Preoperative Secundum Atrial Septal Defect with Coexisting Sinus Node and Atrioventricular Node Dysfunction

EDWARD B. CLARK, M.D., AND JOHN D. KUGLER, M.D.

SUMMARY Sinus node dysfunction in patients after repair of the secundum atrial septal defect has been ascribed to surgical damage. We studied 15 consecutive patients with secundum atrial septal defect before operative intervention. Noninvasive testing included 24-hour electrocardiographic monitoring and a standard 13-lead ECG. Intracardiac electrophysiologic techniques included corrected sinus node recovery time, sinoatrial conduction time, His bundle recording to measure AH and HV intervals, the atrial pacing rate at which atrioventricular node Wenckebach occurred, and atrioventricular nodal refractory period. The ECG revealed an ectopic atrial rhythm in two patients. Intracardiac electrophysiology showed an abnormal corrected sinus node recovery time (range -40 to 800 msec) in 10 patients. Five patients had evidence of atrioventricular nodal dysfunction with prolonged AH interval or abnormal atrial pacing rate at which atrioventricular Wenckebach occurred. These data indicate that sinus node dysfunction or atrioventricular node dysfunction were present before surgical intervention.

SINUS NODE DYSFUNCTION is a frequently recognized abnormality after surgical repair of secundum atrial septal defects (ASDs).1-4 Since the early days of intracardiac repair, the postoperative abnormalities of sinus node function have been ascribed to surgical damage to the node or its artery.5-7 However, in a previous study, we recognized abnormalities of sinus node function in preoperative ECGs.9 Therefore, we undertook this study in 15 patients with physical findings consistent with left-to-right shunt at the atrial level to identify the incidence of electrophysiologic anomalies.

Materials and Methods
The study group included nine girls and six boys, ages 10 months to 18 years, who were seen consecutively from June 1979 to December 1980 (table I). Patient 2 complained of occasional lightheadedness and syncope and patient 10 had a history of dizziness; the others were asymptomatic. All patients had a standard 13-lead ECG and 11 had 24-hour ambulatory electrocardiographic recordings. These studies were evaluated for evidence of sinus node dysfunction, including sinus bradycardia for age; sinus arrest with atrial, junctional or ventricular escape beats or rhythm; any type of sinoatrial exit block; and/or brady-tachy arrhythmias. In addition, careful attention was paid to identify evidence of atrioventricular (AV) node dysfunction, including first-, second- or
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AV block, ventricular escape beats or ventricular rhythm.

Patients 1–8 were sedated with meperidine, 2.5 mg/kg, promethazine, 0.6 mg/kg, and chlorpromazine, 0.6 mg/kg, and patients 9–15 were sedated with the same doses of meperidine and promethazine but without chlorpromazine. Sinus and AV node function studies were performed in conjunction with the diagnostic catheterization. In each patient except patients 1 and 2 (in whom one electrode catheter was used), at least two electrode catheters were used. After introduction by the percutaneous technique and under the guidance of fluoroscopy, one quadripolar catheter was positioned in the high right atrium near the sinoatrial node. The proximal pair of electrodes was used for recording high right atrial depolarization and the distal pair was used for atrial pacing. The second catheter (either tripolar or quadripolar) was positioned across the tricuspid valve orifice to record the low septal right atrial, His and right ventricular depolarizations. Surface ECG leads I, II and III or I, aVF and V1 were simultaneously recorded with the electrograms on an Electronics for Medicine photographic recorder (VR12) at a paper speed of 100 or 250 mm/sec. All electrophysiologic procedures were carried out before angiography.

The intracardiac electrophysiologic measurements included corrected sinus node recovery time (CSNRT) (duration of high right atrial pacing was 30 seconds), total sinoatrial conduction time (SACT), AH and HV intervals on His bundle electrogram, paced atrial rate at which the AV node developed the Wenckebach phenomenon, and the effective and functional refractory periods of the AV node.

In each patient, hemodynamic evaluation revealed a left-to-right shunt of 20–70% measured by oxygen consumption or indocyanine green dye curves. Catheter course and angiography identified an isolated secundum ASD. In patients who subsequently underwent surgical repair, an isolated secundum ASD was confirmed there with no unusual features either in size or location of the defect.

