Can We Stop Anticoagulation After LAAO?

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Development of LAA

5 Weeks

- Pulmonary veins
- Primordial pulmonary vein
- Primordial left atrium

8 Weeks

- Part of left atrium formed from absorbed pulmonary vein tissue
- Entrance of four pulmonary veins
- Smooth-walled part of left atrium
- Left auricle

Hara et al. CCI.2009;74:234–242
Various Shapes of LAA

cactus   cauliflower   chicken wing   windsock
220 Post-mortem analysis (106 male, 114 female)
Age: 72 ± 13 years
Rhythm: Sinus rhythm 143, AF 55
Volume: 770~19,270 mm³ (5,220 ± 3,041 mm³)
Angulation: > 100° (42%)
Narrow diameter: 5~27 mm
Wide diameter: 10~40 mm
Length: 16~51 mm
AF vs. Sinus rhythm: 7060 mm³ vs. 4645 mm³ (P < 0.01)
LAA During AF

Most thrombi (90%) in NVAF develop in LAA

Courtesy of Prof. J Hong
Depending on individual circumstances,

LAA Occlusion
Major Indications in EU

![Graph showing major indications for LAA Occlusion in EU](chart.png)

- 94% of patients
- CHA2DS2-VASc ≥ 2 but contraindication to OAC
- CHA2DS2-VASc ≥ 2 and HAS-BLED ≥ 3
- Embolic events despite OAC
- CHA2DS2-VASc ≥ 2 and end-stage renal failure
- CHA2DS2-VASc ≥ 2 and triple anticoagulant therapy
- CHA2DS2-VASc ≥ 2 and PVI

OAC vs. APT?
Anti-Thrombotic Tx after LAAO

• GAP between previous research and real world practice
  - Study subjects: eligible for OAC (OAC vs. LAAO)

In Real world: contraindication to OAC

• Unanswered question
  - Defining optimal DURATION of OAC/APT
Two Determinants?

Complete Sealing

Endothelialization
Peri-device Leakage < 5 mm

- In Protect AF trial, 92% achieved this goal

- **Is it sufficient to quit OAC?**

- Clinical outcomes are not different (if, leakage < 5 mm)
Is Peri-device Leakage Important?

정주영 공법

7,686 m 중 마지막 270 m
Endothelialization After LAA Occlusion -PLAATO Device, Canine Model-

Endothelialization After WATCHMAN In Canine Model

Implanted device

45 days

Complete endothelialization
Healing Process of Implanted Device

- Explanted ASD or PFO closure device (n = 9)
- Mean interval: 3.4 ± 2.4 years (0.9 – 8.3 years)
- Recurrent thromboembolic events (n = 5)
- Residual shunt/dislocation (n = 3)
- Growing mass on device (n = 1) – hyperplastic tissue formation

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Vogt MO et al. *Int J Cardiol.* 2011; 147: 398-404
Explanted ASD or PFO Devices

Vogt MO et al. *Int J Cardiol.* 2011; 147: 398-404
Hyperplastic Tissue / Thrombi
Another Determinant?

• Absolute timing (6 weeks)?
• Procedural outcome
  - Peri-device leakage
  - Endothelialization
• Blood stasis (HF worsening)

➤ **Total Thrombogenicity?**

• No General Rule!
Endothelialization vs. Stasis?

Indication for LAAO
- Recurrent stroke & major bleeding during OAC
- Successful LAAO
- 2 M & 12 M f/u TEE
  → Complete sealing w/o thrombi during single APT

Endothelialization completion?

At 18 M after LAAO,
- HF exacerbation during stress cardiomyopathy
- LVEF 65% → 28%
- Thrombi on disc surface
- OAC and HF management

At 20 M after LAAO,
(OAC for 2 M & HF Mx)
LVEF 28% → 55%
Thrombi disappeared
OAC → APT
No further thrombotic events up to 36 M after LAAO
Thrombogenicity After Closure

- PFO closure after cryptogenic stroke (n = 24)
- Measure thrombogenicity (prothrombin fragment 1 + 2, TAT III) & platelet activity (P-selectin, CD40)

Enhanced Thrombogenicity – return to baseline 90 days later

Platelet activation – Not significantly changed!

