Conduction Abnormality and Arrhythmia After Transcatheter Closure of Atrial Septal Defect

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Background: The aim of this study was to prospectively perform ambulatory 24-h ECG monitoring to assess the effects of transcatheter closure of atrial septal defect (ASD).

Methods and Results: A total of 235 consecutive subjects (female, n=163; male, n=72; age, 44.6±14.4 years) were enrolled in the study, who were due undergo ASD closure. Holter monitoring was performed before procedure and at 1, 6 and 12 months of follow-up. During the procedure transient supraventricular arrhythmia occurred in 8 patients (3.4%), and bradycardia in 3 (1.3%). In 3 patients (1.3%) an episode of atrial fibrillation occurred in the first hour after the procedure. In 8 patients (3.4%) transient first-degree atrioventricular block was noted. A significant increase in number of supraventricular extrasystoles (SVES)/24 h was noted 1 month after the procedure (P<0.001). On multiple forward stepwise regression analysis, device size and fluoroscopy time had an influence on increase in number of SVES seen 1 month after the procedure (P<0.001).

Conclusions: Transcatheter closure of ASD is associated with a transient increase in supraventricular premature beats and a small risk of conduction abnormalities and paroxysmal atrial fibrillation in early follow-up. Transcatheter closure of ASD does not reduce arrhythmia that appears prior to ASD closure. Larger device size and longer procedure time are associated with increased risk of supraventricular arrhythmia on early follow-up. (Circ J 2014; 78: 2415–2421)

Key Words: Arrhythmia; Atrial septal defect; Percutaneous closure
Amplatzer devices were implanted without any major complication in all eligible individuals. The mean ratio of pulmonary to systemic blood flow or Qp:Qs was 1.54 ± 0.9 (range, 1.1–2.5) on echocardiography.

Transient SVT was observed in 8 patients (3.4%) during the procedure and transient bradycardia to 30 beats/min in 3 (1.3%). The mean procedure time including preceding right heart catheterization was 33.1 ± 9.5 min (range, 18–66 min) and the mean fluoroscopy time was 10.8 ± 6.7 min (range, 5–26 min). Diameter of the implanted ASO devices ranged from 12 to 40 mm (mean, 21.8 ± 7.5 mm). The ratio of ASO device size to ASD size was 1.09 ± 0.06 (range, 1.2–1).

Sinus rhythm (SR) was detected in 210 patients (89.4%) before ASD closure. Persistent AF was observed in 25 (10.6%). Left axis deviation was seen in 20 patients (8.5%), right axis deviation in 65 (27.7%), complete or incomplete right bundle branch block in 49 (20.9%), and first-degree atrioventricular block (AVB) in 33 (14%).

After ASD closure SR was present in 210 patients (89.4%). Persistent AF, observed in 25 subjects (10.6%), remained unchanged over the entire observation period. In 3 patients (1.3%)
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In the first hour after the procedure. Right and left axis deviation as well as first-degree AVB observed prior to ASD closure remained unchanged during follow-up. Table 1 lists ECG findings before and after ASD closure.

<table>
<thead>
<tr>
<th>Electrocardiographic Findings</th>
<th>Before ASD closure</th>
<th>After ASD closure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus rhythm</td>
<td>210 (89.4)</td>
<td>210 (89.4)</td>
</tr>
<tr>
<td>Paroxysmal AF</td>
<td>33 (14)</td>
<td>36 (15.3)</td>
</tr>
<tr>
<td>Persistent AF</td>
<td>25 (10.6)</td>
<td>25 (10.6)</td>
</tr>
<tr>
<td>Right axis deviation</td>
<td>65 (27.7)</td>
<td>65 (27.7)</td>
</tr>
<tr>
<td>Left axis deviation</td>
<td>20 (8.5)</td>
<td>20 (8.5)</td>
</tr>
<tr>
<td>Incomplete RBBB</td>
<td>33 (14)</td>
<td>33 (14)</td>
</tr>
<tr>
<td>Complete RBBB</td>
<td>16 (6.8)</td>
<td>16 (6.8)</td>
</tr>
<tr>
<td>First-degree AVB</td>
<td>33 (14)</td>
<td>36 (15.3)</td>
</tr>
<tr>
<td>SVT</td>
<td>23 (9.8)</td>
<td>51 (21.7)*</td>
</tr>
</tbody>
</table>

Data given as n (%). *P=0.0021. AF, atrial fibrillation; ASD, atrial septal defect; AVB, atrioventricular block; RBBB, right bundle branch block; SVT, supraventricular tachycardia.

