Atrial Septal Aneurysms

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Redundant septum primum → Saccular deformity (part or all of IAS) → protrusion into RA or LA or both.

generally at the level of the fossa ovalis
Phasic septal excursion of ≥ 10 - 15 mm during the cardio-respiratory cycle.

Base of aneurysm = 15 mm

Prevalence

ASA was initially thought to be a rare congenital abnormality, with only about 100 cases had been reported before 1985.

However, with the advent of 2D echo and the widespread use of TEE it became more frequently identified.
• TEE studies: 2 - 10%.
  Pediatric population: 0.9 - 1.7% in Children
  4.9% in infants.

• Olivares-Reyes A et al 2000 reported 500 adults out of
  22,224 patients over a period of 6 1/2 years

• 40% were diagnosed by TEE.

• Prevalence = 2.2%

• Mean age of 65 Y

• F:M ratio = 317 : 183 (63 : 37%).

• 7.9 - 15% of stroke patients (by TEE).

• Commonly associated with PFO:
  33% of patients with ASA also have PFO
  32% had isolated ASA.
Diagnostic criteria

1. Saccular deformity of IAS or the foramen ovale region.

2. Excursion $> 10$ mm into RA or LA or
   Sum of bilateral excursions of $> 10$ mm.

3. Aneurysmal base width $> 15$ mm.

Echo Classifications

Olivares-Reyes A, et al.

Longhini C, et al.

Henley PC, et al.
JACC 1985;6:1370-82.

| Type 1 | Diastole: deviation into RA  
|        | Early Systole: bulging into LA  
|        | Mid-Systole: rightward crossing-over  
|        | + during inspiration or expiration |
| Type 2 | Expiration: Sustained rightward deviation  
|        | Inspiration: Leftward motion only during inspiration in early ventricular systole |
| Type 3 | Remains in RA  
|        | with undulating motion during all phases of the cardio-respiratory cycle |


| Type 1A | The bulging in RA is motionless. |
| Type 1B | The bulging is confined to RA but with rapid phasic oscillation during inspiration |
| Type 2  | The ASA protrudes maximally into LA & is accompanied by excursion into RA |
Pearson Modification


<table>
<thead>
<tr>
<th>Type 1A</th>
<th>Constant protrusion towards RA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1B</td>
<td>Protrusion predominantly to RA with movement towards LA in systole</td>
</tr>
<tr>
<td>Type 1C</td>
<td>Protrusion towards LA with Valsalva</td>
</tr>
<tr>
<td>Type 2C</td>
<td>Fixed protrusion towards the left atrium</td>
</tr>
</tbody>
</table>


1. To which Chamber: RA or LA

2. Mobility:
   - Fixed: only bulges within 1 atrium.
   - Mobile: bulges bi-directionally into both atria.
<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1R</td>
<td>Protrudes from midline to RA throughout the cardio-respiratory cycle.</td>
</tr>
<tr>
<td>2L</td>
<td>Protrudes from midline to LA throughout the cardio-respiratory cycle.</td>
</tr>
<tr>
<td>Fixed</td>
<td></td>
</tr>
<tr>
<td>3RL</td>
<td>Maximal excursion is towards RA with a lesser excursion towards LA.</td>
</tr>
<tr>
<td>Mobile</td>
<td></td>
</tr>
<tr>
<td>4LR</td>
<td>Maximal excursion is towards LA with a lesser excursion towards RA.</td>
</tr>
<tr>
<td>Mobile</td>
<td></td>
</tr>
</tbody>
</table>

Movement is bidirectional & equidistant to the RA & LA during the cardio-respiratory cycle.

MOBILE
Clinical Associations

1. Congenital:
   PFO  ASD  VSD  PDA  valve prolapse  
   Ebstein  T atresia  P atresia

2. Acquired:
   Valvar  CM  IHD  
   HTN (S & P)  Arrhythmias.

3. CV events of embolic origin:
   TIA - CV accidents.

4. Isolated
Echo Associations

<table>
<thead>
<tr>
<th>Association</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>PFO</td>
<td>36%</td>
</tr>
<tr>
<td>LA enlargement</td>
<td>31%</td>
</tr>
<tr>
<td>RA enlargement</td>
<td>11%</td>
</tr>
<tr>
<td>LV enlargement</td>
<td>10%</td>
</tr>
<tr>
<td>RV enlargement</td>
<td>8%</td>
</tr>
<tr>
<td>Valve Prolapse</td>
<td>13%</td>
</tr>
<tr>
<td>Thrombi</td>
<td>1.2%</td>
</tr>
</tbody>
</table>

ECG

Scholz et al reported ECG signs of RA enlargement in a routine ECG of a 39 Y woman with no history of cardiac disease + normal measurements of LA & both ventricles + normal PA pressure (echo).

They suggested that the RA enlargement might be due to ectopic RA rhythm caused by the sacculation of ASA.

Clinically

1. Asymptomatic
2. Cryptogenic stroke.
3. The platypnea-orthodeoxia syndrome.

Platypnea-Orthodeoxia Syndrome

**Platypnoea:**
Dyspnoea induced by upright posture and relieved by recumbency

**Orthodeoxia:**
Arterial deoxygenation accentuated by upright position and improved by recumbency.

Rare
A known complication after lung surgery (months – 1 Y)
Other Cardiac causes:
- Pericardial effusion - Constrictive Pericarditis
- Drug toxicity as Amiodarone.
To create this syndrome, 2 components must coexist:

1. **Anatomic component**: An inter-atrial shunt: (e.g. ASD – PFO - fenestrated ASA).

2. **Functional component**: Deformity induced in the atrial level while rising to an upright from a recumbent position.

**Mechanisms:**
- Key to this syndrome is ↑ RA pressure → R-L atrial shunt.

1. Altered compliance of the right heart in upright position → ↑ RA pressure → R-L shunt through an ASD.

2. Anatomical alterations of RA (e.g. distortion of fossa ovalis & IVC) → change in the direction of blood flow into RA.

3. Mechanical compression by a hydrothorax → ↑ extrinsic pressure on RA

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**Stroke**

ASA may act as facilitator for paradoxical embolism via:

1) May ↑ the PFO diameter due to the highly mobile atrial septal tissue → wider opening of an otherwise small channel

2) May promote R-L by redirecting flow from IVC toward PFO

3) May act as a nidus for local thrombus formation
### Risk for cardiogenic CV embolism

<table>
<thead>
<tr>
<th></th>
<th>All</th>
<th>CV stroke</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>561</td>
<td>245</td>
<td>316</td>
</tr>
<tr>
<td>ASA by TTE</td>
<td>61 (10.8%)</td>
<td>45 (18.3%)</td>
<td>16 (5.1%)</td>
</tr>
<tr>
<td>ASA by TEE</td>
<td>104 (18.5%)</td>
<td>68 (27.7%)</td>
<td>36 (9.9%)</td>
</tr>
<tr>
<td>PFO</td>
<td>87 (15.5%)</td>
<td>56 (22.8%)</td>
<td>31 (9.8%)</td>
</tr>
<tr>
<td>Atrial Shunt</td>
<td>123 (21.9%)</td>
<td>60 (24.4%)</td>
<td>63 (17.4%)</td>
</tr>
</tbody>
</table>

### Management

1. Anti-coagulants / Anti-platelets:
   
   a) Warfarin > Aspirin → ↓ recurrant risk of stroke.

   b) Aspirin is effective in patients with no previous cerebral events.

2. Mechanical closure.