3 (0.67 ± 0.26 ng/dl) than that in Gr 1 (2.15 ± 0.17 ng/dl, p < 0.001) and 2 (2.26 ± 0.04 ng/dl, p < 0.001), respectively. There were no protein expression differences of the calcium handling proteins (CaV1.2, NCX, RyR and SERCA2), Kv7.1, Kir2.1 and Nav1.5 among groups, respectively (Fig C). There was a significant elevations of tyrosine hydroxylase nerve innervation, but not choline acetyltransferase, in Gr 2, compared to Gr 1 and 3 (Fig. D).

**Conclusion:** RDN is capable of suppressing ventricular arrhythmias induced by OSA through autonomic reverse remodeling with decreasing sympathetic overactivity and catecholamine spillover, protecting from the risks of life-threatening arrhythmia and sudden cardiac death.

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**ABSTRACT CE-542:**
Getting an eye on the target before the ablation of SVT
Sunday, May 1, 2022
8:00 AM - 9:00 AM
CE-542-01
**PRECORDIAL ‘REVERSE PATTERN BREAK’: A NOVEL PREDICTOR FOR EPICARDIAL POSTEROSEPTAL ACCESSORY PATHWAYS**
Debabrata Bera MD

**Background:** Posteroseptal (PS, also known as inferior paraseptal) accessory pathways (AP) may occasionally lie within the coronary sinus (CS), its tributaries, in a CS diverticulum (CSD) or along the epicardial surface. We encountered precordial reverse pattern break (RPB) of QRS morphology in some patients with epicardial APs and analysed all ECGs of posteroseptal accessory pathway (PSAP) cases.

**Objective:** We hypothesise that workload response of mitochondria is impaired in atrial cardiomyocytes from patients with AF.

**Methods:** Membrane currents (patch clamp), cytosolic Ca²⁺ (Fluo-3) and NAD(P)H/FAD autofluorescence were recorded in right atrial myocytes from sinus rhythm (CTL) or AF patients. Immunofluorescence labeling together with STED microscopy and EM microscopy were employed to characterize interaction between mitochondria and sarcoplasmic reticulum (SR).

**Results:** Diastolic [Ca²⁺]i of voltage-clamped myocytes was comparable, whereas amplitudes of L-type Ca²⁺ current and triggered of Ca²⁺ transients (CaTs) were decreased by 49% and 43%, respectively. Basal level of NAD(P)H at 0.5 Hz stimulation frequency was comparable in CTL and AF (73.08 ± 2.62%, n = 14/7 vs. 81.05 ± 5.39%, n = 4/3 respectively), suggesting a similar redox index. In all CTL cells, upon increasing stimulation frequency to 3 Hz and subsequent β-adrenergic stimulation, the NAD(P)H level initially decreased but recovered to a level comparable to basal state. In contrast, 35% of AF myocytes lost this capacity to recover. Electron and STED microscopy images (Figure) show a disturbance in mitochondrial organisation and their interaction with the SR.

**Conclusion:** Impaired cytosolic Ca²⁺ handling and disturbed interaction between mitochondrial and Ca²⁺ release sites of the SR may contribute to the impaired redox response of mitochondria to increased workload which we observed in AF patients. This likely results in impaired ATP production and ROS neutralisation, which may finally contribute to atrial arrhythmogenesis in AF patients.

**Table.** Electrophysiological parameters of both atrium among groups.

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV</td>
<td>140.8 ± 7.8</td>
<td>141.9 ± 9.9</td>
<td>144.3 ± 14.2</td>
</tr>
<tr>
<td>LV</td>
<td>122.2 ± 17.9</td>
<td>119.6 ± 17.5</td>
<td>121.8 ± 18.2</td>
</tr>
</tbody>
</table>

X: 2 times; 10X: 10 times; LV: left ventricle; RV: right ventricle; TH: Threshold.