Mean and maximum HR significantly increased in the first month after the procedure as recorded on 24-h ECG monitoring (mean: before, 76.4±15.8 beats/min vs. after, 95.1±13.7 beats/min).

Table 2. Changes in HR/24 h After ASD Closure

<table>
<thead>
<tr>
<th>Changes in HR/24 h</th>
<th>Mean HR/24 h</th>
<th>Maximum HR/24 h</th>
<th>Minimum HR/24 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before closure</td>
<td>76.4±15.8 (50–98)</td>
<td>129.2±34 (110–195)</td>
<td>50.4±16 (37–61)</td>
</tr>
<tr>
<td>At 1 month</td>
<td>95.1±13.7 (45–93)</td>
<td>162.7±31 (109–198)</td>
<td>50.7±11.7 (40–70)</td>
</tr>
<tr>
<td>At 6 months</td>
<td>79.2±16.9 (50–92)</td>
<td>146±30.1 (100–175)</td>
<td>52.9±11.9 (47–61)</td>
</tr>
<tr>
<td>At 12 months</td>
<td>75.8±21 (55–89)</td>
<td>132.5±39 (105–175)</td>
<td>51.9±11.6 (47–63)</td>
</tr>
</tbody>
</table>

P before vs. 1 month | P<0.05 | P<0.001 | NS   |
P before vs. 6 months | NS     | NS     | NS   |
P before vs. 12 months | NS   | NS     | NS   |

Data given as mean±SD (min–max). ASD, atrial septal defect; HR, heart rate.

Table 3. Change in No. SVES/24 h After ASD Closure

<table>
<thead>
<tr>
<th>Change in No. SVES/24 h</th>
<th>SVES/24 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before closure</td>
<td>204</td>
</tr>
<tr>
<td>1 month</td>
<td>4,347**</td>
</tr>
<tr>
<td>6 months</td>
<td>951</td>
</tr>
<tr>
<td>12 months</td>
<td>211</td>
</tr>
</tbody>
</table>

**P=0.0032. ASD, atrial septal defect; SVES, supraventricular extrasystole.

Table 4. Change in No. VES/24 h After ASD Closure

<table>
<thead>
<tr>
<th>Change in No. VES/24 h</th>
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<tbody>
<tr>
<td>Before closure</td>
<td>176</td>
</tr>
<tr>
<td>1 month</td>
<td>196</td>
</tr>
<tr>
<td>6 months</td>
<td>286**</td>
</tr>
<tr>
<td>12 months</td>
<td>182</td>
</tr>
</tbody>
</table>

**P=0.0095. ASD, atrial septal defect; VES, ventricular extrasystole.

In some patients we found that isolated supraventricular ectopic beats initiated AF or supraventricular tachyarrhythmia (Figures 1, 2).

In 8 patients (3.4%) a transient first-degree AVB was noted with SR an episode of AF occurred in the first hour after the procedure and was successfully terminated by electrical cardioversion.

Data as given n (%). *P=0.0021. AF, atrial fibrillation; ASD, atrial septal defect; AVB, atrioventricular block; RBBB, right bundle branch block; SVT, supraventricular tachycardia.
13.7 beats/min, P<0.05; maximum: before, 129.2±34 beats/min vs. after, 162.7±31.7 beats/min, P<0.001). Variations in mean, minimum and maximum HR are given in Table 2.

