
<td>PASD</td>
<td>63</td>
<td>-30</td>
<td>255</td>
<td>53</td>
<td>174</td>
<td>33</td>
<td>0.22</td>
</tr>
<tr>
<td>3mo/M</td>
<td>AVC</td>
<td>145</td>
<td>-120</td>
<td>125</td>
<td>13</td>
<td>79</td>
<td>27</td>
<td>0.11</td>
</tr>
<tr>
<td>6mo/F</td>
<td>AVC</td>
<td>160</td>
<td>-90</td>
<td>180</td>
<td>31</td>
<td>110</td>
<td>42</td>
<td>0.16</td>
</tr>
<tr>
<td>8mo/F</td>
<td>AVC,PDA</td>
<td>138</td>
<td>-150</td>
<td>-50</td>
<td>-</td>
<td>57</td>
<td>22</td>
<td>0.12</td>
</tr>
<tr>
<td>8mo/M</td>
<td>AVC</td>
<td>130</td>
<td>-120</td>
<td>149</td>
<td>55</td>
<td>79</td>
<td>31</td>
<td>0.16</td>
</tr>
<tr>
<td>8mo/F</td>
<td>AVC</td>
<td>150</td>
<td>-150</td>
<td>178</td>
<td>-</td>
<td>50</td>
<td>37</td>
<td>0.13</td>
</tr>
<tr>
<td>1yr-2mo/F</td>
<td>AVC</td>
<td>118</td>
<td>-120</td>
<td>147</td>
<td>80</td>
<td>70</td>
<td>26</td>
<td>0.18</td>
</tr>
<tr>
<td>1yr-4mo/F</td>
<td>AVC</td>
<td>136</td>
<td>-90</td>
<td>168</td>
<td>69</td>
<td>65</td>
<td>37</td>
<td>0.15</td>
</tr>
<tr>
<td>1yr-11mo/F</td>
<td>AVC,IPS</td>
<td>104</td>
<td>-120</td>
<td>145</td>
<td>55</td>
<td>60</td>
<td>40</td>
<td>0.14</td>
</tr>
<tr>
<td>3yr/F</td>
<td>AVC,CoA,CTr</td>
<td>135</td>
<td>-150</td>
<td>197</td>
<td>85</td>
<td>105</td>
<td>45</td>
<td>0.28</td>
</tr>
<tr>
<td>4yr/F</td>
<td>AVC,PDA</td>
<td>115</td>
<td>-90</td>
<td>167</td>
<td>-</td>
<td>73</td>
<td>52</td>
<td>0.18</td>
</tr>
<tr>
<td>7yr/F</td>
<td>AVC,PS</td>
<td>104</td>
<td>-150</td>
<td>130</td>
<td>35</td>
<td>61</td>
<td>39</td>
<td>0.12</td>
</tr>
<tr>
<td>7yr/F</td>
<td>AVC</td>
<td>103</td>
<td>-150</td>
<td>180</td>
<td>60</td>
<td>65</td>
<td>36</td>
<td>0.18</td>
</tr>
<tr>
<td>7yr/M</td>
<td>AVC</td>
<td>103</td>
<td>-120</td>
<td>205</td>
<td>-</td>
<td>104</td>
<td>41</td>
<td>0.20</td>
</tr>
<tr>
<td>11yr/F</td>
<td>AVC</td>
<td>100</td>
<td>-120</td>
<td>202</td>
<td>73</td>
<td>-</td>
<td>-</td>
<td>0.20</td>
</tr>
</tbody>
</table>

Number of subjects 21
Mean ± SD 118 ± 22
Difference from normal NS P <0.001 P <0.001 NS NS
Range 63-160 125-255 13-100 50-174 22-52

Abbreviations PASD = primum atrial septal defect; AVC = atrioventricular communis; PDA = patent ductus arteriosus; IPS = infundibular pulmonic stenosis; PS = valvar pulmonic stenosis; CoA = coarctation of the aorta; CTr = cor triatriatum; PR by IE = PR interval from surface leads during IE; HRA = high right atrium; LRA = low right atrium; H = His bundle; V = ventricle; PR by surface ECG = recorded prior to the IE study; NS = nonsignificant; SD = standard deviation.