Values are mean ± SD. The null hypothesis for no difference between patients with and without sinus node dysfunction by age and magnitude of left-to-right shunt was evaluated using a two-sample $t$ test. A $p$ value $< 0.05$ was considered significant.

Results

Sinus Node Function

Ten patients had findings diagnostic of sinus node dysfunction (table 1). Patients 1 and 2 had frequent slow ectopic atrial or junctional rhythm on the surface ECG recording. Patient 7 had paroxysmal atrial flutter, patient 11 a wandering atrial pacemaker, patient 13 frequent premature atrial complexes and patient 9 occasional Mobitz type II second-degree AV block.

Intracardiac electrophysiologic evaluation revealed that 10 patients had abnormal sinus node function. Eight of the 10 had prolonged CSNRT, two of whom had prolonged SACT. Patients 1 and 7 had a short CSNRT but were classified as abnormal because of sinoatrial node entrance block. This was manifested in patient 1 by a negative CSNRT and in patient 7 by obtaining no reset responses with single premature atrial stimuli into sinus rhythm.

AV Node Function

AV node function studies were abnormal in five of the 15 patients (table 1). In patient 1, the AH interval was prolonged beyond the upper limit of normal for age. Five patients developed AV node Wenckebach during atrial pacing at rates of 120–153 beats/min. AV node Wenckebach below a rate of 160 beats/min is considered abnormal in children. In all patients, the His-to-ventricular activation time was normal. The effective and functional refractory periods of the AV node were normal in each patient in whom they were measured.

The mean age of patients with sinus node dysfunction was 10.6 ± 5.2 years, compared with 2.9 ± 2.3 years for those with normal sinus node function ($t = 3.03$, $p < 0.01$). A direct correlation of shunt size and sinus node dysfunction was also observed. In patients with sinus node dysfunction, the mean Qp/Qs was 2.7 ± 0.1:1 compared with 1.8 ± 0.6:1 in those with normal sinus and AV node function ($t = 1.90$, $p < 0.05$).

Discussion

Other investigators have observed an incidence of arrhythmias of 6–80% after ASD repair (table 2). Several investigators hypothesize that atrial dysrhythmias are a result of surgical injury occurring at the time of operation. However, our data suggest that electrophysiologic abnormalities of the sinus or AV node are present before repair. This possibility is supported by surface ECG findings in children. Clark et al. found junctional rhythm, atrial flutter and ectopic atrial rhythms in children before ASD repair. Each of these rhythms can be associated with sinus node dysfunction in children. Sealy and associates noted preoperative atrial arrhythmias in 10 of 57 patients younger than 20 years of age. Patients may not manifest evidence of sinus node dysfunction on surface ECG recordings; thus, it is reasonable to presume, on the basis of our data, that many of the patients without dysrhythmia (in addition to the dysrhythmia patients) had underlying sinus or AV node dysfunction.

To our knowledge, our study is the only intracardiac electrophysiologic evaluation of preoperative patients who have a secundum ASD. Therefore, we do not know if the 66% incidence of sinus node dysfunction and the 33% incidence of AV node dysfunction are representative. Information obtained in 15 consecutive, nonselected patients appears satisfactory.

Patients with secundum ASD have also been found to have a prolonged PR interval. Chen et al. noted first-degree AV block in 13 of 43 patients before surgical repair, while Hastrieter et al. reported that four of 13 infants had first-degree AV block in association with isolated secundum ASD. Anderson et al.
Table 1. Electrophysiologic Data of Preoperative Patients with Secundum Atrial Septal Defects