As well as changes in HR, the prevalence of paroxysmal supraventricular rhythm disorders also increased in the first month after the procedure. SVES were recorded in 80 patients (34%) before and in 165 (70.2%) 1 month after ASO implantation (P=0.0019). Similarly, transient SVT were seen in 23 (9.8%) prior to and in 51 (21.7%) after the procedure (P=0.0021).

Table 3 lists changes in the number of SVES during the observation period. The mean number of SVES recorded on 24-h ECG monitoring increased in the first month compared to before ASD closure (204±210 vs. 4,347±4,876, respectively, P<0.0001). The number of SVES then gradually decreased. At 6 months the mean number of SVES was 951±2,065 and at 1 year it was no different to that before the procedure (204±210 vs. 211±220, respectively, P=NS).

There were 45 patients (19.1%) who had rhythm disorders in the follow-up period, requiring verapamil to relieve symptoms. The therapy was only temporary, and the rhythm disorders were successfully terminated after 6 months at the latest (mean, 2.6±1.3 months; range, 0.5–6 months).

In contrast, no significant shift in the mean number of VES before and 1 month after ASD closure was found in the 24-h ECG recording (176±199; range, 1–746 vs. 196±211; range, 0–905), respectively, P=NS; Table 4. Six months after ASD closure the number of VES increased to 286±553 (range, 1–4,024; P<0.001), and at 1 year the number of VES decreased and was similar to that before the procedure (182±203 vs. 176±199, respectively, P=NS).

In order to investigate a possible impact of the procedure on rhythm disturbances, 29 clinical, hemodynamic and procedural indices were analyzed. A significant correlation between the number of SVES 1 month after the procedure and fluoroscopy time was identified (r=0.75691; P<0.001; Figure 3). A further correlation between the number of SVES and implanted device size was found (r=0.71855; P<0.001; Figure 4). Canonical correlation analysis showed that the following parameters had an influence on increased SVES 1 month after the procedure: procedure time, fluoroscopy time and device size (P<0.05).

On multiple forward stepwise regression analysis, of all the echocardiography and procedural parameters, only device size (F(7.20)=9.1171; P<0.001, standard error, 2.110) and fluoroscopy time (F(6.11)=9.0370; P<0.001, standard error 2.210) had an influence on the increase in SVES seen 1 month after the procedure. No significant relationships were noted between rhythm disorders and patient age, size of the right atrium and ventricle, size of left atrium, size of the defect or significance of the left-to-right shunt.

**Discussion**

Percutaneous closure of interatrial septal defects has become a standard therapeutic approach in the last few years. Correc-
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Persistence of arrhythmia despite closure of the defect may therefore be the effect of permanent remodeling of the atria exposed to the long-lasting increased pressure. 26,27 In the present study the persistence of arrhythmia may also be related to the very short follow-up period relative to the long antecedent period before closure.

Data evaluating the impact of ASD closure with an Amplatzer device on rhythm and conduction disorders are scarce. The most commonly described disturbances are supraventricular arrhythmias seen during the closure procedure. Transient AVB or ST segment elevation, which are associated with air microembolization in coronary arteries, are observed less frequently. In the present study ASD closure was associated with a small risk of AV conduction abnormalities, in 8 patients (3.4%) transient first-degree AVB was detected in the first hour after the procedure.

In the present study there was a transient increase in the mean and maximum HR 1 month after the procedure. 24,25 In the present study supraventricular tachyarrhythmias, such as paroxysmal AF and SVT, were observed in the preoperative period in most patients, and persistent AF in 25 (10.6%). One year after the procedure, the number and type of supraventricular tachyarrhythmias did not differ significantly as compared with the baseline data.

Numerous experimental and clinical studies show that AF in patients with CHD, including intracardiac shunts, is caused by the ongoing remodeling of the atrial walls. This leads to change of the atrial size and shape, but also results in disproportionate of muscle and connective tissue size and structure, impairment of cardiomyocyte electrical features and metabolism. Persistence of arrhythmia despite closure of the defect may therefore be the effect of permanent remodeling of the atria exposed to the long-lasting increased pressure. 26,27 In the present study the persistence of arrhythmia may also be related to the very short follow-up period relative to the long antecedent period before closure.

