Ablation of Paroxysmal Atrial Fibrillation from PV and Non-PV Triggers

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The majority of paroxysmal atrial fibrillation are originated from the thoracic veins. They are the major source of trigger as well as driver mechanism for atrial fibrillation especially in structurally normal heart or non atrial myopathic pts.

Often clinically manifest as paroxysmal form of atrial fibrillation and PV’s are still the dominant source of atrial fibrillation.

Non-PV Atrial fibrillation have been reported and estimated incidence range 8% to 15%.

Frequent PAC’s only can serve as trigger therefore, they require associated underlying electrophysiological alterations usually pathological changes from atrial myopathy that facilitate for the initiation of atrial fibrillation.

The degree of atrial remodeling has close relationship with the threshold of trigger can initiate atrial fibrillation activities.
PV trigger mapping

Episode 1

Episode 6
HDM: focal trigger from LPV carina

Orion catheter was placed at the carina between upper and lower LPVs
PV trigger EGM: Local trigger and wave break
Trigger beats has characteristics of far field signal
LPV trigger from carina and LPV activations

Trigger beats (1-4)  

beat #5
Focal trigger RSPV Sub-carina region
Findings

• Initiating trigger activities persists for 3 to 4 beats with similar initiating focal activities with varying coupling intervals until the wave break followed.

• All triggers were originated sub-carina

• The earliest EGM showed characteristics of far-field (epicardial?)

• The segmental ablation of trigger area

• The average number of ablations were 3.9 around subcarina
Focal trigger Ablation in Non-atrial myopathic (>1.5 mV) Afib

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<thead>
<tr>
<th></th>
<th>L PVs</th>
<th>RPVs</th>
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<tbody>
<tr>
<td>Total #: 12 pts</td>
<td>8 M; 4 F</td>
<td>7 (LPVT-2, LSPV-4, LIPV – 1)</td>
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<tr>
<td>Age</td>
<td>51.5 ± 7.9 yrs</td>
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<tr>
<td>LAV ( MRA)</td>
<td>53.1 ± 4.4 ml</td>
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<tr>
<td>Ablations</td>
<td>4.1 ± .9</td>
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<td>Follow-up</td>
<td>10.3 ± 3.1 months</td>
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<tr>
<td>Recurrences</td>
<td>1/12</td>
<td>1 (LPV T)</td>
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<tr>
<td>PV stenosis</td>
<td>0</td>
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RSPV and antral activation during CS pacing (posterior view) and focal trigger from RSPV sub carina
Extensive area of scar and low voltage area (atrial myopathy based on voltage < 1.5 mV)
ERAF 1:
Spontaneous ERAF
Mapping trigger with Orion (in RSPV)
Orion in SVC
SVC focal atrial fibrillation mapping
Ablation responses (25 W)
What do we learn from HDM of Afib from SVC cases?

• A total experience of 15 cases (11 cases non-HD mapping and 4 cases of HD mapping) since 2010.

• 13 cases non-HD mapping underwent SVC venogram/Lasso catheter guided distal segment isolation and 4 cases HD mapping underwent focal ablation

• No recurrences

• Important to make good differential diagnoses and mapping work-up (RSPV vs SVC or RSPV-SVC connections)

• The data suggest that needs to question guideline recommended and commonly practiced SVC isolation that can be associated with significant complications
Crista trigger/tachycardia induced A fib in myopathic A fib
Septal and MV annulus triggers in Afib with A myopathy pts
Summaries

• Limited early experiences showed that the HD mapping can identify the focal triggers from PV and non-PV and provide some insight mechanisms that allow us to target the focus only for the elimination of Afib.

• The strategy of focal ablation of trigger source in selected non-atrial myopathic atrial fibrillation pts is effective and resulted significant reduction in ablation lesions.

• Despite many limitations, early result is encouraging and suggesting that empiric PV and/or SVC isolations in non-atrial myopathic A fib pts should be reconsidered.
Thank you for your attention