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Symptoms</th>
<th>ECG ARR</th>
<th>24-hour ECG ARR</th>
<th>Intracardiac electrophysiology</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sinoatrial node</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CSNRT (msec)</td>
</tr>
<tr>
<td>1</td>
<td>13</td>
<td>F</td>
<td>—</td>
<td>1° AV block</td>
<td>Sinus arrest junctional</td>
<td>-40</td>
</tr>
<tr>
<td>2</td>
<td>13</td>
<td>F</td>
<td>Syncope</td>
<td>—</td>
<td>2° AV block SAN arrest</td>
<td>800</td>
</tr>
<tr>
<td>3</td>
<td>18</td>
<td>M</td>
<td>—</td>
<td>—</td>
<td>Normal</td>
<td>450</td>
</tr>
<tr>
<td>4</td>
<td>4/6/12</td>
<td>F</td>
<td>—</td>
<td>—</td>
<td>Normal</td>
<td>470</td>
</tr>
<tr>
<td>5</td>
<td>6/6/12</td>
<td>M</td>
<td>—</td>
<td>—</td>
<td>NA</td>
<td>110</td>
</tr>
<tr>
<td>6</td>
<td>3/9/12</td>
<td>M</td>
<td>—</td>
<td>—</td>
<td>NA</td>
<td>80</td>
</tr>
<tr>
<td>7</td>
<td>6/3/12</td>
<td>M</td>
<td>—</td>
<td>—</td>
<td>Paroxysmal atrial flutter</td>
<td>—</td>
</tr>
<tr>
<td>8</td>
<td>1/4/12</td>
<td>F</td>
<td>—</td>
<td>—</td>
<td>NA</td>
<td>130</td>
</tr>
<tr>
<td>9</td>
<td>13</td>
<td>F</td>
<td>—</td>
<td>—</td>
<td>2 episodes Mobitz II AV block</td>
<td>280</td>
</tr>
<tr>
<td>10</td>
<td>10/6/12</td>
<td>M</td>
<td>Dizziness</td>
<td>—</td>
<td>Normal</td>
<td>340</td>
</tr>
<tr>
<td>11</td>
<td>10/6/12</td>
<td>M</td>
<td>—</td>
<td>—</td>
<td>Wandering atrial pacemaker</td>
<td>390</td>
</tr>
<tr>
<td>12</td>
<td>1/6/12</td>
<td>F</td>
<td>—</td>
<td>—</td>
<td>NA</td>
<td>130</td>
</tr>
<tr>
<td>13</td>
<td>10/12</td>
<td>F</td>
<td>—</td>
<td>—</td>
<td>Frequent PACs</td>
<td>330</td>
</tr>
<tr>
<td>14</td>
<td>16</td>
<td>M</td>
<td>—</td>
<td>—</td>
<td>Normal</td>
<td>440</td>
</tr>
<tr>
<td>15</td>
<td>1/4/12</td>
<td>F</td>
<td>—</td>
<td>—</td>
<td>Normal</td>
<td>120</td>
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<tr>
<td></td>
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<td>Normal*</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>&lt;275</td>
</tr>
</tbody>
</table>

*Wenkebach assumed to be in AV node because His could not be recorded.

Abbreviations: @CL = obtained at sinus cycle length; AH = atrial-His duration; ARR = arrhythmias; AV = atrioventricular; CSNRT = corrected sinus node recovery time; ERP = effective refractory period; FRP = functional refractory period; % green dye = left-right shunt calculated from green dye curves; HV = His to ventricular duration; NA = not available; Qp/Qs = pulmonary-to-systemic flow ratio; S = surgical repair; SACT = sinoatrial conduction time; SAN = sinoatrial node; SAN-EB = SAN entrance block; W@APR = atrial paced AV node Wenckebach rate; PACs = premature atrial complexes.

measured intracardiac conduction times in eight patients with secundum ASD and reported prolongation of the internodal conduction time; however, they did not perform sinus node or AV node function testing.18 Variable degrees of AV block are noted in patients with familial ASDs.17 In our patients, the AH and HV intervals were normal in 14 of 15 patients at rest. Functional abnormalities of the sinus and AV node were only revealed after pacing challenge.

Why should sinus and AV node abnormalities be associated with secundum ASDs? We do not believe this is a phenomenon related to drugs used for pre catheterization sedation. Although no details are available in children, Clapp et al.18 demonstrated a shortening of CSNRT and AV node conduction in awake puppies sedated with meperidine, chlorpromazine and promethazine. Therefore, if any sedative effect was present, one might speculate that it would result in an improvement in function, assuming the human heart responds similarly to these drugs.

Some investigators have suggested that hemodynamic forces from increased atrial blood flow are responsible for the atrial arrhythmias.4-18 Our patients with sinus node dysfunction had a significantly larger Qp/Qs than those without such dysfunction, and patients with sinus node dysfunction were older than those with normal testing. These results suggest that the duration of hemodynamic stress may also be important. However, the sample size is small, so we caution against drawing rigid conclusions on this issue.