Levels of the markers of coagulation and platelet activation at baseline and at 7, 30, and 90 days after transcatheter closure of PFO. A, B, C, D.

Bedard et al. Stroke 2007; 38: 100-104
PLAATO achieved an adequate seal of the neck of LAA w/o significant effect on the structure of the LA and LUPV.
LAA Exclusion Can Worsen HF

- 47 LAAO vs. 141 Non-LAAO
- Retrospective Difference-in-Different analysis for hemodynamic changes before and after LAAO

<table>
<thead>
<tr>
<th>Variables</th>
<th>LAAC group (n=47)</th>
<th>Non-LAAC group (n=141)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>75.1 ± 9.9</td>
<td>74.7 ± 9.7</td>
<td>0.799</td>
</tr>
<tr>
<td>Male (n, %)</td>
<td>20 (42.6%)</td>
<td>60 (42.6%)</td>
<td>1.000</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>24.4 ± 3.8</td>
<td>24.3 ± 3.7</td>
<td>0.953</td>
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<tr>
<td>Paroxysmal AF (n, %)</td>
<td>13 (27.7%)</td>
<td>34 (24.1%)</td>
<td>0.627</td>
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<tr>
<td>HAS-BLED score</td>
<td>2.55 ± 1.41</td>
<td>2.23 ± 1.34</td>
<td>0.156</td>
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<tr>
<td>CHA₂DS₂-VASc score</td>
<td>3.83 ± 1.98</td>
<td>3.32 ± 1.65</td>
<td>0.082</td>
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<tr>
<td>Labile INR (n, %)</td>
<td>6 (12.8%)</td>
<td>20 (14.2%)</td>
<td>0.807</td>
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<tr>
<td>Bleeding (n, %)</td>
<td>8 (17.0%)</td>
<td>23 (16.3%)</td>
<td>0.910</td>
</tr>
<tr>
<td>Stroke or TIA (n, %)</td>
<td>21 (44.7%)</td>
<td>49 (34.8%)</td>
<td>0.223</td>
</tr>
<tr>
<td>Hypertension (n, %)</td>
<td>32 (68.1%)</td>
<td>92 (65.2%)</td>
<td>0.722</td>
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<tr>
<td>Diabetes mellitus (n, %)</td>
<td>6 (12.8%)</td>
<td>21 (14.9%)</td>
<td>0.719</td>
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<td>Congestive heart failure (n, %)</td>
<td>14 (29.8%)</td>
<td>35 (24.8%)</td>
<td>0.502</td>
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<td>Chronic kidney disease (n, %)</td>
<td>5 (10.6%)</td>
<td>13 (9.2%)</td>
<td>0.775</td>
</tr>
<tr>
<td>Vascular diseases (n, %)</td>
<td>7 (14.9%)</td>
<td>14 (9.9%)</td>
<td>0.349</td>
</tr>
</tbody>
</table>

**Abbreviations:** TEE: transesophageal echocardiography; LAAC: left atrial appendage closure; BMI: body mass index; INR: international normalized ratio; TIA: transient ischemic attack; AF: atrial fibrillation; bpm: beats per minute; EF: left ventricular ejection fraction;
E/E’ Elevation After LAAO

\[ p = 0.028 \]
\[ \text{DID} = +2.25 \]
More LA Remodeling After LAAO

\[ p = 0.011 \]
\[ \text{DID} = +5.85 \]
The Smaller LA, The Worse

- Smaller LA will experience the worse deterioration of the diastolic function after LAAO
- Patients with paroxysmal AF without LA enlargement (less remodeled LA) are more likely to experience the worsening of heart failure after LAAO

Phan QT et al. Preparing Submission
Acceptable Endpoint

- Stable anchoring of device
- Complete sealing w/o leakage

Minimize or eliminate Dead Space → Better (?)

Generally, acceptable!
CONCLUSIONS

Individualized anti-thrombotic strategy depending on patients’ condition

1. Completeness of sealing
2. Endothelialization
3. Hemodynamic state (HF exacerbation, etc.)
4. Individual patient’s response to medications (seeking a new parameter to assess integrated thrombogenicity)
Thank you for Your Attention

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