**Figure 1. Demonstration of a sequential recording of the His bundle potential and high right atrial electrogram used to obtain conduction intervals in a preoperative patient with an endocardial cushion defect. A) Surface leads I, II, and III recorded simultaneously with three His bundle electrograms from a quadrupolar catheter with 10 mm interelectrode distances. B) Same format with the catheter moved to the high right atrium, superior vena cava junction. The high right atrium-to-low right atrium interval obtained by subtracting LRA-V from HRA-V is 85 msec which is markedly prolonged.**
recordings with 200 mm/sec paper speed from the onset of the first to the onset of the next potential. The onset of the potentials was defined as follows: HRA, the earliest deflection of the HRA potential; LRA, the first rapid deflection (deviating more than 45° from the baseline) of the LRA potential; H, the earliest deflection of the sharp, distinct potential following the LRA on the His bundle electrogram (HBE); V, the earliest deflection of ventricular depolarization, whether occurring first on the IE or on the surface ECG. Our definition of HRA depolarization is different from the other electrograms. In order to minimize missing any atrial activation, we took any deflection on the HRA electrogram in a patient with high to low activation sequences as the depolarization of the atrium near the sinus node.

In order to avoid confusing a right bundle (RB) potential with an H potential, only HBE recordings showing a large LRA potential were accepted. The potential after the LRA potential and farthest from the V potential was selected as the H potential. All other potentials were designated RB potentials.

The HRA-LRA was obtained by subtracting the sum of the LRA-H and H-V intervals from the HRA-V in 19 patients, since simultaneous recording of HRA and LRA was performed in only four studies.

Identification of the His potential was not possible in five studies because the catheter tended to flip into the left atrium during the clockwise rotation.

Control Values

The conduction intervals in patients with ECD were compared with values from 27 subjects (age 3 days to 18 years) with a normal heart confirmed by catheterization and angiography and normal conduction by surface ECG (table 2). Previously published normal values for children and young adults, based on 85 subjects with normal conduction, 66 of whom had congenital cardiac defects, did not differ significantly from the normal values used here. Values for HRA-LRA were available in only 13 normals, but the intervals of an expanded group of 13 normals and 28 patients with congenital heart defects and normal conduction by surface ECG produced similar mean values and ranges. The HRA-LRA conduction time was determined indirectly in the majority, as described for the patient group.

Normal values for the P-R interval with regard to age and heart rate as reported by Alimurung and Massell were compared to the P-R interval on surface ECG.

Results

There was no statistically significant difference between any of the conduction intervals in patients with PASD and AVC, so they are reported as one group.

Table 2. Normal Conduction Intervals

<table>
<thead>
<tr>
<th></th>
<th>Mean ± 1 SD (msec)</th>
<th>Range (msec)</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>HRA-LRA</td>
<td>23 ± 9</td>
<td>6-35</td>
<td>13</td>
</tr>
<tr>
<td>LRA-H</td>
<td>75 ± 18</td>
<td>50-115</td>
<td>27</td>
</tr>
<tr>
<td>H-V</td>
<td>40 ± 9</td>
<td>25-55</td>
<td>27</td>
</tr>
<tr>
<td>P-R</td>
<td>127 ± 17</td>
<td>100-160</td>
<td>27</td>
</tr>
<tr>
<td>Heart rate</td>
<td>113 ± 30</td>
<td>60-180</td>
<td>27</td>
</tr>
</tbody>
</table>

Unoperated Patients (table 1)

The resting heart rate did not differ significantly from the normal controls. The mean variation in heart rate between recording the low and high right atrial depolarization was 4 ± 3 (sd) beats/min.