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In the present study there was a transient increase in the mean and maximum HR 1 month after the correction, which was not seen at 6 or 12 months. Similarly, the mean number of SVES on Holter ECG increased at 1 month after defect closure and subsequently decreased at 6 months; and 12 months later there was no difference compared with baseline. Probably the changes in mean/maximum HR were accounted for by the increase in SVES. In contrast, there were no apparent changes in the number of ventricular arrhythmias 1 month after device implantation.

Hill et al, who analyzed Holter ECG recordings of 41 patients before and after percutaneous ASD II closure, drew sim-
ilar conclusions.26 They observed a significant increase in supraventricular tachyarrhythmia without changes in VES.

Exacerbation of supraventricular arrhythmia after defect closure may be related to the procedure itself. An invasive character of the intervention, instrumentalization and device implantation may provoke rhythm disturbances. Exacerbation of supraventricular arrhythmias after defect closure may trigger pathological automatism.

Injury and scars from device implantation or replacement fibrosis may also provide a substrate for reentry. The dense fibrosis creates conduction block that can define borders of reentry circuits. Fibrosis also contributes to the creation of slow conduction that is necessary for reentry.

At the microscopic level, fibrosis separates myocyte bundles, forcing the excitation wave front to take a circuitous course through the bundles. In addition, conductivity between cells can be diminished. This uncoupling of myocyte bundles, and to some extent the myocytes within the bundles, slows conduction, although action potentials and ion channels in the myocytes can be relatively normal. Slow conduction and fibrous anatomic barriers set the stage for reentry.

We showed a correlation between the number of SVES in the first month and fluoroscopy time. Fluoroscopy time may be regarded as an index of procedure complexity. Any difficulties during the intervention will prolong fluoroscopy time.

The more time that is needed for appropriate implantation of the device, the more harm may be done to the atrial wall and septum, which may evoke arrhythmia. An additional correlation between Amplatzer device size and number of SVES was also found. The size of the device, similarly to fluoroscopy time, may predict difficulties in the intervention. In contrast, a greater weight and size of the device solely may have an impact on atrial walls and provoke supraventricular rhythm disorders.

Transient supraventricular arrhythmia was also reported after surgical ASD correction.22,23,27 Karwot et al compared the Holter ECG results of 91 patients before and after either surgical or percutaneous intervention.28 They found that during 2.5–5.5 years of observation, supraventricular arrhythmia was more frequent after surgical than percutaneous intervention (35% vs. 2.1%, respectively; P<0.05).28

In the present study, supraventricular arrhythmia occurring de novo after device implantation was transient and resolved over 1 year. The present results are consistent with previously published data.29–33 None of the rhythm disorders recorded in the present patients had an impact on postoperative period. No additional hospitalizations or other clinical incidences were associated with arrhythmias.

In some patients, we found that isolated supraventricular ectopic beats initiated AF or supraventricular tachyarrhythmia. Mazzanti et al demonstrated that either long- or short-coupled extrasystolic beats initiated ventricular fibrillation,31 hence it is interesting that this happened for AF as well. Thus temporary use of anti-arrhythmia agents may be necessary after ASD closure to prevent AF.

In the present group 45 patients (19.1%) with new-onset rhythm abnormalities in the first month had to be given anti-arrhythmia medication (calcium channel blocker), which was discontinued after rhythm normalization at 2.6±1.3 months (range, 0.5–6 months) on average.

It is therefore advisable to conduct a thorough follow-up after ASD II closure, including ECG monitoring, especially in the early post-procedural period.

Conclusions
Transcatheter closure of secundum ASD is associated with a transient increase in supraventricular premature beats and a small risk of AV conduction abnormalities and paroxysmal AF in early follow-up. Transcatheter closure of secundum ASD does not reduce arrhythmia that appears prior to ASD II closure. Larger device size and longer procedure time are associated with increased risk of supraventricular arrhythmia on early follow-up.

Disclosures
Name of Grant: No grants obtained for the study.

References


