1091-117

Do Changes in Local Cardiac Electrophysiology Influence Ventricular Fibrillation Cycle Length?

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Prior studies show a high correlation between ventricular fibrillation cycle length (VFCL) and refractory period. Given the changes in local cardiac electrophysiology following infarction, we hypothesized that local VFCL is influenced by local electrophysologic heterogeneity.

Methods: Eighteen dogs-6 normal, 6 each 5 day and 8 week infarct (post-left anterior descending artery ligation) had electrophysiological study. VF was induced by programmed electrical stimulation. Mean VFCL (5-8 seconds after induction) was determined and correlated with effective refractory period (ERP), activation recovery time (ART), ERP/ART (index of post-repolarization refractoriness), late diastolic threshold, ERP and ART dispersion (ERPD,ARTD) determined at 112 sites on the anterior left ventricle.

Results: The VFCL was significantly longer in 5 day (149±35 ms) and 8 week (129±18 ms) infarct dogs than normal dogs (102±15 ms). The correlation coefficient (R²) between global VFCL and electrophysiological properties over the entire plaque was much higher than between local VFCL and EP properties at each site (Table).

Conclusions: VFCL in normal and infarcted myocardium shows a poor correlation with global VFCL and electrophysiological properties over the entire plaque was much higher than between local VFCL and EP properties at each site (Table).

R² of VFCL to electrophysiological properties.

<table>
<thead>
<tr>
<th>Global Local</th>
<th>All Norma</th>
<th>5 day Infant</th>
<th>8 week Infant</th>
<th>All Normal</th>
<th>5 day Infant</th>
<th>8 week Infant</th>
</tr>
</thead>
<tbody>
<tr>
<td>ERP300</td>
<td>0.03</td>
<td>0.11</td>
<td>0.17</td>
<td>0.03</td>
<td>0.09</td>
<td>0.06</td>
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<tr>
<td>ART300</td>
<td>0.01</td>
<td>0.05</td>
<td>0.34</td>
<td>0.01</td>
<td>0.02</td>
<td>0.25</td>
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<td>ERP/ART</td>
<td>0.00</td>
<td>0.28</td>
<td>0.77</td>
<td>0.00</td>
<td>0.04</td>
<td>0.04</td>
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<tr>
<td>Threshold</td>
<td>0.29</td>
<td>0.16</td>
<td>0.04</td>
<td>0.00</td>
<td>0.07</td>
<td>0.23</td>
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<tr>
<td>Maximum ERP</td>
<td>0.38</td>
<td>0.04</td>
<td>0.13</td>
<td>0.37</td>
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<td>0.00</td>
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<td>Maximum ARTD</td>
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<td>0.78</td>
<td>0.03</td>
<td>0.07</td>
<td>0.12</td>
<td>0.11</td>
</tr>
</tbody>
</table>

*p < 0.05 for linear regression

1091-118A

Evidence Against the Efficacy of Current Chest Wall Protection for Sudden Death Due to Premordial Blows (Commotio Cordis)

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Background: Blunt nonpenetrating chest blows causing sudden cardiac death (commotio cordis) continue to be reported in association with organized/recreational competitive sports, usually in healthy young children. These events have raised consideration of whether protective equipment could possibly prevent such catastrophes.

Methods: Using a Commotio Cordis Registry of 124 confirmed cases were surveyed.

Results: Of the 124 events, 105 were associated with various sporting activities, and 25 of these (24%) occurred in young athletes (mean age 14 ± 8; all male) who were equipped in playing with standard, commercially available gear regarded as providing a large measure of protection from the consequences of blows to the chest wall: 1) chest wall barriers with padding comprised largely of closed or open-cell polymer foam covered by fabric or hard shell, in hockey (N = 12), football (N = 5), lacrosse (N = 3) and baseball (N = 2) and, 2) baseball projectiles marketed commercially as reduced injury "safety", or "training" balls comprised largely of rubber of various texture, in 3 children without chest barriers. In hockey (non-goalie) the puck appeared to strike the chest wall directly, either due to the angulation of the shot which circumvented the standard shoulder/chest protector, or because the player instinctively raised his arms to obstruct a slap-shot, causing the chest barrier to migrate cephalad and allowing the puck to impact unprotected precordium. In 7 of the 25 equipped athletes (3 lacrosse goalies, 2 baseball catchers, 2 hockey goalies), all with standard design chest protectors, the projectile which was judged to have struck the chest barrier directly, but nevertheless resulted in the commotio cordis event.

Conclusions: Many commercially available chest barriers or safety projectiles do not provide absolute protection from sudden death due to commotio cordis. These tragic events, primarily in young children, underline the importance of developing preventive strategies by designing more effective equipment to provide a higher level of protection from ventricular fibrillation following a precordial blow.

1091-119

Purkinje Network Is the Origin of the Beasts in Catecholaminergic Bidirectional Ventricular Tachycardia and in the Initiating Beat of Short Coupling Variant of Torsade de Pointes

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Background: Catecholaminergic Bidirectional Ventricular Tachycardia (CBVT) and Short-Coupled Variant of Torsade de Pointes (SCtVP) cause syncope and sudden cardiac death (SCD). Methods: This study evaluated the origin of the beats initiating and perpetuating tachyarrhythmias in 8 patients (pts) with CBVT (Group 1) and in 3 pts with SCtVP (Group 2). DNA analysis was performed in 5 out of 11 pts. Results: The most prevalent origin was the right anteroseptal wall in 2 pts in Group 1 and 1 pt in Group 2. Electrophysiologic information was present in 5 out of 11 pts from both groups. All beats recorded at earliest site of activation with the Right Bundle Branch Block (RBBB) with Left Axis (LAx) morphology presented a Purkinje potential (PK) preceding the ventricular electrogram both in sinus rhythm and during VT. A left bundle branch block (LBBB) configuration beat assessed in 2 pts out 3 with CBVT and in 2 out of 3 with SCtVP presented a PK preceding V electrogram. The mean QRS duration was 114 and 140 ms for RBBB with LAx and LBBB respectively. One pt from Group 1 was submitted to radiofrequency ablation of one morphology (RBBB + LAx) and 1 pt from Group 2 for two morphologies (RBBB + LAx) and LBBB. No mutations were identified in the Cardiac Ryanodine Receptor Gene (RYR2).

Conclusion: The Purkinje network is the origin of the ventricular beats in CBVT and in the initiating beat of SCtVP. Ablation therapy may be indicated for selected patients in the SCtVP group when the initiating beat is correctly identified.