The P-R interval measured during IE exceeded the normal range in 14 of the 19 (74%) patients in whom it was measured. The mean value of 170 ± 31 msec was 43 msec longer than the mean of the normals (P < 0.001). The mean variation in P-R interval between HRA and LRA recording was 8 ± 4 msec.

The HRA-LRA or internodal conduction time exceeded the normal range in 15 of 18 patients measured and was normal in three. The mean value of 58 ± 20 msec was significantly longer than the normal mean, which it exceeded by 35 msec (P < 0.001).

The LRA-H or A-V nodal conduction time was prolonged in one patient (174 msec) and normal in 16. The mean value of 82 ± 30 msec did not differ significantly from the normal mean.

The H-V or His-Purkinje conduction time was normal in 16 and equivocal in one (22 msec). The mean value of 37 ± 8 msec did not differ significantly from the normal mean. Previous data on the normal values for H-V intervals are conflicting. Our study showed no change with age whereas the studies of Roberts and Abella showed an increase with age.

The P-R interval of the surface ECG prior to catheterization had a mean of 0.16 ± 0.04 sec. It exceeded the normal limits for age and heart rate in 11 (52%). In seven of these, the only prolonged interval was the HRA-LRA; one had both prolonged HRA-LRA and LRA-H, and one had normal intervals by IE. In two subjects, HRA was not recorded but LRA-H and H-V were normal.

Postoperative Patients (table 3)

One patient, a 21-year-old woman who was one of the subjects of a previous report, had complete A-V block acquired at operation for a PASD 14 years previously. She had a syncopal attack one year after surgery and was treated with isoproterenol and had no further episodes until the day of admission, when she had an Adams-Stokes attack. Electrophysiologic study found complete A-V block in the bundle of His as demonstrated by split His potentials. The ventricular rate was 42. She was treated with an implanted pacemaker.

HRA-LRA was prolonged in three of the five patients in whom it was measured. LRA-H was slightly prolonged in two of seven. The H-V interval was prolonged in two of eight patients. One of the patients with prolonged H-V had had complete A-V block following the operation for a PASD six years previously in another hospital, and was treated with a temporary pacemaker for ten days until she returned to sinus rhythm. During the last year before the study, she had had several fainting spells or tonic seizures of undetermined nature. The P-R interval of 0.18 sec was within the normal range for age and heart rate. The A-V nodal conduction was normal and she responded normally to atrial pacing up to a maximum rate of 181/min; therefore she was treated with diphenylhydantoin rather than a pacemaker. She had only
one seizure during 18 months of follow-up and this presumably was of cerebral origin.

Among the nine patients studied postoperatively, five were found to have abnormal atrioventricular conduction. In two of these the P-R interval was within normal limits. One patient with prolonged P-R had normal A-V conduction but prolonged internodal conduction.

### Discussion

A prolonged P-R interval has been reported to occur in greater than 50% of patients with ECD.\(^1\) The most constant electrocardiographic feature of endocardial cushion defect is a left or superior QRS mean frontal plane axis and a counter-clockwise frontal plane QRS loop, as in left anterior hemiblock pattern.\(^14\) While this characteristic QRS abnormality indicates an abnormal pattern of ventricular activation, it does not necessarily indicate atrioventricular conduction disturbance.

Fifty-two percent of our unoperated patients had a P-R interval longer than normal for age and heart rate, while the others most often had values for P-R in the upper part of the normal range. The mean P-R was significantly prolonged compared to normal. Our findings by IE show that the prolonged P-R interval is due to prolonged internodal conduction (HRA-LRA). Previous studies in PASD have been consistent with this finding.\(^6\) The intra-atrial conduction was studied in greater detail by Waldo et al.\(^7\) by direct endocardial recording and stimulation during open heart surgery. Compared to controls with other congenital heart defects, the internodal conduction time was uniformly prolonged by a mean value of 37 msec, which is comparable to our value of 35 msec in intact patients, compared to normal controls. Waldo et al. further suggested an abnormal intra-atrial conduction pattern and the authors speculated that the anterior and middle internodal pathways could be interrupted or distorted in their course across the atrial septum due to the location of the atrial septal defect.

