Impact of Pacing on Systemic Ventricular Function in L-Transposition of the Great Arteries

Sophie C. Hofferberth, MBBS, Mark E. Alexander, MD, Douglas Y. Mah, MD, Victor Bautista-Hernandez, MD, Pedro J. del Nido, MD, Francis Fynn-Thompson, MD

Boston Children’s Hospital, Harvard Medical School

AATS 2015
Disclosures

• None of the authors in this study have any commercial relationships to disclose
Congenitally Corrected Transposition of the Great Arteries (ccTGA)

- ccTGA is a complex cardiac anomaly defined by atrioventricular (AV) and ventriculoarterial discordance

- Native ccTGA physiology leaves morphological RV to support systemic circulation
  - Late onset RV dysfunction
  - Systemic AV regurgitation

- Surgical management has evolved to anatomical repair
  - Excellent perioperative/intermediate outcomes
  - late systemic ventricular dysfunction remains significant problem\(^1,2\)

Pacing and Systemic Ventricular Dysfunction

- ccTGA has high incidence of spontaneous and procedure-related heart block

- Pacemaker insertion associated with late-onset systemic ventricular dysfunction post anatomical repair\(^1,2\)

- Univentricular (single site) pacing shown to be significant risk factor for ventricular dysfunction

- Biventricular pacing (BiVP) appears to preserve systemic ventricular function

Methods

Aim

• Compare the impact of univentricular versus biventricular pacing on systemic ventricular function in all pts with ccTGA

Methods

• Retrospective analysis of all pts with diagnosis of ccTGA who received pacemakers between 1993 and 2014

• Study endpoints
  – Timing and indication for pacemaker insertion
  – Location of ventricular lead(s)
  – Qualitative ventricular function
  – Clinical outcomes
# Baseline Patient Characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Number (%) or Median (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total number of pts</td>
<td>53</td>
</tr>
<tr>
<td>Male/Female</td>
<td>31/22</td>
</tr>
<tr>
<td>Median age at initial pacer insertion</td>
<td>1.8 years (6 days - 42 years)</td>
</tr>
<tr>
<td>Median weight, Kg</td>
<td>10.8 (3.5 to 69.0)</td>
</tr>
<tr>
<td>Segmental anatomy</td>
<td></td>
</tr>
<tr>
<td>S,L,L</td>
<td>49 (92)</td>
</tr>
<tr>
<td>I,D,D</td>
<td>4 (8)</td>
</tr>
<tr>
<td>Associated CV anomalies</td>
<td></td>
</tr>
<tr>
<td>VSD</td>
<td>42 (79)</td>
</tr>
<tr>
<td>Pulmonary stenosis</td>
<td>25 (47)</td>
</tr>
<tr>
<td>Pulmonary atresia</td>
<td>10 (19)</td>
</tr>
<tr>
<td>Ebstein-like anomaly</td>
<td>9 (17)</td>
</tr>
<tr>
<td>Single coronary artery</td>
<td>3 (6)</td>
</tr>
</tbody>
</table>
Pacing Details

Primary univentricular pacing

• 42 pts

  - No further pacing intervention
    N = 25
  - Upgraded to BiV pacing
    N = 17

• Epicardial lead location
  - Anatomic LV, n = 28
  - Anatomic RV, n = 11
  - Ambiguous data, n = 3

Primary biventricular pacing

• 11 pts
Timing of Initial Pacemaker Insertion

• Spontaneous heart block, n = 8

• Time of surgical intervention preparing for anatomical repair, n = 9
  – PA Banding, n = 8
  – BT shunt, n = 1

• Time of anatomical repair, n = 8

• Post anatomical repair, n = 28
Outcomes: Primary Univentricular Pacing

- Initial univentricular pacer
  - Systemic ventricular dysfunction: N = 22
    - Upgrade to BiVP: N = 14
      - Persistent systemic ventricular dysfunction: N = 7
        - 1 in-hospital death (heart failure)
        - 1 late death (heart failure)
        - 1 transplant
      - Improved systemic ventricular function: N = 7
        - No further pacing intervention
    - No further pacing intervention: N = 8
  - No ventricular dysfunction: N = 20
    - Upgrade to BiVP: N = 3
    - No further pacing intervention: N = 17
      - 2 late deaths (heart failure)
In initial univentricular pacing, N = 42

Systemic ventricular dysfunction, N = 22

- Lead placement on anatomic LV, N = 13
  - Sub-systemic lead, N = 6
  - Sub-pulmonary lead, N = 7
- Lead placement on anatomic RV, N = 9
  - Sub-systemic lead, N = 4
  - Sub-pulmonary lead, N = 5

No difference in risk of systemic ventricular dysfunction based on initial lead location whether analyzed by ventricular morphology or physiologic function.
Outcomes: Primary Biventricular Pacing

• N = 11
• Median age = 1.2 yrs (range, 5 mths to 16 yrs)
• Median weight = 10.9 kg (range, 5 to 59 kg)
• 6 male, 5 female
• No perioperative complications
• No major CV complications
   – (i.e. no mortality/heart failure/transplantation)

None of the pts treated with primary biventricular pacing developed systemic ventricular dysfunction at latest follow up
Univentricular versus Biventricular Pacing
Late Follow-up

- Overall median follow up = 3.7 yrs (range, 4 days - 22.5 yrs)

- **Primary biventricular pacing (n = 11)**
  - Median follow up = 3 yrs (range, 4 d - 9 yrs)

- **Primary univentricular pacing (n = 42)**
  - 25 pts: no further pacing intervention
    - Median F/U = 4.2 yrs (range, 4 d - 16 yrs)
  - 17 pts: upgraded to BiVP
    - Median time from initial univentricular pacer insertion to BiVP upgrade = 3 yrs (range, 3 mths - 22 yrs)
  - Median time from upgrade to demonstrated improvement in ventricular function (6 pts) = 8 wks (range, 2 – 20 wks)
Limitations

• Retrospective, single-center analysis

• Extended follow-up in univentricular pacing cohort may display natural history of systemic RV in addition to consequences of single site pacing

• Variable location of biventricular leads driven by uncertainty over exact site of latest electrical activation and limited surgical access

• Some pts upgraded to biventricular pacing at time of further anatomic correction

• Subjective classification of ventricular function in paced ventricles
Conclusions

• ccTGA pts have a high frequency of both AV block and late ventricular dysfunction

• While ventricular pacing is clearly required, single site ventricular pacing appears to contribute to earlier ventricular dysfunction
  – Neither morphologic nor physiologic consideration of single lead placement predicts late ventricular dysfunction

• Upgrade to BiVP offers a reasonable (50%) chance of short term normalization of ventricular function

• Primary BiVP prevents (or at least delays) development of ventricular dysfunction
Thank you