Another possibility is the concurrent development of the atrial septum and nodal tissue. Anderson et al.20 recently reviewed the morphogenesis of the specialized conducting tissue. The sinoatrial and AV nodes develop from rings of specialized cells that exist between the primordial chambers of the heart tube.
development. The resultant alteration in morphogenesis may present clinically as a secondum ASD and be more subtly evident as abnormalities in the sinus and AV node function.

We believe these observations have long-term importance for children who have had a secondum ASD repaired. Pediatric cardiologists are becoming more aware of sinus node dysfunction as a cause of syncope. Several children have required pacemaker implantation for syncope after ASD.\(^{10,11}\) We suggest that postoperative patients be followed for life, because the incidence of arrhythmias increases with age.

Our findings call for further investigation. The electrophysiologic measurements performed in our patients added only 15 minutes to the length of the catheterization. In selected centers with suitable equipment and personnel, we believe prospective analysis of conducting system should be carried out in patients with secondum ASDs before surgical repair.

Acknowledgment

The authors thank the staff of the Cardiovascular Diagnostic Laboratory, Jan Carr, Bob West, Steve Jones and Mary White for their technical support for this study and Sue Kucera and Mary Fry for their secretarial assistance.

References

2. Reid JM, Stevenson JC: Cardiac arrhythmias following successful surgical closure of atrial septal defect. Br Heart J 29: 742, 1967
15. Hastreiter AR, Wennemark JR, Miller RA, Paul MH: Secundum atrial septal defects with congestive heart failure during in-
Regression of Myocardial Hypertrophy: Electrocardiographic-Echocardiographic Correlations After Aortic Valve Replacement in Patients with Chronic Aortic Regurgitation

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SUMMARY Serial electrocardiographic and echocardiographic left ventricular (LV) studies were performed in 21 patients before and after aortic valve replacement (AVR) for chronic aortic regurgitation. Changes in voltage (SV, + Rv) after AVR were assessed and evaluated relative to changes in LV mass. Muscle cross-sectional area (CSA) derived from echocardiographic dimension and wall thickness data was used as an index of LV muscle mass (LV hypertrophy > 10 cm²/m²). In 15 patients, voltage was reduced after AVR: Seven had normal voltage (48 ± 17 mm to 25 ± 6 mm, p < 0.005) and eight still had increased voltage (61 ± 17 mm to 40 ± 4 mm, p < 0.01). Patients with normal voltage had complete regression of hypertrophy by echocardiography (CSA decreased from 13 ± 3 cm²/m² to 9 ± 1 cm²/m², p < 0.025), while those who had persisted increased voltage had incomplete regression (15 ± 2 cm²/m² to 11 ± 2 cm²/m², p < 0.001). Reduction in voltage generally occurred in the first 6 months after AVR. Three patients with unchanged voltage had evidence of paraprosthetic regurgitation and minimal change in CSA. Three other patients with persistent LV enlargement without paraprosthetic regurgitation had a severe intraventricular conduction delay. Data from 59 electrocardiographic-echocardiographic studies before and after AVR revealed a strong correlation (r = 0.81) between voltage and muscle CSA.

After surgical correction of chronic aortic regurgitation, regression of LV hypertrophy can be assessed by serial electrocardiographic studies. These ECG data identify patients with complete, incomplete or no regression of LV hypertrophy.

ELECTROCARDIOGRAPHIC evidence of left ventricular hypertrophy (LVH) is generally associated with angiographic, echocardiographic, and autopsy evidence of increased LV myocardial mass. However, there is little ECG evidence to support the idea that regression of LVH is an expected consequence if the stimulus for hypertrophy is removed. After aortic valve replacement for chronic aortic regurgitation, LV mass may be reduced; a few patients achieve normal LV mass and others exhibit little or no regression of hypertrophy. The value of the ECG in assessing these structural changes has not been studied in detail. Therefore, we designed the present study to test the hypothesis that ECG voltage is related to LV mass in patients with chronic aortic regurgitation and that the extent of regression of LVH after aortic valve replacement is reflected by a commensurate decrease in ECG voltage.

Methods Twenty-one patients who underwent aortic valve replacement for chronic aortic regurgitation between January 1975 and June 1979 participated in this study. Admission required pre- and postoperative ECGs and...
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_Circulation._ 1982;65:976-980
doi: 10.1161/01.CIR.65.5.976

_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

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