No previous study has reported values for the HRA-LRA interval in ECD in hearts which had not been opened surgically. The HRA usually appears before the onset of the P wave rather than exactly at its onset, the latter being less well defined on high paper speed recordings. Thus, we find that HRA-LRA is a more accurate measurement of internodal conduction than the P-A (beginning of P wave on surface ECG to LRA) interval used by others.\(^6\) However, two catheters are required to record simultaneous HRA and LRA potentials, which complicates the procedure. Sequential recording of HRA and LRA potentials was used in the majority of our patients as well as our normal controls.

Our study confirms one part of the previous studies in ECD, that A-V nodal conduction is normal in the great majority of patients.\(^6\) Only one patient was found to have abnormalities in this region and this could be coincidental. While complete A-V block is a known but rare complication during the natural course of ECD,\(^1,6\) it is not yet known in which part of the conduction system this nonsurgical block occurs in ECD.

We found the H-V interval to be normal in 16 of our 17 preoperative studies and possibly short in one. This confirms the data of Waldo who found the H-V normal in all of his 13 patients studied during open-heart surgery.\(^7\) The H-V was thought to be abnormally short in six of the eight patients reported by Miller et al.\(^8\) and three of the seven studied by Goodman et al.\(^8\) These investigators thought that the short H-V resulted from the short distance between the A-V node and the earliest branching of the posterior fascicle of the left bundle branch. However, Rosenbaum et al.\(^8\) pointed out that the 1.6 to 3.6 mm shorter distance reported would mean very little in terms of conduction time, as the velocity of conduction in the proximal Purkinje system is approximately 2 mm per msec. The short H-V interval previously reported in ECD and found in one of our patients could be due to mistaking a bundle branch potential for an H potential. On occasion, an obvious RB potential may be recorded together with a fairly large atrial potential in children, yet further withdrawal of the catheter reveals the H potential. We have found the His bundle potential particularly difficult to record in patients with ECD. This may be due to the posteroinferior displacement of the His bundle together with the difficulty in obtaining a stable position of the catheter in the usual region for recording. For the purpose of identifying conduction defects, it is important to know that the limits for the intervals in patients with ECD are similar to those found in normal hearts and most other congenital defects.

The findings in our nine patients studied postoperatively were similar to four of our unoperated patients. In one postoperative patient there was complete block in the bundle of His. Two patients had prolonged A-V nodal conduction and two had prolonged His-Purkinje conduction. Three of the four patients with prolonged A-V conduction had normal P-R intervals. One of those with a prolonged LRA-H had decreased her P-R by 0.02 sec postoperatively. One

### Table 3. Pertinent Clinical Data and Findings by Intracardiac Electrography in Nine Postoperative Patients with Endocardial Cushion Defect

<table>
<thead>
<tr>
<th>Age/Sex</th>
<th>Diagnosis and postoperative period</th>
<th>Resting heart rate</th>
<th>PR by IE (msec)</th>
<th>HRA-LRA (msec)</th>
<th>LRA-H (msec)</th>
<th>H-V (msec)</th>
<th>PR by surface ECG (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>3/M</td>
<td>PASD, 4 mo</td>
<td>108</td>
<td>65</td>
<td>95</td>
<td>95</td>
<td>0.18</td>
<td></td>
</tr>
<tr>
<td>7/M</td>
<td>PASD, 3 mo</td>
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<td>200</td>
<td>51</td>
<td>100</td>
<td>0.20</td>
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<tr>
<td>10/M</td>
<td>PASD, 6 yr</td>
<td>77</td>
<td>200</td>
<td>130</td>
<td>50</td>
<td>0.18</td>
<td></td>
</tr>
<tr>
<td>12/F</td>
<td>PASD, 6 yr</td>
<td>75</td>
<td>100</td>
<td>70</td>
<td>74</td>
<td>0.18</td>
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<tr>
<td>16/F</td>
<td>PASD, 11 yr</td>
<td>71</td>
<td>120</td>
<td>47</td>
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<tr>
<td>17/F</td>
<td>PASD, 6 mo</td>
<td>66</td>
<td>175</td>
<td>120</td>
<td>120</td>
<td>0.18</td>
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<td>21/F</td>
<td>PASD, CHB, 14 yr</td>
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<td>26</td>
<td>26</td>
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<td>0.18</td>
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<tr>
<td>4/F</td>
<td>AVC, 12 wk</td>
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<td>0.18</td>
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<td>8/F</td>
<td>AVC, TF, 18 mo</td>
<td>100</td>
<td>35</td>
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<td>100</td>
<td>0.14</td>
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</table>

Abbreviations: CHB = complete A-V block; TF = tetralogy of Fallot.
MANNITOL AND CELL SWELLING IN ISCHEMIA/Powell et al. 603

patient with prolonged P-R had a normal A-V conduction but prolonged internodal conduction. The surface ECG thus may not accurately represent the status of the A-V conduction system in patients with ECD, particularly in the postoperative state. The prognostic significance of the abnormalities uncovered by IE remains to be determined.

References

The Protective Effect of Hyperosmotic Mannitol in Myocardial Ischemia and Necrosis

WM. JOHN POWELL, JR., M.D., DONALD R. DiBONA, PH.D., JORGEFlores, M.D., AND ALEXANDER LEAF, M.D.

SUMMARY Morphologic and hemodynamic changes that occur following coronary occlusion are examined. The effectiveness of hyperosmotic mannitol in lessening the extent of myocardial damage is assessed and mechanisms for its action discussed. Forty and 60 min of coronary vascular occlusion followed by 15 and 45 min of reflow were associated with a persistence of ischemia following reflow of blood, as established by infusions of silastic into the aortic root.

Electron microscopic studies demonstrated myocardial and endothelial cell swelling at the end of the reflow period. The process of cell swelling appeared to be initiated during the period of arterial occlusion. This cell swelling was reduced by elevation of serum osmolality by 30–40 mOsm above control with the administration of mannitol during and following occlusion. There was an associated 40–50% reduction of vascular resistance following occlusion if mannitol was administered. In addition, the extent of necrosis, which was widespread in untreated hearts 12 hours after occlusion, was strikingly less in the hearts of dogs which received mannitol. Thus, in ischemic myocardium, elevation of osmolality by mannitol reduces myocardial necrosis, probably through its restoration of normal cell volume.

THE HEMODYNAMIC EFFICACY of the use of hyperosmotic mannitol in experimentally induced myocardial ischemia has been documented.1 Elevation of extracellular osmolality during a period of constriction of a coronary artery produced the following acute effects: 1) a decrease in amount of ST-segment elevation over the ischemic muscle;2) an increase in collateral blood flow to the area of ischemia;3) an improvement in the function of the ischemic myocardium; and 4) a reduction in oxygen consumption of ischemic cardiac muscle.4 These favorable effects on function were all discernible in the acute experimental animal preparation. The present study was designed to answer the important clinical question of whether or not treatment with hyperosmotic mannitol protects ischemic myocardial cells from necrosis. Our results, based on studies carried out in the experimental canine model, indicate that it does. The study attempts to determine the mechanism of action of mannitol using histologic measures. A preliminary report of the data has been presented.5

Methods

The experiments were performed in 80 adult mongrel dogs, anesthetized with intravenous sodium pentobarbital

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Supported by grants N01 HV 71443, HL 17665, PHS AM17372, and HL 06664 (HEPP) and NIH contract N01 HV 53002.

This work was presented at the Annual Meeting of the American Society for Clinical Investigation in Atlantic City, New Jersey, in May 1973.

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Received September 2, 1975; revision accepted May 10, 1976.
Intracardiac electrography in endocardial cushion defects.
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Circulation. 1976;54:599-603
do: 10.1161/01.CIR.54.4.599
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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